Air Toxics Risk Assessment Reference Library

Volume 1

Technical Resource Manual

U.S. Environmental Protection Agency
Office of Air Quality Planning and Standards
Research Triangle Park, NC

EPA-453-K-04-001A
www.epa.gov/air/oaqps

April 2004

EPA-453-K-04-001A
April 2004

Air Toxics Risk Assessment Reference Library
Volume 1
Technical Resource Manual

Prepared by:
ICF Consulting
Fairfax, Virginia

Prepared for:

Nona Smoke, Project Officer
Office of Policy Analysis and Review
Contract No. EP-D-04-005
Work Assignment No. 0-2

Rachael Schwartz, Project Officer
Clean Air Marketing Division
Contract No. 68-W-03-028
Work Assignment No. 11

Bruce Moore, Project Officer
Office of Air Quality Planning and Standards
Contract No. 68-D01-052
Work Assignment No. 0-08
Work Assignment No. 0-09

U.S. Environmental Protection Agency
Office of Air Quality Planning and Standards
Emissions Standards Division
Research Triangle Park, NC

Disclaimer

The information and procedures set forth here are intended as a technical resource to those conducting air toxics risk assessments. This Technical Resource Document does not constitute rulemaking by the Agency, and cannot be relied on to create a substantive or procedural right enforceable by any party in litigation with the United States. As indicated by the use of non-mandatory language such as “may” and “should,” it provides recommendations and does not impose any legally binding requirements.

The statutory provisions and EPA regulations described in this document contain legally binding requirements. This document is not a regulation itself, nor does not it change or substitute for those provisions and regulations. While EPA has made every effort to ensure the accuracy of the discussion in this guidance, the obligations of the regulated community are determined by statutes, regulations, or other legally binding requirements. In the event of a conflict between the discussion in this document and any statute or regulation, this document would not be controlling.

The general description provided here may not apply to a particular situation based upon the circumstances. Interested parties are free to raise questions and objections about the substance of this guidance and the appropriateness of the application of this guidance to a particular situation. EPA and other decision makers retain the discretion to adopt approaches on a case-by-case basis that differ from those described in this guidance where appropriate. EPA may take action that is at variance with the recommendations and procedures in this document and may change them at any time without public notice. This is a living document and may be revised periodically. EPA welcomes public input on this document at any time.

Reference herein to any specific commercial products, process, or service by trade name, trademark, manufacturer, or otherwise, does not necessarily constitute or imply its endorsement, recommendation, or favoring by the United States Government.

Acknowledgments

The U.S. Environmental Protection Agency's Air Toxics Risk Assessment reference library is a product of the EPA's Office of Air Quality, Planning, and Standards (OAQPS) in conjunction with EPA Regions 4 and 6 and the Office of Policy Analysis and Review. The interoffice technical working group responsible for library development includes Dr. Kenneth L. Mitchell (Region 4), Dr. Roy L. Smith (OAQPS), Dr. Deirdre Murphy (OAQPS), and Dr. Dave Guinnup (OAQPS). In addition to formal peer review, an opportunity for review and comment on Volumes 1 and 2 of the library was provided to various stakeholders, including internal EPA reviewers, state and local air agencies, and the private sector. The working group would like to thank these many internal and external stakeholders for their assistance and helpful comments on various aspects of these two books. (Volume 3 of the library is currently under development and is expected in late 2004.) The library is being prepared under contract to the U.S. EPA by ICF Consulting, Robert Hegner, Ph.D., Project Manager.

Authors, Contributors, and Reviewers

Authors

Kenneth L. Mitchell, Ph.D.
U.S. EPA Region 4

Roy L. Smith, Ph.D.
U.S. EPA OAQPS

Deirdre Murphy, Ph.D.
U.S. EPA OAQPS

External Peer Reviewers

Doug Crawford-Brown, Ph.D., University of North Carolina at Chapel Hill
Michael Dourson, Ph.D., D.A.B.T., Toxicology Excellence for Risk Assessment
Eric Hack, M.S., Toxicology Excellence for Risk Assessment
Bruce Hope, Ph.D., Oregon Department of Environmental Quality
Howard Feldman, M.S., American Petroleum Institute
Barbara Morin, Rhode Island Department of Environmental Management
Patricia Nance, M.A., M.Ed., Toxicology Excellence for Risk Assessment
Charles Pittinger, Ph.D., Toxicology Excellence for Risk Assessment, Exponent

Additional Contributors & Reviewers

John Ackermann, Ph.D., U.S. EPA Region 4
Carol Bellizzi, U.S. EPA Region 2
George Bollweg, U.S. EPA OAQPS
Pamela C. Campbell, ATSDR
Ruben Casso, U.S. EPA Region 6
Motria Caudill, U.S. EPA Region 5
Rich Cook, U.S. EPA, OTAQ
Paul Cort, U.S. EPA Region 9
David E. Cooper, Ph.D., U.S. EPA OSWER
Dave Crawford, U.S. EPA OSWER
Stan Durkee, U.S. EPA Office of Science Policy
Neal Fann, U.S. EPA OAQPS
Bob Fegley, U.S. EPA Office of Science Policy
Gina Ferreira, USEPA Region 2
Gerald Filbin, Ph.D., U.S. EPA OPEI
Danny France, U.S. EPA Region 4
Rick Gillam, U.S. EPA Region 4
Thomas Gillis, U.S. EPA OPEI
Barbara Glenn, Ph.D., U.S. EPA National Center for Environmental Research
Dave Guinnup, Ph.D., U.S. EPA OAQPS
Bob Hetes, U.S. EPA National Health and Environmental Effects Research Laboratory
James Hirtz, U.S. EPA Region 7
Ofia Hodoh, M.S., U.S. EPA Region 4
Ann Johnson, U.S. EPA OPEI
Brenda Johnson, U.S. EPA Region 4
Pauline Johnston, U.S. EPA ORIA
Stan Krivo, U.S. EPA Region 4
Deborah Luecken, U.S. EPA National Exposure Research Laboratory
Thomas McCurdy, U.S. EPA National Exposure Research Laboratory
Megan Mehaffey, Ph.D., U.S. EPA NERL
Latoya Miller, U.S. EPA Region 4
Erin Newman, U.S. EPA Region 5
David Lynch, U.S. EPA OPPTS
Ted Palma, M.S., U.S. EPA OAQPS
Michele Palmer, U.S. EPA Region 5
Solomon Pollard, Jr., Ph.D., U.S. EPA Region 4
Anne Pope, U.S. EPA OAQPS
Marybeth Smuts, Ph.D., U.S. EPA Region 1
Michel Stevens, U.S. EPA National Center for Environmental Assessment
Allan Susten, Ph.D., D.A.B.T., ATSDR
Henry Topper, Ph.D., U.S. EPA OPPTS
Pam Tsai, Sc.D., D.A.B.T., U.S. EPA Region 9
Susan R. Wyatt, U.S. EPA (retired)
Jeff Yurk, M.S., U.S. EPA Region 6

(This page intentionally left blank)

Table of Contents

PART I BACKGROUND

Chapter 1 Introduction

1.1 Introduction 1

1.2 The Special Concerns of Urban Areas 2

1.3 Promoting Localized Assessment 4

1.4 The Risk-Based Approach 4

1.5 The Purpose of this Reference Manual 5

1.6 The Layout of this Reference Manual 6

1.7 The Relationship of this Manual to Volumes 2 and 3 9

References 10

Chapter 2 Clean Air Act Requirements and Programs to Regulate Air Toxics

2.1 Introduction 1

2.2 HAPs and their Sources: Stationary, Mobile, and Indoor Sources 2
- 2.2.1 Hazardous Air Pollutants (HAPs) 2
- 2.2.2 Stationary Sources: The Pre-1990 CAA "Risk-Only" Approach 4
- 2.2.3 Stationary Sources and the 1990 Clean Air Act Amendments: A "Technology First, Then Risk" Approach 4
  - 2.2.3.1 Step 1: The Technology-based Approach 5
  - 2.2.3.2 Step 2: The Risk-based Approach 5
- 2.2.4 Mobile Sources of Air Toxics Rule 6
- 2.2.5 Indoor Air and Indoor Air Toxics 8
  - 2.2.5.1 Potential Sources of Indoor Air Toxics 9
  - 2.2.5.2 Indoor Air Toxics 9
  - 2.2.5.3 Health Risks and Indoor Pollutants 11

2.3 Progress in Understanding and Reducing Toxic Air Pollution 12
- 2.3.1 Trends 12
- 2.3.2 NATA National Scale Assessment 14

2.4 Other Air Pollutants of Potential Concern 16
- 2.4.1 Criteria Air Pollutants 16
- 2.4.2 Chemicals on the Toxics Release Inventory 20
- 2.4.3 Toxic Chemicals that Persist and Which Also May Bioaccumulate 21
- 2.4.4 Overlaps and Differences Between Chemical "Lists" 22

2.5 Reports to Congress on Air Toxics Issues 23
2.5.1 Air Toxics Deposition to the Great Waters 23
2.5.2 Mercury Study Report to Congress 24
2.5.3 Utility Report to Congress 25
2.5.4 Residual Risk Report to Congress 26
2.5.5 Integrated Urban Strategy Report to Congress 27
2.5.6 Other Reports 27

References 27

Chapter 3 EPA's Risk Assessment Process for Air Toxics: History and Overview

3.1 Introduction 1

3.2 A Short History of the Development of Human Health Risk Assessment and Risk Management Approaches for Air Toxics 1
3.2.1 The 1983 National Academy of Sciences Report 1
3.2.2 The 1994 National Research Council Report 3
3.2.3 The CRARM 4
3.2.4 Development of Human Health Risk Assessment at EPA 6

3.3 Air Toxics Human Health Risk Assessment: Overview of the Process 9
3.3.1 Air Toxics Risk Assessment: What Is the Question? 10
3.3.2 Air Toxics Risk Assessment: The Process 12
3.3.2.1 Planning, Scoping, and Problem Formulation 13
3.3.2.2 Analysis Phase 14
3.3.2.3 Risk Characterization 15
3.3.3 Tiered Assessment Approaches 16

3.4 Uncertainty and Variability in Air Toxics Risk Assessment 18
3.4.1 Distinguishing Uncertainty and Variability 19
3.4.2 Sources of Uncertainty in Air Toxics Risk Assessment 20
3.4.3 Sources of Variability in Air Toxics Risk Assessment 22
3.4.4 Characterizing Uncertainty and Variability 23
3.4.5 Tiered Approach to Uncertainty and Variability 24
3.4.6 Assessment and Presentation of Uncertainty 26

References 29

Chapter 4 Air Toxics: Chemicals, Sources, and Emissions Inventories

4.1 Introduction 1

4.2 Air Toxics 1
4.2.1 Introduction to Air Toxics Chemical Lists 1
4.2.2 Hazardous Air Pollutants (HAPs) 4
4.2.3 Criteria Air Pollutants 5
4.2.4 Toxics Release Inventory (TRI) Chemicals 6
4.2.5 Toxic Chemicals That Persist and Which Also May Bioaccumulate 6
4.2.6 Other Chemicals 11

4.3 Sources of Air Toxics 11

4.3.1 Point Sources 12
4.3.2 Nonpoint Sources 13
4.3.3 On-Road and Nonroad Mobile Sources 14
4.3.4 Sources Not Included in the NEI or TRI 15
4.3.4.1 Indoor Sources 16
4.3.4.2 Natural Sources 18
4.3.4.3 Formation of Secondary Pollutants 19
4.3.4.4 Other Sources Not Included in NEI or TRI 20

4.4 Emissions Inventories 20
4.4.1 National Emissions Inventory (NEI) 21
4.4.2 Toxics Release Inventory (TRI) 25

References 29

PART II HUMAN HEALTH RISK ASSESSMENT: INHALATION

Chapter 5 Getting Started: Planning and Scoping the Inhalation Risk Assessment

5.1 Introduction 1

5.2 Framework and Process for Air Toxics Risk Assessments 1
5.2.1 Framework for Cumulative Risk Assessment 1
5.2.2 General Framework for Residual Risk Assessment 3
5.2.3 The Air Toxics Risk Assessment Process 3
5.2.4 Overview of Inhalation Exposure Assessment 6
5.2.4.1 Exposure and Exposure Assessment: What's the Difference? 6
5.2.4.2 Components of an Exposure Assessment 7

5.3 Planning and Scoping 8
5.3.1 Why is Planning and Scoping Important? 9
5.3.2 The Planning and Scoping Process 9
5.3.2.1 What is the Concern? 9
5.3.2.2 Who Needs to be Involved? 10
5.3.2.3 What is the Scope? 12
5.3.2.4 Why is There a Problem? 13
5.3.2.5 How will Risk Managers Evaluate the Concern? 13
5.3.2.6 Lessons Learned on Planning and Scoping 13

References 15

Chapter 6 Problem Formulation: Inhalation Risk Assessment

6.1 Introduction 1

6.2 Developing the Conceptual Model 1

6.3 Developing the Analysis Plan 4
6.3.1 Identification of the Sources 5
6.3.2 Identification of the Chemicals of Potential Concern 6

6.3.2.1 Toxicity-Weighted Screening Analysis 6
6.3.2.2 Risk-Based Screening Analysis 7

6.3.3 Identification of the Exposure Pathways/Routes 8
6.3.3.1 Characteristics of the Assessment Area 12
6.3.3.2 Scale of the Assessment Area 14
6.3.3.3 Use of Modeling versus Monitoring 14
6.3.3.4 Estimation of Exposure 17
6.3.3.5 Evaluation of Uncertainty 20
6.3.3.6 Preparation of Documentation 20
6.3.4 Identification of the Exposed Population 20
6.3.5 Identification of the Endpoints and Metrics 21

6.4 Data Quality in the Risk Assessment Process 22

References 24

Chapter 7 Quantification of Exposure: Development of the Emissions Inventory for the Inhalation Risk Assessment

7.1 Introduction 1

7.2 Process for Developing an Emissions Inventory 1
7.2.1 Planning 2
7.2.2 Gathering Information 3
7.2.3 Estimating Emissions 3
7.2.3.1 Direct Measurement 5
7.2.3.2 Emission Estimation Models 5
7.2.3.3 Emission Factors 8
7.2.3.4 Mass Balance 9
7.2.3.5 Engineering Judgment 9
7.2.4 Compiling Data Into a Database 9
7.2.4.1 Selection of Production Rates 9
7.2.4.2 Unusual Conditions: Process Upsets, Accidental Releases, and Maintenance 11
7.2.5 Data Augmentation 12
7.2.6 Quality Assurance/Quality Control 13
7.2.7 Documentation 14
7.2.8 Access to Data 14

7.3 Data Sources 14
7.3.1 Permit Files 15
7.3.2 Regional Inventories 15
7.3.3 Industry Profiles 16
7.3.4 AP-42 Emissions Factors 17
7.3.5 Factor Information Retrieval System 17
7.3.6 Locating and Estimating Documents 18
7.3.7 RCRAInfo 18
7.3.8 Emissions and Dispersion Modeling System (EDMS) 19
7.3.9 Summary 19

References 21

Chapter 8 Quantification of Exposure: Dispersion, Transport, and Fate of Air Toxics in the Atmosphere

8.1 Introduction 1

8.2 Dispersion and Atmospheric Transport of Air Pollutants 1
- 8.2.1 General Types of Releases 2
- 8.2.2 Characteristics of Releases that Affect Dispersion and Transport 3
- 8.2.3 Physical and Meteorological Factors Affecting Air Toxics Dispersion and Transport . 7

8.3 Fate of Air Toxics in the Atmosphere 13
- 8.3.1 Physical Processes Removing Air Toxics 13
- 8.3.2 Chemical Reactions that Remove Air Toxics 15
- 8.3.3 Chemical Reactions that Result in the Secondary Formation of Pollutants 16
- 8.3.4 Overall Persistence of Air Toxics in the Atmosphere 17

References 19

Chapter 9 Assessing Air Quality: Modeling

9.1 Introduction 1

9.2 Air Quality Modeling 1
- 9.2.1 The Overall Structure of an Air Quality Model 1
- 9.2.2 Types of Models: Scientific Principles 6
- 9.2.3 Modeling Deposition 7
- 9.2.4 Screening vs. Refined Models 8
- 9.2.5 Specific Data Required for Modeling 9
- 9.2.6 Sources of Air Quality Models and Information 11
- 9.2.7 Examples of Air Quality Models 12
- 9.2.8 Emissions from Soil 18

9.3 Air Quality Modeling Examples 19

References 19

Chapter 10 Assessing Air Quality: Monitoring

10.1 Introduction 1

10.2 Air Toxics Monitoring: Recent Advances 2

10.3 Monitoring for Air Toxics Risk Assessments: Why Monitor? 4

10.4 Planning for Air Toxics Monitoring 9
- 10.4.1 General Planning Approach 10
- 10.4.2 Specific Planning Issues 14

10.5 Implementing Air Toxics Monitoring 19
10.5.1 Locating Monitors and Selecting Sample Size 19
10.5.1.1 Locating Monitors 19
10.5.1.2 Selecting Sample Size 24
10.5.1.3 Setting Up a Monitoring/Sampling Program 25
10.5.2 Data Analysis and Reporting 28
10.5.3 The Use of Monitoring Data to Calculate Exposure Concentrations 29

10.6 Monitoring Methods, Technologies, and Costs 31
10.6.1 Ambient Air Monitoring 33
10.6.2 Sampling Costs 35

10.7 Archiving Air Toxics Monitoring Data 35

10.8 Using Air Monitoring Data to Evaluate Source Contribution 37

References 38

Chapter 11 Estimating Inhalation Exposure

11.1 Introduction 1

11.2 Estimating Inhalation Exposure Concentrations 1
11.2.1 General Approaches for Deriving Exposure Concentrations 2
11.2.2 Common Ways to Estimate Exposure Concentrations 3

11.3 Exposure Modeling 6
11.3.1 Inhalation Exposure Modeling 9
11.3.2 Microenvironment Concentration: How is it Developed? 12
11.3.3 Sources of Data for Human Activity for Inhalation (and other) Exposure Assessments 13
11.3.4 Examples of Inhalation Exposure Models 16
11.3.5 Exposure Modeling Examples 19

11.4 Personal Monitoring 20

11.5 Exposure to a Population: Common Descriptors 22

11.6 Evaluating Uncertainty 22

11.7 Presenting the Results of an Exposure Assessment 23

References 23

Chapter 12 Inhalation Toxicity Assessment

12.1 Introduction 1
12.1.1 Hazard Identification and Dose-Response Information 1
12.1.2 Dose-Response Assessment Methods 5

12.2 Hazard Identification 8
12.2.1 Weight of Evidence – Human Carcinogenicity 9
12.2.2 Identification of Critical Effect(s) – Non-Cancer Endpoints 11

12.3 Dose-Response Assessment for Cancer Effects 12
12.3.1 Determination of the Point of Departure (POD) 13
12.3.2 Derivation of the Human Equivalent Concentration 14
12.3.3 Extrapolation from POD to Derive Carcinogenic Potency Estimates 17

12.4 Dose-Response Assessment for Derivation of a Reference Concentration 19
12.4.1 Determination of the Point of Departure and Human Equivalent Concentration 20
12.4.2 Application of Uncertainty Factors 22

12.5 Sources of Chronic Dose-Response Values 24

12.6 Acute Exposure Reference Values 26

12.7 Evaluating Chemicals Lacking Health Reference Values 31
12.7.1 Use of Available Data Sources 31
12.7.2 Route-to-Route Extrapolation 31

12.8 Dose-Response Assessment for Mixtures 32

References 35

Chapter 13 Inhalation Risk Characterization

13.1 Introduction 1

13.2 Quantification of Cancer Risk and Noncancer Hazard 4
13.2.1 Cancer Risk Estimates 5
13.2.1.1 Characterization of Individual Pollutant Risk 5
13.2.1.2 Characterization of Cancer Risk from Exposure to Multiple Pollutants 6
13.2.2 Noncancer Hazard Estimates 8
13.2.2.1 Characterizing Individual Pollutant Hazard for Chronic Exposures 8
13.2.2.2 Characterizing Multiple Pollutant Hazard for Chronic Exposures 9
13.2.2.3 Characterizing Hazard for Acute Exposures 11
13.2.3 Quantifying Risk From Background Sources 12

13.3 Interpretation and Presentation of Inhalation Cancer Risks and Noncancer Hazards 13
13.3.1 Presenting Risk and Hazard Estimates 17
13.3.2 Exposure Estimates and Assumptions 17
13.3.3 Toxicity Estimates and Assumptions 18
13.3.4 Assessment and Presentation of Uncertainty in Risk Characterization 19
13.3.4.1 Practical Approaches to Uncertainty Assessment 20
13.3.4.2 Presentation of Uncertainty Assessment 23
13.3.5 Additional Information 23

References 25

PART III HUMAN HEALTH RISK ASSESSMENT: MULTIPATHWAY

Chapter 14 Overview and Getting Started: Planning and Scoping the Multipathway Risk Assessment

14.1 Introduction 1

14.2 Overview of Multipathway Air Toxics Risk Assessment 3
14.2.1 Planning, Scoping, and Problem Formulation 3
14.2.2 Analysis 4
14.2.3 Risk Characterization 5

14.3 Overview of Multipathway Exposure Assessment 5

14.4 Planning and Scoping 9
14.4.1 Identifying the Concern 9
14.4.2 Identifying The Participants 9
14.4.3 Determining the Scope of the Risk Assessment 9
14.4.4 Describing the Problem 10
14.4.5 Determining How Risk Managers Will Evaluate the Concern 10

14.5 Tiered Multipathway Risk Assessments 10

References 11

Chapter 15 Problem Formulation: Multipathway Risk Assessment

15.1 Introduction 1

15.2 Developing the Multipathway Conceptual Model 1

15.3 Developing the Multipathway Analysis Plan 2
15.3.1 Identification of the Sources 3
15.3.2 Identification of the Chemicals of Potential Concern 3
15.3.3 Identification of the Exposure Pathways/Routes 3
15.3.3.1 Characteristics of the Assessment Area 4
15.3.3.2 Scale of the Assessment Area 5
15.3.3.3 Use of Modeling vs. Monitoring 5
15.3.3.4 Quantitation of Exposure 6
15.3.3.5 Evaluation of Uncertainty 7
15.3.3.6 Preparation of the Documentation 7
15.3.4 Identification of the Exposed Population 7
15.3.5 Identification of Endpoints and Metrics 7

15.4 Exposure Assessment Approach 7
15.4.1 Scenario Approach 8
15.4.2 Population-Based Approach 11

References 12

Chapter 16 Quantification of Exposure: Development of the Emissions Inventory for the Multipathway Risk Assessment

16.1 Introduction 1

16.2 Developing the Emissions Inventory 1

Chapter 17 Quantification of Exposure: Chemical and Physical Properties Affecting Multimedia Fate and Transport

17.1 Introduction 1
17.1.1 Measures of Persistence 1
17.1.2 Metrics of Bioaccumulation 5

17.2 Chemical and Physical Properties that Affect Persistence and Bioaccumulation 8

17.3 Evaluating Persistence and Bioaccumulation in Exposure Assessments 10

References 11

Chapter 18 Quantification of Exposure: Multimedia Modeling

18.1 Introduction 1

18.2 Multimedia Fate and Transport Modeling 1
18.2.1 Basis of Multimedia Models 1
18.2.2 Multimedia Exposure Models 2

18.3 Key Parameters/Inputs for Multimedia Models 5

18.4 Examples of Multimedia Modeling 8

References 10

Chapter 19 Quantification of Exposure: Multimedia Monitoring

19.1 Introduction 1

19.2 Why Monitor? 1

19.3 Planning and Implementing Issues 1

19.4 Monitoring and Sampling Methods, Technologies and Costs 3
19.4.1 Method Selection 3
19.4.2 Available Methods 7

References 12

Chapter 20 Exposure Metrics for Multimedia Assessment

20.1 Introduction 1

20.2 Generic Equation for Dietary Intake 3

20.3 Estimating Exposure Concentrations 4

20.4 Calculating Intake Variable Values 6
- 20.4.1 Consumption Rate 7
- 20.4.2 Exposure Frequency 10
- 20.4.3 Exposure Duration 10
- 20.4.4 Body Weight 12

20.5 Calculating Averaging Time Value 12

20.6 Combining Exposure Estimates Across Pathways 13

20.7 Exposure Models 14

References 16

Chapter 21 Ingestion Toxicity Assessment

21.1 Introduction 1

21.2 Hazard Identification 2

21.3 Predictive Approach for Cancer Effects 2
- 21.3.1 Determining the Point of Departure (POD) 2
- 21.3.2 Deriving the Human Equivalent Dose 2
- 21.3.3 Extrapolating from POD to Derive the Oral Cancer Slope Factor 3

21.4 Dose-response Assessment for Derivation of a Reference Dose 3

21.5 Sources of Human Health Reference Values for Risk Assessment 4

References 5

Chapter 22 Multipathway Risk Characterization

22.1 Introduction 1

22.2 Cancer Risk Estimates 2
- 22.2.1 Characterizing Individual Pollutant Ingestion Risk - Scenario Approach 2
- 22.2.2 Characterizing Risk from Exposure to Multiple Pollutants - Scenario Approach 3
- 22.2.3 Combining Risk Estimates across Multiple Ingestion Pathways - Scenario Approach . . 4
- 22.2.4 Evaluating Risk Estimates from Inhalation and Ingestion Exposures 4

22.3 Noncancer Hazard 4
22.3.1 Characterizing Individual Pollutant Hazard - Scenario Approach 5
22.3.2 Multiple Pollutant Hazard 6
22.3.3 Evaluating Hazard Estimates From Inhalation and Ingestion Exposures 6

22.4 Interpretation and Presentation of Risks/Hazards 7

References 8

PART IV ECOLOGICAL RISK ASSESSMENT

Chapter 23 Overview and Getting Started: Problem Formulation

23.1 Introduction 1

23.2 Overview of Air Toxics Ecological Risk Assessment 4
23.2.1 Problem Formulation 6
23.2.2 Analysis 8
23.2.3 Evaluation of Ecological Effects 9
23.2.4 Ecological Risk Characterization 9

23.3 Planning and Scoping 9
23.3.1 What is the Concern? 10
23.3.2 Identifying The Participants 11
23.3.3 Determining the Scope of the Risk Assessment 12
23.3.4 Study-Specific Conceptual Model 12
23.3.4.1 Identifying Receptors of Concern 13
23.3.4.2 Identifying Assessment Endpoints and Measures of Effects 15
23.3.5 Analysis Plan and Quality Assurance Program Plan (QAPP) 16

23.4 Tiered Ecological Risk Assessments 21

References 23

Chapter 24 Analysis: Characterization of Ecological Exposure

24.1 Introduction 1

24.2 Characterization of Exposure 1
24.2.1 Quantifying Releases 3
24.2.2 Estimating Chemical Fate and Transport 3
24.2.2.1 Physical and Chemical Parameters 3
24.2.2.2 Multimedia Modeling 3
24.2.2.3 Multimedia Monitoring 4
24.2.3 Quantifying Exposure 5
24.2.3.1 Metrics of Exposure 5
24.2.3.2 Dimensions of Exposure 7
24.2.3.3 Exposure Profile 9
24.2.3.4 Evaluating Variability and Uncertainty 9

References 10

Chapter 25 Analysis: Characterization of Ecological Effects

25.1 Introduction 1

25.2 Ecological Response Analysis 1
25.2.1 Stressor-Response Analysis 1
25.2.1.1 Ecological Effect Levels 1
25.2.1.2 Selection of TRVs for a Particular Assessment 7
25.2.1.3 Stressor-Response Curves 11
25.2.1.4 Species Sensitivity Distribution 11
25.2.2 Linking Measures of Effects to Assessment Endpoints 12

25.3 Stressor-Response Profile 16

25.4 Evaluating Variability and Uncertainty 16

References 16

Chapter 26 Ecological Risk Characterization

26.1 Introduction 1

26.2 Risk Estimation 2
26.2.1 Single-Point Exposure and Effects Comparisons 2
26.2.2 Comparisons Involving the Entire Stressor-Response Relationship 3
26.2.3 Comparisons Involving Variability 4
26.2.4 Process Models 4

26.3 Risk Description 5
26.3.1 Lines of Evidence 5
26.3.2 Significance of the Effects 6

26.4 Risk Characterization Report 7

26.5 Evaluating Variability and Uncertainty 9

References 9

PART V RISK-BASED DECISION MAKING

Chapter 27 Risk Management

27.1 Introduction 1

27.2 Role of Risk Management in Regulating Hazards 1

27.3 Types of Risk Management Decisions Related to Air Toxics 4

27.4 Use of Risk Estimates in Decision-Making 5

27.5 Process for Making Risk Management Decisions 9
27.5.1 Define the Problem and Put it in Context 9
27.5.2 Analyze the Risks Associated with the Problem in Context 9
27.5.3 Examine Options for Addressing the Risks 11
27.5.4 Make Decisions about Which Options to Implement 12
27.5.5 Take Actions to Implement the Decisions 13
27.5.6 Conduct an Evaluation of the Action's Results 14

27.6 Information Dissemination 14

References 15

Chapter 28 Community Involvement

28.1 Introduction 1

28.2 Why is Community Involvement Important? 1

28.3 When to Involve the Community 2

28.4 How to Involve the Community 2
28.4.1 Understand Goals, Objectives, and Responsibilities for Effective Community Involvement 4
28.4.2 Identify Community Concerns and Interest 5
28.4.3 Plan Community Involvement Strategy and Activities 5
28.4.4 Identify Possible Tools and Implement Community Involvement Activities 6
28.4.5 Provide Opportunity for Continued Public Interaction 7
28.4.6 Release of Risk Assessment and Risk Management Documents 8

References 10

Chapter 29 Risk Communication

29.1 Introduction 1

29.2 Risk Perception 2

29.3 Your Risk Communication Strategy 2

29.4 Risk Comparisons 3

29.5 Implementing Risk Communication Strategies 5
29.5.1 Presentation of Risk Results 5
29.5.2 Working with the Media 8

References 14

PART VI SPECIAL TOPICS

Chapter 30 Public Health Assessment

30.1 Introduction 1

30.2 History of Public Health Assessment 2

30.3 Relationship of Public Health Assessment to Risk Assessment 3

30.4 What Is Public Health Assessment? 4

30.5 How Is a Public Health Assessment Conducted? 6
- 30.5.1 Conduct Scoping 6
- 30.5.2 Obtain Study Area Information 7
- 30.5.3 Community Involvement/Outreach/Response to Community Concerns 7
- 30.5.4 Exposure Evaluation 8
- 30.5.5 Health Effects Evaluation 9
- 30.5.6 Draw Public Health Conclusions 12
- 30.5.7 Recommend Public Health Actions 13
- 30.5.8 Prepare PHA Documents 13

References 14

Chapter 31 Probabilistic Risk Assessment

31.1 Introduction 1

31.2 Tiered Approach for Risk Assessment 2

31.3 Methods for Probabilistic Risk Assessment 4

31.4 Presenting Results for Probabilistic Risk Assessment 6

References 10

Chapter 32 Use of Geographic Information Systems (GIS) in Risk Assessment

32.1 Introduction 1

32.2 Selecting a GIS 2

32.3 Acquiring and Using Demographic Data 4
- 32.3.1 U.S. Census Data 5
- 32.3.2 Current and Small-Area Demographic Estimates 5
- 32.3.3 Public Health Applications 7
- 32.3.4 Data Access and Distribution 7

32.4 Cartographic Concepts 7
- 32.4.1 Generalization, Simplification, and Abstraction 10
- 32.4.2 Map Projections 10

32.5 Using the Internet as a GIS Tool 10

32.6 Current GIS Applications at EPA 11
32.6.1 ORD/ESD 12
32.6.2 ATtILA 12
32.6.3 ReVA 12

32.7 GPS Technology 13

References 15

GLOSSARY

APPENDICES

Appendix A **Listing of all HAPs**

Appendix B **Guide to Federal Agencies that Oversee Air Toxics**

Appendix C **Recommended Dose-Response Values for HAPs**

Appendix D **Methodology for Identifying PB-HAP Compounds**

Appendix E **Overview of Air Toxics Emission Sources**

Appendix F **Specific HAPs Included in the National Emissions Inventory (NEI)**

Appendix G **Atmospheric and Meteorological Concepts Relevant to Dispersal, Transport, and Fate of Air Toxics**

Appendix H **Data Quality Evaluation**

Appendix I **Use of Air Monitoring Data to Develop Estimates of Exposure Concentration (Data Analysis and Reduction)**

Appendix J **Air Monitoring and Sampling Methods**

Appendix K **Equations For Estimating Concentrations of PB-HAP Compounds in Food and Drinking Water**

PART I

BACKGROUND

Chapter 1 Introduction

Table of Contents

1.1 Introduction 1

1.2 The Special Concerns of Urban Areas 2

1.3 Promoting Localized Assessment 4

1.4 The Risk-Based Approach 4

1.5 The Purpose of this Reference Manual 5

1.6 The Layout of this Reference Manual 6

1.7 The Relationship of this Manual to Volumes 2 and 3 9

References 10

1.1 Introduction

The mission of the United States Environmental Protection Agency (EPA) is to protect human health and to safeguard the natural environment – air, water, and land – upon which life depends.[1] Following this mission, the Agency has implemented a variety of laws and programs that require and encourage the safe use and management of toxic chemicals. Many of these programs focus on understanding the consequences of releasing chemicals to the air, land, and water and working to reduce those releases when they pose too great a risk (see Glossary for definition of risk in this Reference Library). This manual describes the programs and technical tools that EPA uses to evaluate and address chemicals that are released to the air from many different types of sources, and which have the potential to harm people and the environment.

The potential impacts of chemicals released to the air depend on a number of factors, including the quantity of chemicals in the air, how the chemicals move and transform in the environment, the length of time people or the environment is exposed, and the toxic nature of the chemicals. The human health effects of exposure to air pollutants can range from no response, responses that are relatively minor and reversible (such as mild eye irritation), responses that are more serious and debilitating (such as aggravation of asthma) and, in some cases, fatal responses. Air pollution also can cause negative impacts on the environment, including distress and death in plants and animals, as well as damage to buildings and important cultural sites.

In the mid-20th century, Congress recognized the potential for air pollution to cause these kinds of problems and responded by enacting the Clean Air Act (CAA). Since that time, this Act, as amended, has provided the primary authority that EPA uses to develop programs for protecting people and the environment from the harmful effects of air pollution across the United States.

A key component of the current version of the CAA (most recently amended by the 1990 CAA Amendments) is the requirement that EPA significantly reduce emissions to the air of chemicals that are known or suspected to cause serious health problems, such as cancer or birth defects. As a starting point in this effort, the Act explicitly identifies 188 **hazardous air pollutants (HAPs)**[a] for regulation. This group of chemicals is also commonly referred to as the HAPs, **toxic air pollutants** or, simply, **air toxics**. (The CAA also covers another important group of chemicals, known as **criteria air pollutants**; these are discussed in Chapter 2.)

Many different types of sources can release air toxics. These sources include stationary facilities that release large quantities of HAPs to the air (known as **major sources**); stationary facilities that release smaller amounts of HAPs to the air (known as **area sources**); on-road and nonroad **mobile sources** (such as cars, trucks, and construction equipment) that release HAPs to the air; **indoor sources** of air toxics (such as paint and cleaning products); and **natural sources** of air toxics (such as volcanoes). Chapter 4 provides a detailed description of how EPA identifies and, in the case of anthropogenic (manmade) sources, regulates each of the various types of sources of air toxics.

---

[a] Since the original Act, which listed 189 chemicals, one chemical (caprolactam) has been delisted, leaving 188 HAPs. EPA is also in the process of considering proposals to delist methyl ethyl ketone (MEK and ethylene glycol butyl ether (EGBE)). EPA has the authority to add and delete chemicals from the original list based on specified criteria [CAA Section 112(b)(3)].

1.2 The Special Concerns of Urban Areas

In urban areas, toxic air pollutants are of particular concern because people and sources of emissions are concentrated in the same geographic area. Since most people live in urban areas, this proximity leads to the potential for large numbers of people to be exposed to numerous air pollutants. While some of these urban chemical exposures tend to be fairly similar across the country (e.g., ambient air concentrations of benzene from petroleum use tend to be similar across the lower 48 states), studies also indicate that the concentrations of air toxics in many urban (and some nonurban) areas can vary significantly from one location to the next (e.g., concentrations in areas with petroleum refineries may be higher than in areas that do not have petroleum refineries). The sources of urban emissions tend to be relatively small in size but large in number, such as gas stations or mobile sources. In addition, these emissions are typically found at ground level where people are more likely to be exposed to them.

Urban air toxics also have a potential to elevate health risks among particular urban sub-populations, including children, the elderly, and persons with existing illnesses. In addition, the prevalence of minority and low-income communities in urban industrial and commercial areas, where concentrations of air toxics may be greatest, increases the likelihood of elevated exposures among these subpopulations.

Considering the large number of people potentially at risk from air toxics exposures, Congress directed in the 1990 CAA amendments that elevated outdoor (also called **ambient**) concentrations of air toxics in large urban areas be substantially reduced. In response to this mandate, EPA developed an **Integrated Urban Air Toxics Strategy**. This Urban Strategy, which was published in the *Federal Register* on July 19, 1999,[2] has since become EPA's **Air Toxics Strategy (The Strategy)** and is part of the overall national effort to reduce air toxics. The Strategy attempts to address all the significant stationary, mobile, and indoor sources necessary to achieve protection of public health and the environment. The specific goals of the Strategy are to:

- Attain a 75 percent reduction in incidence of cancer attributable to exposure to HAPs emitted by stationary sources;

- Attain a substantial reduction in public health risks posed by HAP emissions from area sources; and

- Address disproportionate impacts of air toxics hazards across urban areas.

The Strategy identifies four main areas of action to help achieve these goals:

- **Develop regulations addressing sources of air toxics at the national and local levels**. Pursuant to this effort, the Agency will continue its work to develop rules that require reductions in air toxics emissions from stationary facilities (such as manufacturing plants, electric power plants, gas stations, and dry cleaners), as well as from cars, trucks, and other mobile sources and their fuels. EPA has historically developed and implemented many such standards over the years, and the Strategy indicates the need for additional standards to reduce risks in urban areas.

- **Initiate local and community-based projects to address specific multi-media pollutants (e.g., mercury) and cumulative risks within urban areas**. The CAA requires EPA to "encourage and support area-wide strategies developed by the state or local air pollution control agencies" to address air toxics in urban areas. EPA is developing tools (such as this Reference Library) and is working with communities to assess and reduce risks at the community level.

  The Strategy also recognizes the need to assess the risks from exposures to indoor air toxics and to develop non-regulatory, voluntary programs to address those risks. The Strategy also points out that air pollutants may move into other environmental media such as soil and water resulting in multimedia (i.e., more than just air) concerns. EPA is engaged in a number of activities that recognize the ability of many air toxics to deposit out of the air and bioaccumulate in biota consumed by humans and ecological receptors (e.g., deposition of mercury in watersheds, with subsequent uptake by fish).

  In risk assessment, the term "receptor" generally refers to an individual person or ecological component that is potentially exposed to a stressor (air toxic). In modeling, the term sometime refers to the location where impacts are predicted.

- **Conduct air toxics assessments to identify areas of concern, prioritize efforts to reduce risks, and track progress**. The Strategy identifies a variety of national-level assessment activities that will help EPA identify urban areas of particular concern, characterize the risks that air toxics pose, and track the progress toward meeting overall air toxics program goals. EPA is implementing the National Air Toxics Assessment (NATA) to address this goal. NATA includes:
  - Expanding air toxics monitoring;
  - Improving and periodically updating emissions inventories;
  - Assessing national- and local-scale air quality by using multimedia and exposure modeling;
  - Continuing to research the exposures to, and health effects of, toxic chemicals in ambient and indoor air; and
  - Using and improving exposure and assessment tools.

  These activities will help EPA and other stakeholders[b] better understand air toxics risks, as well as risk reductions associated with emissions control standards and other initiatives aimed at reducing emissions. A particularly high-profile aspect of NATA has been the national-scale assessment of 1996 emissions that produced predictions of county-level estimates of air toxics concentrations and calculated risks for a subset of HAPs that EPA believes pose most of the urban area risk. For additional information this particular analysis, see EPA's *The National-Scale Air Toxics Assessment*.[3] The national scale assessment of 1999 emissions is currently being performed and will be released in 2004 (see Chapter 2).

[b]This reference manual uses the term "stakeholder" broadly to include all parties with a potential interest in a given air toxics risk assessment, including regulators, the regulated community, community partners, and individual members of the public.

- **Perform education and outreach**. Given the scientific complexity inherent in air toxics issues, EPA recognizes that the success of the overall air toxics program depends on the public's understanding of the nature of air toxics risks and the activities that can help reduce those risks. To further this understanding, EPA will support education and outreach efforts at the national level and through its state, local, and tribal (S/L/T) partners (e.g., government, industry, community). This reference manual, for example, is an outgrowth of this educational/outreach effort.

For additional information on the Integrated Urban Strategy see EPA's *Air Toxics Strategy: Overview*[4].

1.3 Promoting Localized Assessment

While substantial reductions have been achieved through federal standards, EPA is evaluating the need for additional emissions controls at the national level. However, since the mix of sources and pollutants in specific geographic areas can be quite variable, one element of an effective approach for reducing any remaining unacceptable risks is to understand the cumulative impacts at the local level, target the problem areas, and tailor risk reduction strategies to the local circumstances in those areas.

To encourage reductions of air toxics emissions at the local community level, EPA Headquarters and Regional Offices are working collaboratively with S/L/T and community partners. This team effort has focused on education/information exchanges, identification and assessment of pollution prevention and control options, and promotion of voluntary measures and innovative solutions to assess and address community air pollution problems.

While EPA has the authority to issue standards to address certain air toxics risks, in many cases these risks may be more appropriately and more effectively addressed at the S/L/T level, rather than at the federal level. Specifically, S/L/T air agencies may wish to address issues that are of concern on a state-wide, area-wide, community-wide, or individual neighborhood basis, and for areas in the immediate vicinities of specific air toxics sources. Some S/L/T governments are already addressing some of these issues; others are just beginning to develop their own programs.

1.4 The Risk-Based Approach

While there are several methodologies to assess potential health impacts of air toxics on populations at the local level, the risk-based approach is perhaps the most effective.

The methodology described here, called **risk assessment,** is the process for evaluating:

- The sources of air toxics released to the environment;
- How the released chemicals move and change in the environment;
- Who may be exposed to the chemicals and at what levels;
- How exposures may occur;
- The toxic effects of the chemicals in question and how potent; and
- How likely it is that the potentially exposed people will experience harm because of the exposures.

This manual also discusses the ecological risk assessment process, which assesses the impact of air toxics on ecological receptors such as aquatic organisms and terrestrial mammals.

This kind of information can be extremely helpful to decision makers as they try to balance the competing concerns of protecting public health, fostering economic development, and evaluating issues of fairness and equity, among others. Specifically, risk assessment can provide:

- A predictive estimate of the potential health risks posed by air toxics, which may help determine the need for action;
- A basis for determining the levels of chemicals that can be released to the air without posing unacceptable risks to public health and the environment;
- A basis for comparing potential health impacts of various pollution reduction alternatives;
- A consistent process for evaluating and documenting threats to public health and the environment from toxic air pollution; and
- A basis for comparing risks from various exposure scenarios (e.g., the risk from breathing contaminated air compared to the risk from eating contaminated food).

Performing an air toxics risk assessment is often challenging. Risk assessments can be resource and time-intensive, depending on the specific questions being asked and the level of detail needed for informed decision making. Risk assessments usually require input from a number of scientists and engineers with a variety of skills (e.g., chemistry, toxicology, statistics, modeling, meteorology, monitoring). Decision makers may also need to acquire new skills in order to understand and use the risk assessment results. Finally, although they are based on science, risk assessments often rely on the best judgment of the analysts in the face of various uncertainties.

There has not been, up to this point, a unified and comprehensive reference manual on the methods and tools that are currently available to perform air toxics risk assessments *per se*. This document is EPA's attempt to fill that void.

1.5 The Purpose of this Reference Manual

The primary purpose of this reference manual (Volume 1) is to provide, in one single place, descriptions of the major methods and technical tools that are commonly used to perform air toxics risk assessments. Specifically, the manual attempts to cover all the common basic technical approaches that are used to evaluate: how people in a particular place (e.g., a city or neighborhood) may be exposed; what chemicals they may be exposed to and at what levels; how toxic those chemicals are; and how likely it is that the exposures may result in adverse health outcomes. Topics include uncertainty and variability, basic toxicology and dose-response relationships, air toxics monitoring and modeling, emissions inventory development, and risk characterization. This manual also discusses approaches for using the results of a risk assessment in the risk management decision-making process. Links to more detailed references on each subject are presented, along with EPA contacts. Additionally, EPA's Fate, Exposure, and Risk Analysis (FERA) web site (www.epa.gov/ttn/fera) provides up-to-date tools for air toxics risk assessment, including computer models, databases, and other information used by EPA and others for air pollutant human exposure modeling, multimedia modeling, and risk.

To provide readers with a broad perspective on the potential impacts of air toxics (in addition to information on the risk assessment process), this manual also includes a discussion of a

complementary process called a **public health assessment** (PHA). One of the objectives of a PHA is to evaluate whether existing cases of illness in a community may possibly have resulted from past exposures to particular toxins (based on epidemiological principles). This process is routinely carried out at Superfund sites by the Agency for Toxic Substances and Disease Registry (ATSDR) in addition to EPA's Superfund risk assessment process. PHAs often involve the use of capabilities beyond those required for risk assessment, including medical skills. S/L/T air agencies generally will not perform such assessments themselves; however, because questions about current or past illnesses and deaths in communities often arise during the risk assessment process, information about the PHA process is offered to help S/L/T air agencies and other stakeholders understand the rudiments of the process and whom to contact for more information and help.

1.6 The Layout of this Reference Manual

This reference manual is divided into six Parts, each of which are divided further into three or more chapters. Chapters are numbered consecutively. A number of Appendices provide more detailed reference materials.

- **Part I (Background)** provides a general introduction to air toxics risk assessment and is divided into four chapters.
  - Chapter 1 (this chapter) provides an introduction to the manual.
  - Chapter 2 begins with an overview of the CAA as well as major regulations, programs, and initiatives that relate to air toxics risk reduction.
  - Chapter 3 provides an overview of risk assessment and the risk-based decision making framework, including an introduction to tiered approaches to risk assessment.
  - Chapter 4 identifies the set of chemical pollutants that are the focus of this manual and describes the general categories of air toxics sources and the primary emissions inventories (which contain information on the nature and magnitude of emissions released from various sources).

- **Part II (Human Health Risk Assessment: Inhalation)** provides a discussion of the methods and tools used to evaluate risks to human health via the inhalation pathway. It is divided into nine chapters.
  - Chapter 5 provides an overview of the inhalation risk assessment process, discusses the initial planning and scoping process that needs to be completed before the risk assessment begins, and describes the **exposure assessment**, which will usually comprise the bulk of the effort for most air toxics risk assessments.
  - Chapter 6 describes the problem formulation phase which results in the development of the conceptual model and analysis plan for the risk assessment.
  - Chapter 7 describes how to develop an emissions inventory for the risk assessment.
  - Chapter 8 discusses the factors that affect the movement and, in some cases, chemical transformation of chemicals in the atmosphere following release (i.e., the fate and transport of chemicals in the atmosphere).
  - Chapter 9 provides an overview of the use of computer modeling to predict the movement, fate, and transport of air toxics in the atmosphere. It also describes the major computer models that are commonly used for this purpose.
  - Chapter 10 provides an overview of monitoring methods that are commonly used to measure ambient concentrations of air toxics in the atmosphere.

- Chapter 11 provides information for estimating exposure concentrations for inhalation analyses, including exposure modeling.
- Chapter 12 provides an overview of **toxicity assessment** for air toxics.
- Chapter 13 provides information for completing the **risk characterization**, including uncertainty analysis and how to present the results of the risk assessment.

- **Part III (Human Health Risk Assessment: Multipathway)** provides a discussion of the methods and tools used to evaluate risks to human health when air toxics that are highly persistent or bioaccumulative are present in emissions. The focus of the multipathway risk assessment is to evaluate the potential exposures associated with ingesting soil, food, and water that has become contaminated with these chemicals after deposition from the atmosphere to surfaces, such as soils and surface waters. This Part is divided into nine chapters.
  - Chapter 14 provides an overview of the multipathway risk assessment process, discusses the initial planning and scoping process that needs to be completed before the risk assessment begins, and describes the multipathway **exposure assessment**.
  - Chapter 15 describes problem formulation for the multipathway risk assessment.
  - Chapter 16 describes how to develop an emissions inventory for the multipathway risk assessment.
  - Chapter 17 discusses the factors that affect the movement and, in some cases, chemical transformation of air toxics in soil, water, sediment, and biota.
  - Chapter 18 provides an overview of the computer modeling used to predict the movement, fate, and transport of toxics in soil, water, sediment, and biota and describes the major multimedia computer models commonly used by risk assessors.
  - Chapter 19 provides an overview of monitoring methods used to measure ambient concentrations of air toxics in soil, water, sediment, and biota.
  - Chapter 20 provides a summary of the process and assumptions used to estimate chemical intake rates – the key measure of exposure used to assess ingestion risks – including exposure modeling.
  - Chapter 21 provides an overview of the toxicity assessment for air toxics that are persistent and which may also have a high potential to bioaccumulate in food chains.
  - Chapter 22 provides information on how to complete the risk characterization for the multipathway risk assessment, including uncertainty analysis and how to present the results of the risk assessment.

- **Part IV (Ecological Risk Assessment)** provides an overview of the methods and tools used to evaluate risks to ecological receptors (e.g., birds, mammals, plants, and ecological communities) due to exposure to air toxics. This Part is divided into four chapters.
  - Chapter 23 provides an overview of the ecological risk assessment process and discusses the initial planning and scoping process that needs to be completed before the risk assessment begins.
  - Chapter 24 provides information on characterizing exposure for the ecological risk assessment.
  - Chapter 25 provides information on characterizing ecological effects, including development of the stressor-response profile.
  - Chapter 26 provides information on how to complete the risk characterization for the ecological risk assessment, including the analysis of uncertainty, and how to present the results of the ecological risk assessment.

- **Part V (Risk-Based Decision Making)** discusses the process by which the information from the risk assessment can be used to inform risk management decisions and two important aspects of that process. This Part is divided into three chapters.
  - Chapter 27 provides an overview of the risk management process, including the types of decisions that may need to be made and how the risk assessment informs the decision-making process.
  - Chapter 28 provides an overview of the importance of stakeholder involvement in the risk assessment and management process and provides information for developing and implementing a stakeholder involvement plan.
  - Chapter 29 provides information for developing and implementing a risk communication strategy for helping members of the community and the media understand the risk assessment results and how they are being used in the decision-making process.

- **Part VI (Special Topics)** provides an overview of three tools or procedures that may be used as part of performing or reporting a risk assessment.
  - Chapter 30 provides an overview of the process by which public health agencies may evaluate the public health implications posed by the emissions from air toxic sources in a community. The public health assessment, if performed, is a complementary process to risk assessment.
  - Chapter 31 discusses probabilistic risk assessment, which is aimed at describing risks as a distribution (or range) of potential outcomes.
  - Chapter 32 provides an overview of the use of Geographical Information System (GIS) tools in the process of conducting risk assessments and reporting results.

- The **Glossary** defines key terms and acronyms.

- **Appendix A** provides a listing of all HAPs along with their status as a Toxics Release Inventory (TRI) chemical, a Section 112(k) high priority urban toxic, and a Mobile Source Air Toxic.

- **Appendix B** provides a guide to the agencies and organizations that oversee air toxics regulations.

- **Appendix C** provides recommended dose-response values for cancer and noncancer effects for all HAPs.

- **Appendix D** presents the decision process by which the persistent, bioaccumulative HAP compounds (PB-HAPs) were selected.

- **Appendix E** provides an overview of all CAA designated air toxics Source Categories, including the most common HAPs in emissions, typical industries, and applicable maximum achievable control technology (MACT) standards.

- **Appendix F** provides a list of all of the specific pollutants and compound groups included in the 1999 National Emissions Inventory (NEI) along with their Chemical Abstract Services (CAS) numbers.

- **Appendix G** provides an overview of meteorology as it relates to the movement of air toxics in the atmosphere. This appendix also provides information on sources of meteorological data for modeling air toxics dispersal and transport.

- **Appendix H** discusses the process of evaluating and reducing a monitoring data set (e.g., air, water, soil sample results) into a grouping of data that are useable for exposure evaluation.

- **Appendix I** provides a general overview of how a reduced monitoring data set (developed by the methods in Appendix H) may be used to estimate exposure concentrations.

- **Appendix J** provides an overview of available air toxics monitoring methods.

- **Appendix K** provides the equations for calculating the concentrations of PB-HAPs in non-air media (e.g., soil, food, water).

1.7 The Relationship of this Manual to Volumes 2 and 3

This manual is the first volume of a three-volume set. **Volume 1: Technical Resource Manual** discusses the overall air toxics risk assessment process and the basic technical tools needed to perform these analyses. The manual addresses both human health and ecological analyses. It also provides a basic overview of the process of managing and communicating risk assessment results. Other evaluations (such as the public health assessment process) are described to give risk assessors, risk managers, and other stakeholders a more holistic understanding of the many issues that may come into play when evaluating the potential impact of air toxics on human health and the environment.

Volume 2: Facility-Specific Assessment builds on the technical tools described in Volume 1 by providing an example set of tools and procedures that may be used for source-specific or facility-specific risk assessments, including tiered approaches to source- or facility-specific risk analysis.

Volume 3: Community-Level Assessment builds on the information presented in Volume 1 to describe to communities how they can evaluate and reduce air toxics risks at the local level. The volume will include information on screening level and more detailed analytical approaches, how to balance the need for assessment versus the need for action, and how to identify and prioritize risk reduction options and measure success. Since community concerns and issues are often not related solely to air toxics, the document will also present readily available information on additional multimedia risk factors that may affect communities and strategies to reduce those risks. The document will provide additional, focused information on stakeholder involvement, communicating information in a community-based setting, and resources and methodologies that may play a role in the overall process. Note that EPA's Office of Pollution Prevention and Toxics has also developed a "Community Air Screening How To Manual" that will be available in 2004 and will be discussed in Volume 3 (Volume 3 will be available in late 2004).

References

1. U.S. Environmental Protection Agency. *Agency Mission Statement*. Updated June 11, 2002. Available at: http://www.epa.gov/history/org/origins/mission.htm. (Last accessed March 2004).

2. U.S. Environmental Protection Agency. 1999. *National Air Toxics Program: The Integrated Urban Strategy*. *Notice*. *Federal Register* 64:38705. July 19, 1999. Available at: http://www.access.gpo.gov/su_docs/fedreg/a990719c.html (the PDF file is indexed at: http://frwebgate.access.gpo.gov/cgi-bin/getdoc.cgi?dbname=1999_register&docid=99-17774-filed.pdf).

3. U.S. Environmental Protection Agency. Technology Transfer Network National Air Toxics Assessment. *The National-Scale Air Toxics Assessment*. Updated September 18, 2002. Available at: http://www.epa.gov/ttn/atw/nata/index.html. (Last accessed March 2004).

4. U.S. Environmental Protection Agency. Technology Transfer Network Air Toxics Web site. *Air Toxics Strategy: Overview*. Updated February 10, 2004. Available at: http://www.epa.gov/ttn/atw/urban/urbanpg.html (Last accessed March 2004).

Chapter 2 Clean Air Act Requirements and Programs to Regulate Air Toxics

Table of Contents

2.1 Introduction 1

2.2 HAPs and their Sources: Stationary, Mobile, and Indoor Sources 2
- 2.2.1 Hazardous Air Pollutants (HAPs) 2
- 2.2.2 Stationary Sources: The Pre-1990 CAA "Risk-Only" Approach 4
- 2.2.3 Stationary Sources and the 1990 Clean Air Act Amendments: A "Technology First, Then Risk" Approach 4
  - 2.2.3.1 Step 1: The Technology-based Approach 5
  - 2.2.3.2 Step 2: The Risk-based Approach 5
- 2.2.4 Mobile Sources of Air Toxics Rule 6
- 2.2.5 Indoor Air and Indoor Air Toxics 8
  - 2.2.5.1 Potential Sources of Indoor Air Toxics 9
  - 2.2.5.2 Indoor Air Toxics 9
  - 2.2.5.3 Health Risks and Indoor Pollutants 11

2.3 Progress in Understanding and Reducing Toxic Air Pollution 12
- 2.3.1 Trends 12
- 2.3.2 NATA National Scale Assessment 14

2.4 Other Air Pollutants of Potential Concern 16
- 2.4.1 Criteria Air Pollutants 16
- 2.4.2 Chemicals on the Toxics Release Inventory 19
- 2.4.3 Toxic Chemicals that Persist and Which Also May Bioaccumulate 20
- 2.4.4 Overlaps and Differences Between Chemical "Lists" 22

2.5 Reports to Congress on Air Toxics Issues 22
- 2.5.1 Air Toxics Deposition to the Great Waters 23
- 2.5.2 Mercury Study Report to Congress 23
- 2.5.3 Utility Report to Congress 24
- 2.5.4 Residual Risk Report to Congress 25
- 2.5.5 Integrated Urban Strategy Report to Congress 26
- 2.5.6 Other Reports 26

References 27

2.1 Introduction

In a general sense, an air pollutant is any substance introduced into the air by human activities (currently, approximately 75,000 industrial chemicals are produced or imported into the United States,[a] and science knows many millions more). Some air pollutants may take the form of solid particles, liquid droplets, or gases. Many different types of air pollutants can injure health and/or harm the environment (see Common Air Pollutants box).

Common Air Pollutants

acid aerosols
asbestos
carbon monoxide (CO)
carbonyl compounds
ground level ozone
metals
nitrogen oxides (NO_x)
particulate matter (PM)
propellants
radon
refrigerants
semivolatile organic compounds
sulfur dioxide (SO_2)
volatile organic compounds

In early versions of the Clean Air Act (CAA), Congress identified six criteria air pollutants for regulation. In addition to these pollutants, the1990 CAA Amendments focused EPA's efforts on another group of pollutants, the 188 Hazardous Air Pollutants (**HAPs**).[b] Additionally, EPA has identified 21 mobile source air toxics, 20 of which are also HAPs and the other one is "diesel particulate matter and diesel exhaust organic gases" (see Chapter 4).

The group of six criteria air pollutants occur commonly throughout the U.S. and are derived from numerous and diverse mobile and stationary sources. EPA has set National Ambient Air Quality Standards (NAAQS) for these pollutants based on health and welfare-related criteria (see Section 2.4.1 and http://www.epa.gov/ttn/naaqs/). No such national ambient air quality standards currently exist for HAPs, although regulatory programs are in place to address emissions of HAPs. In addition, air pollutants from indoor sources are of concern (with many of the chemicals emitted indoors overlapping with the criteria and HAP lists). EPA, however does not currently regulate indoor air.

A Note on Terminology

The terms "**air toxics**" and "**toxic air pollutants**" are often used interchangeably with "**hazardous air pollutants**" (which is a Clean Air Act phrase specific to the 188 pollutants that are the focal point of section 112 of the Act – see http://www.epa.gov/ttn/atw/188polls.html). For the purposes of this reference library, however, the term "air toxics" is used in the more general sense to refer generally to any air pollutant (other than criteria pollutants) that has the potential to cause adverse impacts to human health or the environment.

Criteria air pollutants are six common air pollutants determined to be hazardous to human health and for which EPA has established National Ambient Air Quality Standards (NAAQS). The six criteria air pollutants are carbon monoxide, lead, nitrogen dioxide, ozone, sulfur dioxide, and particulate matter.

The CAA is the primary federal law that regulates air emissions of HAPs. The Act applies to a number of different types of sources; these include small and large stationary facilities such as factories and neighborhood dry cleaners, as well as mobile sources such as cars and trucks. The original CAA was passed in 1963 and has been

[a] TSCA Chemical Substance Inventory, http://www.epa.gov/opptintr/newchems/invntory.htm

[b] CAA section 112(b)(1) lists 189 HAPs, but since the original Act, one chemical (caprolactam) has been delisted, leaving 188 HAPs (61 FR 30816, June 18, 1996).

amended since that time on a number of occasions, most recently in 1990. Congress intended the 1990 amendments to resolve unaddressed or insufficiently addressed air pollution problems such as acid rain, ground-level ozone, and stratospheric ozone depletion. The 1990 amendments also dramatically affected how EPA was to approach the issue of air toxics. For example, previous versions of the Act required EPA itself to identify pollutants as HAPs one-by-one and to set health-based standards for each. Given the problems that arose in working to implement this approach, Congress restructured the approach for air toxics in the 1990 amendments. The discussion below describes this current approach.

Specifically, this chapter provides an overview of the CAA requirements that are specific to HAPs, with emphasis on **stationary sources**, **mobile sources**, and **indoor sources** of HAPs. The chapter also provides insight into other aspects of air quality that play a role in understanding the air toxics problem. The chapter concludes with a brief description of some of the important studies EPA was required to perform under the Act to better understand the nature of the air toxics problem. The full text of the Act can be accessed at http://www.epa.gov/oar/caa. EPA has also developed a plain English guide to the Act that can be accessed at http://www.epa.gov/oar/oaqps/peg_caa/pegcaain.html.

2.2 HAPs and their Sources: Stationary, Mobile, and Indoor Sources

2.2.1 Hazardous Air Pollutants (HAPs)

Hazardous air pollutants (HAPs) are those 188 listed pollutants and groups of pollutants[c] that EPA knows or suspects cause cancer or other serious human health effects, such as reproductive effects or birth defects, or adverse environmental effects (Appendix A presents the full list).[1] Examples of HAPs include benzene, which is found in gasoline; perchloroethlyene, which is emitted by most dry cleaning facilities; methylene chloride, which is used as a solvent and paint stripper by a number of industries; dioxin; asbestos; toluene; and compounds of metals such as cadmium, mercury, chromium, and lead. Congress has given EPA the authority to add and subtract chemicals from that list, following established criteria [CAA Section 112(b)(3)]. According to summary data compiled by EPA, an estimated 5.1 million tons of HAPs were released from stationary and mobile sources in the U.S. in 1999.

Major Source – Any source or group of stationary sources located within a contiguous area and under common control that emits or has the potential to emit considering controls, in the aggregate, 10 tons per year (tpy) or more of any hazardous air pollutant or 25 tpy or more of any combination of hazardous air pollutants [CAA section 112(a)(1)].

Area Source – any stationary source of hazardous air pollutants that is not a major source ... not includ[ing] motor vehicles or nonroad vehicles subject to regulation under title II [CAA section 112(a)(2)].

People exposed to HAPs at sufficient concentrations and for a sufficient duration of time may have an increased chance of developing cancer or experiencing other serious health effects. These health effects can include damage to the immune system, as well as neurological,

[c] CAA section 112(b)(1) lists 189 HAPs, but since the original Act, one chemical (caprolactam) has been delisted, leaving 188 HAPs (61 FR 30816, June 18, 1996)

reproductive (e.g., reduced fertility), developmental, respiratory, and other health effects. In addition to exposure from breathing air toxics, some HAPs such as mercury compounds can deposit onto soils or surface waters, where they can be taken up by plants and animals (see Chapter 4). Like humans, ecological systems may experience adverse health problems if exposed to sufficient quantities of HAPs over time (ecological risk assessment is discussed in Part IV of this reference manual).

People may be exposed to HAPs in many ways, including:

- Breathing contaminated air;
- Eating contaminated food products, such as fish from contaminated waters; meat, milk, or eggs from animals that fed on contaminated plants; and fruits and vegetables grown in contaminated soil on which HAPs have been deposited;
- Drinking water contaminated by HAPs;
- Ingesting contaminated soil. Young children are especially vulnerable because they often ingest soil from their hands or from objects they place in their mouths; and
- Touching (making skin contact with) contaminated soil, dust, or water (for example, during recreational use of contaminated water bodies).

Anthropogenic sources of HAPs include stationary sources (e.g., factories, refineries, power plants), mobile sources (e.g., cars, trucks, buses), and indoor sources (e.g., some building materials and cleaning solvents). Some HAPs are also released from natural sources such as volcanoes.

The Urban Air Toxics

In 1999, EPA identified a group of 33 HAPs (the *Urban Air Toxics*) as those most important to health risks in urban areas (see Section 1.1).

acetaldehyde	coke oven emissions	manganese compounds
acrolein	dioxin	mercury compounds
acrylonitrile	1, 2-dibromoethane	methylene chloride[(b)]
arsenic comounds	propylene dichloride	nickel compounds
benzene	1, 3-dichloropropene	polychlorinated biphenyls (PCBs)
beryllium compounds	ethylene dichloride[(a)]	polycyclic organic mater (POM)
1,3-butadiene	ethyene oxide	quinoline
cadmium compounds	formaldehyde	1, 1, 2, 2-tetrachlorethane
carbon tetrachloride	hexachlorobenzene	tetrachloroethylene[(c)]
chloroform	hydrazine	trichloroethylene
chromium compounds	lead compounds	vinyl chloride

[(a)] also represented as 1,2-dichloroethane
[(b)] also represented as dichloromethane
[(c)] also represented as perchloroethylene

2.2.2 Stationary Sources: The Pre-1990 CAA "Risk-Only" Approach

Prior to 1990, the CAA directed EPA to regulate toxic air pollutants from stationary sources based on the risks each pollutant posed to human health. Specifically, the Act directed EPA to:

- Identify all pollutants that caused "serious and irreversible illness or death," and
- Develop standards to reduce emissions of these pollutants to levels that provided an "ample margin of safety" for the public.

In other words, EPA was tasked with identifying the chemicals to be considered HAPs and setting standards for chemical emissions that would not only be "safe," but would be safe with an "ample margin" to the public. (A discussion of what the term "ample margin of safety" means is presented in Chapter 27. A discussion as how to interpret risk levels such as "one in a million" is provided in Chapter 13.) EPA turned to a method called "risk assessment" in performing this task because it provided the tools necessary to evaluate the potential risks posed by hazardous chemicals released to the air.[(d)]

While attempting to understand and control air toxics during the 1970s and 1980s, EPA became involved in many legal, scientific, and policy debates over which pollutants to regulate and how stringently to regulate them. Much of the debate focused on what kinds of risk assessment methods to use, what assumptions in the process were appropriate, the amount of data needed to justify regulation, questions about the costs to industry and benefits to human health and the environment, and decisions about "how safe is safe" (see additional discussion in Chapter 3).

While EPA and the scientific community gained valuable knowledge about risk assessment methods during this time, the chemical-by-chemical regulatory approach – an approach based solely on risk – proved difficult. In fact, between 1970 and 1990 EPA regulated only seven pollutants (asbestos, benzene, beryllium, inorganic arsenic, mercury, radionuclides, and vinyl chloride) in this manner. Standards for sources of HAPs, known as the **National Emissions Standards for Hazardous Air Pollutants** or **NESHAPs**, cut annual air toxics emissions by an estimated 125,000 tons. However, the process did not work quickly enough to address pressing air pollution concerns.

2.2.3 Stationary Sources and the 1990 Clean Air Act Amendments: A "Technology First, Then Risk" Approach

Realizing the shortcomings of the "chemical-by-chemical" risk-based decision framework for stationary sources and acknowledging the gaps in scientific and analytical information, Congress adopted a new strategy in 1990. Specifically, Congress revised section 112 of the Act to mandate a more practical, phased approach to reducing emissions of toxic air pollutants.

---

[d]People have been assessing risk in various ways for thousands of years, so in one sense, "risk assessment" is an ancient practice. However, methods to quantitatively assess risk for specific applications are a more recent development. As noted above, the methods necessary to assess the risks posed by air toxics are an even more recent development and are the subject of this discussion.

2.2.3.1 Step 1: The Technology-based Approach

This new approach has two components. In the first phase, EPA identifies categories of stationary sources that emit large amounts of HAPs and then develops pollution reduction regulations – called **Maximum Achievable Control Technology** or **MACT** standard - for those sources.[e] The MACT standards adopted by EPA are *technology-based* (not risk-based), which means EPA requires emission reductions based on an evaluation of the emission reductions that the best-performing similar sources are already achieving.

Specifically, when developing a MACT standard for a particular source category, EPA looks at the level of emissions already being achieved by the best-performing similar sources through clean processes, control devices, work practices, or other methods. The CAA specifies baselines (often referred to as the "MACT floors") for the new standards. At a minimum, a MACT standard must achieve, throughout the industry, a level of emissions control that is at least equivalent to the MACT floor. EPA can establish a more stringent standard after considering cost, non-air quality and environmental impacts, and energy requirements (section 112(d)(2) of the CAA).

The MACT floors specified in the CAA are different for existing sources and new sources. For existing sources, the MACT floor must equal the average emissions limitations achieved by the best-performing 12 percent of sources in that source category, if there are 30 or more existing sources. If there are fewer than 30 existing sources, then the MACT floor must equal the average emissions limitation achieved by the best-performing five sources in the category. For new sources, the MACT floor must equal the level of emissions control achieved in practice by the best-controlled similar source.

EPA has issued MACT standards for a variety of industrial source categories, including chemical plants, oil refineries, aerospace manufacturers, and steel mills, and smaller sources, such as dry cleaners, commercial sterilizers, secondary lead smelters, and chromium electroplating facilities. EPA has also issued standards pursuant to section 129 of the Clean Air Act to control emissions of certain toxic pollutants from solid waste combustion facilities. A comprehensive list of final MACT rules and regulations for the MACT program can be found at http://www.epa.gov/ttn/atw/mactfnl.html. EPA's proposed timetable for finalizing the remaining standards is available at http://www.epa.gov/ttn/atw/mactprop.html. When fully implemented, all of these standards will reduce air toxics emissions by several million tons per year – more than 10 times the reductions achieved prior to 1990.

2.2.3.2 Step 2: The Risk-based Approach

In the second phase of the process, EPA reviews the technology-based MACT standards to ensure that these standards have adequately reduced risk within an "ample margin of safety." In this second assessment, the Agency must adopt additional standards to address any significant risks remaining (also called **residual risks**) after the first phase implementation of the technology-based standards (section 112(f)(2)(A) of the CAA). This time lag between the

[e] MACT standards are also considered NESHAPs.

technology and risk-based phases allows EPA to evaluate the best way to use risk assessment as a tool for assessing residual risks (see Chapter 3).

Within eight years after promulgation of MACT standards for each category or subcategory of sources, EPA must promulgate standards for such category or subcategory if the MACT standard for the category or subcategory does not protect public health with an ample margin of safety or to prevent, taking into consideration costs, energy, safety, and other relevant factors, an adverse environmental effect (section 112(f)(2)(A)). In 1999, EPA reported to Congress on its residual risk assessment framework and included a discussion of its methods, data, and tools.[2]

EPA has begun to assess residual risk for several source categories, including coke ovens, dry cleaning, gasoline distribution Stage I, commercial ethylene oxide sterilizers, halogenated solvent cleaning, industrial cooling towers, and magnetic tape manufacturing.

2.2.4 Mobile Sources of Air Toxics Rule

Mobile sources is a term used to describe a wide variety of vehicles, engines, and equipment that generate air pollution and that move, or can be moved, from place to place. Mobile sources pollute the air through combustion and fuel evaporation. These emissions contribute greatly to air pollution nationwide and are the primary cause of air pollution in many urban areas. EPA has identified 21 mobile source air toxics (MSATs) (see box below). Twenty of these are also listed as HAPs in CAA section 112(b); the remaining one (diesel particulate matter and diesel exhaust organic gases) is a mixture that includes many HAPs.[3] The two major divisions or types of mobile sources include:

- **On-road** (highway) sources include vehicles used on roads for transportation of passengers or freight. These include passenger cars, light-duty trucks (pickup trucks, minivans, passenger vans, and sport-utility vehicles), heavy-duty vehicles, and motorcycles. On-road vehicles may be fueled with gasoline, diesel fuel, or alternative fuels such as alcohol or natural gas.

- **Nonroad** (off-road) sources include vehicles, engines, and equipment used for construction, agriculture, transportation, recreation, lawn and garden care, and many other purposes. These include equipment and vehicles fueled with diesel fuel, gasoline, propane, or natural gas. Mobile sources include boats, aircraft, and locomotives. Not all mobile sources are "self-propelled." They can include portable generators, air compressors, chainsaws, trimmers, and shredders.

EPA uses an integrated approach (including regulations) to reduce pollution from mobile sources. From better engine design to better transit options, EPA's approach addresses:

- Vehicles, engines, and equipment;
- The fuels they use; and
- The people who operate them.

Mobile Source Air Toxics Listed in 2001 Rule[3]

- acetaldehyde
- acrolein
- arsenic compounds[a]
- benzene
- 1,3-butadiene
- chromium compounds[a]
- diesel particulate matter and diesel exhaust organic gases (DPM + DEOG)
- dioxin/furans[b]
- ethylbenzene
- formaldehyde
- n-hexane
- lead compounds[a]
- manganese compounds[a]
- mercury compounds[a]
- methyl tertiary butyl ether (MTBE)
- naphthalene
- nickel compounds[a]
- polycyclic organic matter (POM)[c]
- styrene
- toluene
- xylene

[a] Although the different metal compounds may differ in their toxicity, the on-road mobile source inventory contains emissions estimates for total metal compounds (i.e., the sum of all forms).
[b] This entry refers to two large groups of chlorinated compounds. In assessing their cancer risks, their quantitative potencies are usually derived from that of the most toxic, 2,3,7,8-tetrachlorodibenzodioxin.
[c] Polycyclic organic matter includes organic compounds with more than one benzene ring, and which have a boiling point greater than or equal to 100 degrees Celsius. A group of seven polynuclear aromatic hydrocarbons, which have been identified by EPA as probable human carcinogens (benz(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, chrysene, 7,12-dimethylbenz(a)anthracene, and indeno(1,2,3-cd)pyrene) are used here as surrogates for the larger group of POM compounds.

This approach includes national engine and fuel standards, as well as state requirements (e.g., engine maintenance, traffic flow/roadway design) established to enable attainment of the NAAQS for the criteria pollutants. The approach also involves extensive collaboration among EPA, state local and tribal (S/L/T) governments, transportation planners, individual citizens, and vehicle, engine, and fuel manufacturers and has been responsible for greatly reducing mobile source air pollution during the last 30 years.

In addition to achieving air toxics emissions reductions as a result of actions aimed at reductions in criteria pollutants, the 1990 CAA Amendments contain provisions specific to air toxics. These amendments direct EPA to address emissions of air toxics from motor vehicles and their fuels. Specifically, section 202(l) of the Clean Air Act instructs EPA to:

- Study the need for and feasibility of controlling emissions of toxic air pollutants associated with motor vehicles and their fuels. This section identifies benzene, 1,3-butadiene, and formaldehyde for particular consideration. EPA completed this study in 1993 and updated it in 1999.

- Set standards for HAPs from motor vehicles, their fuels, or both. Those standards are to be promulgated under section 202(a) or section 211(c) of the Act and must address at least benzene and formaldehyde. EPA is to base these standards on available technology, taking into account existing standards; costs, noise, energy, and safety factors; and lead time. EPA promulgated a rulemaking in accordance with CAA section 202(l) on March 29, 2001 (66 FR 17230).

The many vehicle and fuel changes in the last 25 years have greatly reduced air toxics emissions from highway vehicles. For example, the removal of lead from gasoline has essentially eliminated on-road mobile source emissions of this highly toxic substance in the United States. In addition, results of recent modeling indicate that current and planned programs will reduce emissions of mobile source air toxics by about one million tons (about 35 percent) between 1996 and 2007; on-highway emissions of benzene, formaldehyde, 1,3-butadiene, and acetaldehyde by 67 to 76 percent between 1990 and 2020; and on-highway diesel particulate matter by 94 percent between 1990 and 2020.[4] New cars using reformulated gasolines are capable of emitting more than 90 percent less air toxics on a per-mile basis than the uncontrolled models of 1970; new trucks and buses are designed to emit less than half the air toxics of their 1970 counterparts. Overall air toxics emissions will continue to decrease as older vehicles leave the fleets and as new regulatory programs take effect. However, the number of vehicles on the road and the number of miles they travel is continuing to grow. Without additional controls, growth in vehicle travel will offset progress in reducing air toxics.

Rulemakings and Voluntary Efforts to Reduce MSATs and other Air Pollutants

- Tier 2 gasoline/sulfur rulemaking (http://www.epa.gov/otaq/tr2home.htm)
- Reducing nonroad diesel emissions (http://www.epa.gov/nonroad/)
- Voluntary diesel retrofit program (http://www.epa.gov/otaq/retrofit)
- Best Workplaces for Commuters (http://www.commuterchoice.gov)
- Clean School Bus USA (http://www.epa.gov/cleanschoolbus.
- It All Adds Up to Cleaner Air (http://www.italladdsup.gov)

2.2.5 Indoor Air and Indoor Air Toxics

Indoor pollution sources that release gases or particles into the air are the primary cause of indoor air quality problems in homes and other buildings. Inadequate ventilation can increase indoor pollutant levels by not bringing in enough outdoor air to dilute emissions from indoor sources and by not carrying indoor air pollutants out of the building. High temperature and humidity levels can also increase concentrations of some pollutants.

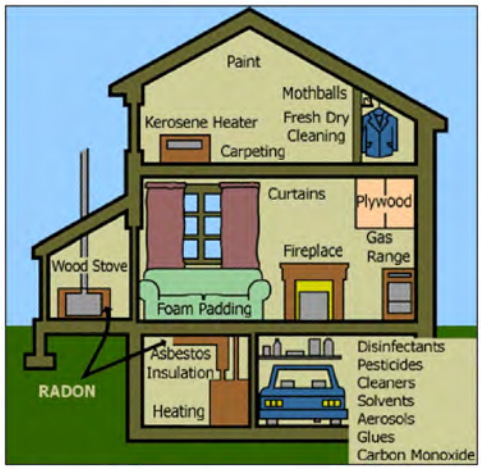
The importance of indoor air exposures to the total risk from air toxics is a relatively new finding. The contribution of indoor sources was not really recognized until the early 1980s when EPA performed the Total Exposure Assessment Methodology (TEAM) studies, which showed that the indoor concentrations of some air toxics can be significantly higher than outdoor concentrations. Since that time, numerous studies have confirmed that finding. In addition, the fact that Americans spend about 90 percent of their times indoors makes these exposures even more important

2.2.5.1 Potential Sources of Indoor Air Toxics

There are many potential sources of indoor air toxics in any home or building. These sources include combustion sources such as oil, gas, kerosene, coal, wood, and tobacco products; building materials and furnishings as diverse as deteriorated, asbestos-containing insulation, and cabinetry or furniture made of certain pressed wood products; products for household cleaning and maintenance (e.g., pesticides), personal care, or hobbies; and outdoor sources such as radon and other air pollution that penetrate into the indoor space.

Sources of Indoor Air Toxics

- Indoor air can become contaminated from numerous sources.
- Indoor air can have significantly higher concentrations of air toxics than outdoor air.
- EPA currently does not regulate indoor sources of air toxics.



The relative importance of any single source depends on how much of a given pollutant it emits and how hazardous those emissions are. In some cases, factors such as the age of the source and whether it is properly maintained are significant. For example, an improperly adjusted gas stove can emit significantly more carbon monoxide than one that is properly adjusted.

Some sources, such as building materials, furnishings, and household products like air fresheners, release pollutants more or less continuously (usually at a decreasing rate with age). Other sources, related to activities carried out in the home, release pollutants intermittently. These include smoking, the use of unvented or malfunctioning stoves, furnaces, or space heaters, the use of solvents in cleaning and hobby activities, the use of paint strippers in redecorating activities, and the use of cleaning products and pesticides in housekeeping. High pollutant concentrations can remain in the air for long periods after some of these activities.

How Does Outdoor Air Enter a House?

Outdoor air enters and leaves a house by: infiltration, natural ventilation, and mechanical ventilation. In a process known as infiltration, outdoor air flows into the house through openings, joints, and cracks in walls, floors, and ceilings, and around windows and doors. In natural ventilation, air moves through opened windows and doors. Air movement associated with infiltration and natural ventilation is caused by air temperature differences between indoors and outdoors and by wind. Finally, there are a number of mechanical ventilation devices, from outdoor-vented fans that intermittently remove air from a single room, such as bathrooms and kitchens, to air handling systems that use fans and duct work to continuously remove indoor air and distribute filtered and conditioned outdoor air to strategic points throughout the house. The rate at which outdoor air replaces indoor air is described as the air exchange rate. When there is little infiltration, natural ventilation, or mechanical ventilation, the air exchange rate is low, and pollutant levels can increase.

2.2.5.2 Indoor Air Toxics

Although EPA does not regulate indoor air pollution levels, it does take a proactive approach. The Agency provides a broad range of information about indoor air-related

risks, as well as the steps to reduce them, through the use of public awareness campaigns, guidance document dissemination, training course delivery, the operation of several linked hotlines and clearinghouses, and other outreach efforts. Useful resources on indoor air quality from the Agency are also available online.[5][6] EPA's activities to reduce exposures to indoor air toxics are many and include publishing guidelines about radon testing and result interpretation; persuading parents and caregivers of young children not to smoke indoors; and providing information to homeowners, school administrators, and office managers on the proper use of products and materials indoors, including appropriate maintenance and ventilation.

In 2001, EPA issued the Healthy Buildings, Healthy People (HBHP) report, a vision for indoor environmental quality in the 21st century.[7] The report covers three general areas: (1) why human health indoors deserves the scrutiny, concern, and action of policy makers; (2) a vision statement of EPA's vision, goals, broad strategies, and guiding principles to address indoor air quality issues; and (3) potential actions that EPA or others may pursue. The report also provides an overview of current indoor environmental program priorities in various offices within EPA and examines the roles of the Agency's partners in indoor environmental protection, including Federal, S/L/T organizations, and stakeholders.

EPA's objective is to realize major human health gains over the next 50 years by upgrading indoor environments. The Agency has set five goals and strategies to accomplish this objective:

- Achieve major health gains and improve professional education;
- Foster a revolution in the design of new and renovated buildings;
- Stimulate nationwide action to enhance health in existing structures;
- Create and use innovative products, materials, and technologies; and
- Promote health-conscious individual behavior and consumer awareness.

In addition to providing information on actions and strategies that can be taken to protect people indoors, EPA's vision acknowledges the important role individuals play in protecting their own health and the health of those around them.

EPA's specific goals to reduce the health risks from indoor air for 2005 include:

- 700,000 homes with high radon levels will be mitigated and 1 million homes with radon-resistant construction techniques will be constructed;
- The proportion of households in which children ages six and under are regularly exposed to smoking will be reduced from 27 percent in 1994 to 15 percent;
- Five percent of office buildings will be managed with indoor air quality practices consistent with EPA's *Building Air Quality* guidance;[8]
- Fifteen percent of the nation's schools will adopt good indoor air quality practices consistent with EPA's *Indoor Air Quality Tools for Schools* guidance;[9]
- One million children with asthma will have reduced exposure to indoor asthma triggers; and
- 200,000 low-income adults with asthma and 2.5 million people with asthma overall, will have reduced exposures to indoor asthma triggers.

Additional information on EPA's indoor air programs can be found EPA's Indoor Air Toxics web site.[5]

2.2.5.3 Health Risks and Indoor Pollutants

The health risks from a few indoor air toxics (e.g., radon, environmental tobacco smoke, benzene, lead, and asbestos) are well known and have been the subject of risk assessments both within and outside EPA. EPA's best estimate of annual lung cancer deaths from radon is currently about 21,000 (with an uncertainty range of 8,000 to 45,000). Environmental tobacco smoke is estimated to cause an additional 3,000 lung cancer deaths in non-smokers each year. EPA estimates that environmental tobacco smoke may also significantly aggravate symptoms of asthma for 200,000 children and may affect as many as 1,000,000 children to some extent. A California report estimates that environmental tobacco smoke causes 9,700 to 18,600 cases of low birth weight in infants each year and 35,000 to 62,000 cardiovascular deaths among non-smokers.[10]

To prioritize activities for other chemicals typically found in indoor air, EPA's Office of Radiation and Indoor Air (ORIA) is sponsoring a screening-level, risk-based analysis, which is currently in draft form and being revised. Some of the chemicals that may be of concern in indoor air, based on the draft ranking, are provided in the box to the right. However, it should be noted that the final results of this analysis may be significantly different. It should also be noted that, because monitoring data were only available for 112 chemicals and only 59 chemicals could be ranked, many chemicals found indoors might rank higher, given more complete information.

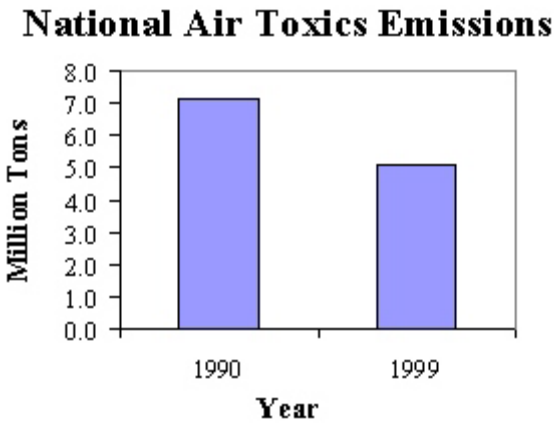
Some Pollutants of Potential Concern Indoors

- Formaldehyde
- Heptachlor
- 1,4-Dichlorobenzene
- Aldrin
- Chloroform
- Dieldrin
- Benzene
- Chlordane
- Tetrachloroethylene
- Acetaldehyde
- Trichloroethylene
- Dichlorvos
- Methylene chloride
- Lindane

Both acute and chronic cancer and noncancer health effects were addressed in the analysis, which focused on inhalation exposure only. Ten monitoring studies provided 213 concentration records for 112 air toxics including metals, aldehydes, volatile organic compounds (VOCs), and semivolatile organic compounds (SVOCs). Studied microenvironments included office buildings, residences, and schools. The general methodology used in the analysis echoed that used by the stationary source program to choose a list of urban HAPs for the Integrated Urban Air Toxics Strategy (64 FR 38706).

The study also estimated the indoor source contribution to indoor concentrations by subtracting associated outdoor concentrations from indoor concentrations. The listed pollutants were found to have large indoor source components. Note, however, that four of the listed pollutants (i.e., heptachlor, aldrin, dieldrin, and chlordane) are pesticides that are no longer in use but may continue to be of concern due to their persistence in the environment and the presence of unused and uncollected stocks.[11]

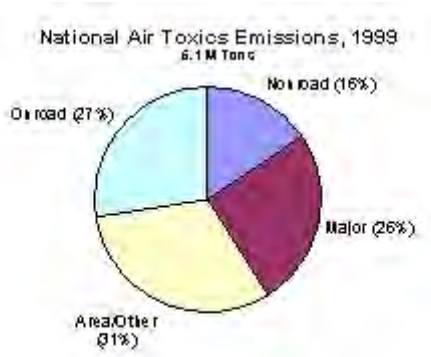
2.3 Progress in Understanding and Reducing Toxic Air Pollution



While monitoring data is critical to understanding and reducing toxic air pollution, EPA and S/L/T governments do not currently maintain as extensive a nationwide monitoring network for outdoor concentrations of air toxics as they do for many of the other pollutants (such as ozone and particulate matter). And, while EPA and S/L/T regulatory agencies do collect monitoring data for a number of toxic air pollutants, both the chemicals monitored and the geographic coverage of the monitors vary among individual S/L/T partners. EPA is working with these regulatory partners to build upon the existing monitoring sites to create a national outdoor monitoring network for a number of toxic air pollutants. The Agency's goal is to improve the scientific and technical competency of existing outdoor air monitoring networks in order to be more responsive to the public and the scientific and health communities; in this way, EPA can accommodate future needs in the face of scarce resources.

2.3.1 Trends

Monitoring data that are available can help air pollution control agencies track local trends in toxic air pollutants around the country. EPA began a pilot city monitoring project in 2001 with the intention to help answer several important national network design questions (e.g., sampling and analysis precision, sources of variability, and minimum quantitation levels). Based on the results of this year-long study and an analysis of historical monitoring data, the Agency is establishing a network of 22-city National Air Toxics Trends Sites (NATTS) that will help develop national trends for several pollutants of concern. For the latest information on national air toxics monitoring, see www.epa.gov/ttn/amtic/airtxfil.html.

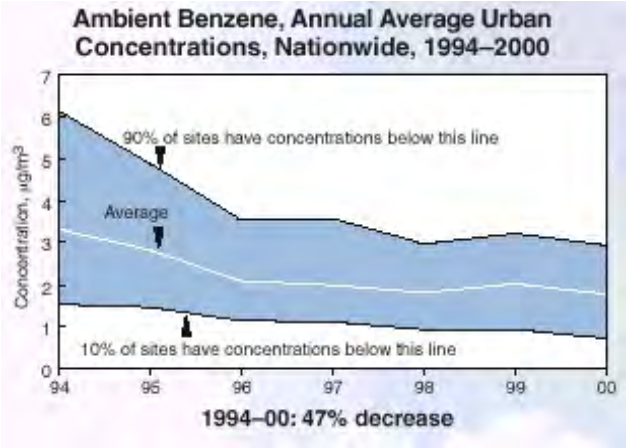


As shown in this pie chart, based on 1999 estimates (the most recent year of available data in the National Emissions Inventory (NEI) for air toxics), the emissions of HAPs are relatively equally divided between four types of sources: on road, non road, major, and area/other sources. However, this distribution varies from city to city.

Based on the data in the NEI, estimates of nationwide outdoor air toxics emissions have dropped approximately 29 percent between baseline (1990-1993) and 1999. Thirty-three of these air toxics (the Urban Air Toxics), which are considered to pose the greatest threat to public health in most urban areas, have similarly dropped 31 percent. Although changes in how EPA compiled the national inventory over time may account for some differences, EPA and S/L/T regulations, as well as voluntary reductions by industry have also achieved large reductions in air toxic emissions.

National Air Toxics Trends Stations (NAATS) Sites		
January 2003 Startup ●	**January 2004 Startup** ■	**Pilot Programs** ▲
Providence, RI Roxbury, MA New York, NY Washington, DC Decatur (Atlanta), GA Hazard, KY** Detroit, MI Deer Park (Houston), TX St. Louis, MO Bountiful, UT Grand Junction, CO** San Jose, CA Seattle, WA	Chittenden County, VT** Rochester, NY Tampa, FL Chesterfield, SC** Chicago, IL Mayville, WI Harrison County, TX** Phoenix, AZ La Grande, OR**	Barcelona/San Juan, PR Providence, RI Keeney Knob, WV Tampa, FL Detroit, MI Rio Rancho, NM Cedar Rapids, IA San Jacinto, CA Grand Junction, CO Seattle, WA ** rural site
Source: EPA's *Latest Findings on National Air Quality*[12]		

Trends for individual air toxics vary from pollutant to pollutant. Benzene, the most widely monitored toxic air pollutant, is emitted from cars, trucks, oil refineries, and chemical processes. The graph at right shows measurements of benzene taken from 95 urban monitoring sites around the country. These urban areas generally have higher levels of benzene than other areas of the country. These site measurements show, on average, a 47 percent drop in benzene levels from 1994 to 2000 (see adjacent graph). During this period,



EPA phased in new (so-called "tier 1") car emission standards, implemented the federal reformulated gasoline program in several parts of the country, and required reductions in emissions of benzene and other HAPs from oil refineries and chemical manufacturers. EPA estimates that, nationwide, benzene emissions from all sources dropped 20 percent from 1990 to 1996.

2.3.2 NATA National Scale Assessment

As part of its National Air Toxics Assessment (NATA)[f] activities, EPA has developed a national-scale risk characterization for 33 toxic air pollutants (Exhibit 2-1), based on 1996 emissions data. This set of pollutants is similar to the list of 33 Urban Air Toxics except that diesel particulate matter is included and dioxin is not. EPA used computer modeling of the 1996 NEI air toxics data as the basis for developing health risk estimates. The goal of the national-scale assessment risk characterization is to identify those air toxics which may be of potential concern in terms of contribution to population risk. The results are being used to, among other things, set priorities for the collection of additional air toxics data (e.g., emissions data and ambient monitoring data). EPA plans to update the national scale assessment every three years.

A number of important limitations and uncertainties are associated with the national scale assessment (see Summary of Limitations, Variability, and Uncertainty in the 1996 National-Scale Air Toxics Assessment box). Nonetheless, the results provide important information for priority setting. For example, the following map shows the distribution of relative predicted cancer risk attributed to exposures to outdoor sources of air toxics across the continental United States as estimated by the national-scale assessment. The highest ranking 20 percent of counties in terms of risk (622 counties) contain almost three-fourths of the U.S. population. Three air toxics (chromium, benzene, and formaldehyde) appear to pose the greatest nationwide carcinogenic risk. This map does not include the potential risk from diesel exhaust emissions because the existing health data were not deemed sufficient to develop a numerical estimate of cancer risk for this pollutant. However, exposure to diesel exhaust is widespread, and EPA has concluded that diesel exhaust is a likely human carcinogen and ranks it with the other substances that the national-scale assessment suggests pose the greatest relative risk. One toxic air pollutant, acrolein, is estimated to pose the highest potential nationwide for chronic adverse effects other than cancer. For more information about NATA activities, see www.epa.gov/ttn/atw/nata.

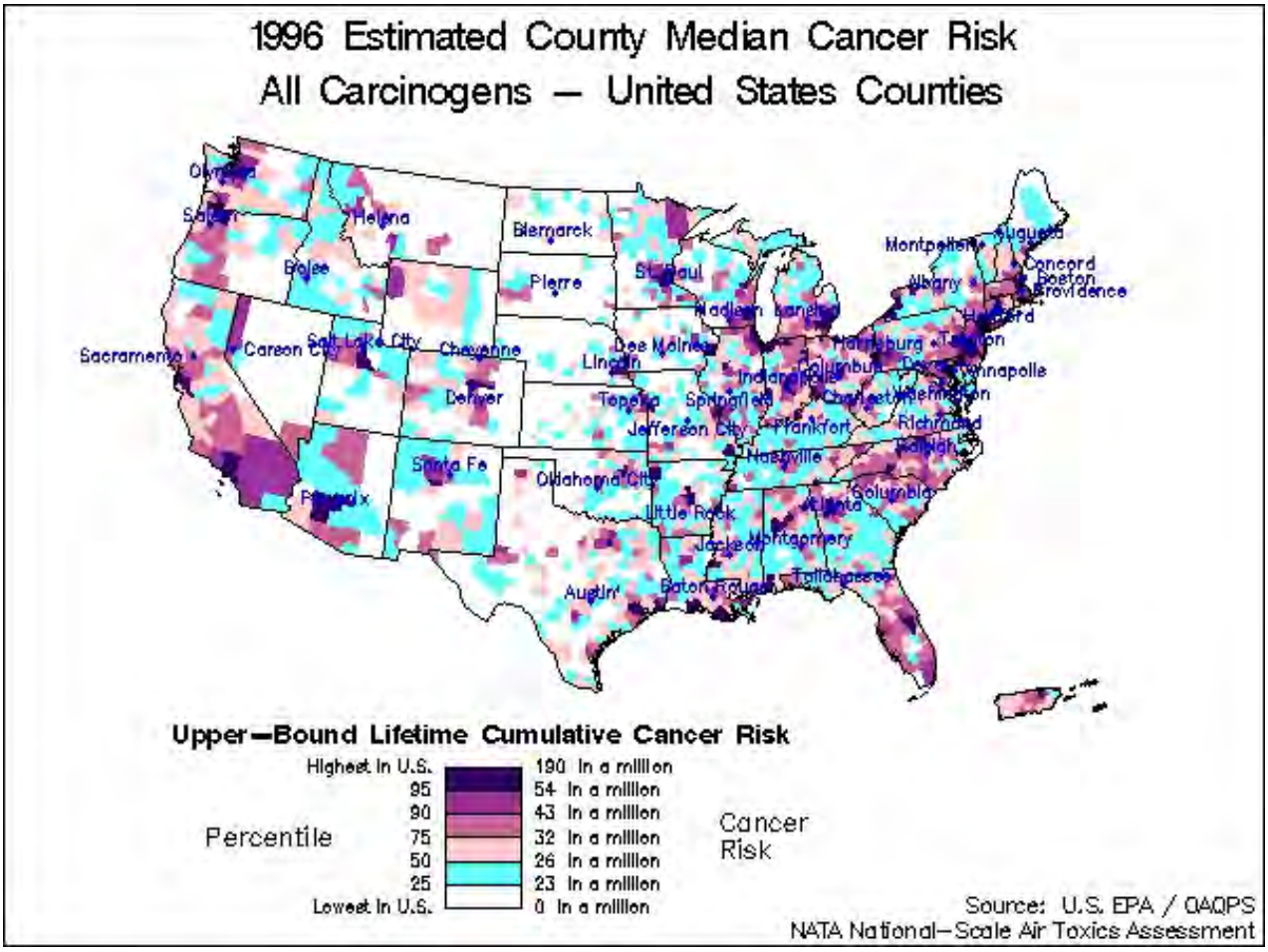
This technical assessment represents an important step toward characterizing air toxics nationwide. It is designed to help identify general patterns in air toxics exposure and risk across the country, but is not recommended as a tool to characterize or compare risk at local levels (e.g., to compare risks from one part of a city to another). More localized assessments, including monitoring and modeling, provide a more appropriate way to accurately characterize local-scale risk.

---

[f] NATA is EPA's ongoing comprehensive evaluation of air toxics in the U.S. These activities include expansion of air toxics monitoring, improving and periodically updating emission inventories, improving national- and local-scale modeling, continuing research on health effects and exposures to both ambient and indoor air, and improving assessment tools (http://www.epa.gov/ttn/atw/nata/index.html).

Exhibit 2-1. The 33 Pollutants Included in the National-Scale Air Toxics Assessment		
acetaldehyde	coke oven emissions	mercury compounds
acrolein	1,3-dichloropropene	methylene chloride
acrylonitrile	diesel particulate matter	nickel compounds
arsenic compounds	ethylene dibromide	perchlorothylene
benzene	ethylene dichloride	polychlorinated biphenyls (PCBs)
beryllium compounds	ethylene oxide	polycyclic organic matter (POM)[a]
1,3-butadiene	formaldehyde	propylene dichloride
cadmium compounds	hexachlorobenzene	quinoline
carbon tetrachloride	hydrazine	1,1,2,2-tetrachloroethane
chloroform	lead compounds	trichloroethylene
chromium compounds	manganese compounds	vinyl chloride

[a]Also represented as 7-PAH
EPA plans eventually to include all 188 HAPs in the NATA national-scale assessment
Source: http://www.epa.gov/ttn/atw/nata/34poll.html



Summary of Limitations, Variability, and Uncertainty in the 1996 National-Scale Air Toxics Assessment[a]

- **Limitations**. The NATA results provide macro-level data on emissions, ambient air concentrations, exposures, and risks across broad geographic areas (such as counties, states and the nation) at a moment in time. As such, they help the EPA identify specific air toxics compounds, and specific source sectors such as stationary sources or mobile sources, which generally produce the highest exposures and risks in the country. But the results are also based on assumptions and methods that limit the range of questions that can be answered reliably. The data cannot be used to identify exposures and risks for specific individuals, or even to identify exposures and risks in small geographic regions such as a specific census tract. Also, these data are not appropriate for determining impacts close to particular facilities. These limitations, or caveats, must always be kept in mind when interpreting the results, and the results should be used only to address questions for which the assessment methods are suited.

- **Variability**. Emissions, air concentrations, exposures and risks are not the same throughout the U.S., and are not the same for every person. Some geographic areas have higher concentrations than others; there are some periods of time when the concentration is higher at a given location than at other times. Some individuals have an exposure and/or risk below the national average, while others have an exposure and/or risk above the national average. It is necessary, therefore, to have some idea of how the ambient air toxics concentrations, exposures, and risks vary throughout the U.S. Such a process is called a variability analysis.

- **Uncertainty**. EPA seeks to protect health with reasonable confidence. Scientific estimates of air concentrations, exposures, and risks, however, always involve simplifying assumptions that make the assessment possible given available information and resources. These assumptions introduce uncertainties into the results, since there is never complete confidence that the assumptions are entirely correct. It is necessary to understand the size of these uncertainties, the level of confidence that can be placed in any statement related to the assessment, and how this confidence affects the ability to make reasoned decisions. Such a process is called an uncertainty analysis.

[a]More detailed discussion of specific limitations, variability, and uncertainty associated with the 1996 national-scale assessment is provided in three individual pages accessed by links from http://www.epa.gov/ttn/atw/nata/natsalim2.html.

2.4 Other Air Pollutants of Potential Concern

As previously noted, there are many other air pollutants that may be harmful to public health and the environment and, for some of these chemicals, other programs may already be in place to help control them. This section discusses several groups of air pollutants, some of which overlap with the list of 188 HAPs.

2.4.1 Criteria Air Pollutants

Pursuant to the CAA, EPA has set standards, also known as **National Ambient Air Quality Standards (NAAQS)**, for six **criteria pollutants** (Exhibit 2-2). The Clean Air Act requires these standards to be set at levels that protect public health with an adequate margin of safety and without consideration of cost. These standards serve two important purposes: first, they provide information to the public about whether the air in their community is healthful; and second, they

present state and local governments with the targets they must meet to achieve clean air. EPA requires that each state containing areas that do not attain the standards develop a written plan for cleaning the air in those areas. The plans developed are called state implementation plans (SIPs). Through these plans, the states outline efforts that they will make to try to correct the levels of air pollution and bring their areas back into attainment.

Exhibit 2-2. National Ambient Air Quality Standards (NAAQS)

Pollutant	Standard Value*		Standard Type
Carbon Monoxide (CO)			
8-hour Average	9 ppm	(10 mg/m^3)	Primary
1-hour Average	35 ppm	(40 mg/m^3)	Primary
Nitrogen Dioxide (NO_2)			
Annual Arithmetic Mean	0.053 ppm	(100 μg/m^3)	Primary & Secondary
Ozone (O_3)			
1-hour Average	0.12 ppm	(235 μg/m^3)	Primary & Secondary
8-hour Average	0.08 ppm	(157 μg/m^3)	Primary & Secondary
Lead (Pb)			
Quarterly Average	1.5 μg/m^3		Primary & Secondary
Particulate (PM_{10}) *Particles with diameters of 10 micrometers or less*			
Annual Arithmetic Mean	50 μg/m^3		Primary & Secondary
24-hour Average	150 μg/m^3		Primary & Secondary
Particulate ($PM_{2.5}$) *Particles with diameters of 2.5 micrometers or less*			
Annual Arithmetic Mean	15 μg/m^3		Primary & Secondary
24-hour Average	65 μg/m^3		Primary & Secondary
Sulfur Dioxide (SO_2)			
Annual Arithmetic Mean	0.030 ppm	(80 μg/m^3)	Primary
24-hour Average	0.140 ppm	(365 μg/m^3)	Primary
3-hour Average	0.500 ppm	(1300 μg/m^3)	Secondary

* Parenthetical value is an approximately equivalent concentration

Four of these pollutants (CO, Pb, NO_2, and SO_2) result primarily through direct emissions from a variety of sources. PM results from direct emissions, but is also commonly formed from emissions of nitrogen oxides (NO_x), sulfur oxides (SO_x), ammonia, organic compounds, and other gases in the atmosphere. Sources of fine particles ($PM_{2.5}$) include many types of combustion activities (e.g., motor vehicles, power plants, wood burning) and certain industrial processes. Ozone is not directly emitted from sources, but is formed when NO_x and VOCs react in the presence of sunlight.

Exposure to the criteria pollutants is associated with numerous effects on human health, including increased respiratory symptoms, hospitalization for heart or lung diseases, and even premature death. The CAA established two types of NAAQS for the criteria pollutants:

- **Primary standards** are designed to establish limits to protect public health, including the health of sensitive populations such as asthmatics, children, and the elderly.

- **Secondary standards** set limits to protect public welfare, including protection against visibility impairment and adverse effects on crops, vegetation, and building materials.

Many of the health effects associated with the criteria pollutants can happen within a few hours or days after breathing polluted air. Thus, EPA has developed an index, called the Air Quality Index or AQI, for reporting daily air quality. The AQI can be thought of as a yardstick that runs from 0 to 500. The higher the AQI value, the greater the level of air pollution and the greater the health danger. For example, an AQI value of 50 represents good air quality and little potential to affect public health, while an AQI value over 300 represents hazardous air quality. Most States now provide this information to their citizens on either their own website or through the EPA's AirNow website (http://www.epa.gov/airnow/where/).

Air Quality Index

Air Quality Index (AQI) Values	Levels of Health Concern	Colors
0 to 50	Good	Green
51 to 100	Moderate	Yellow
101 to 150	Unhealthy for sensitive groups	Orange
151 to 200	Unhealthy	Red
201 to 300	Very Unhealthy	Purple
301 to 500	Hazardous	Maroon

Despite the progress made in the last 30 years, millions of people live in counties in which monitoring data show unhealthy air for one or more of the six criteria pollutants. EPA's most recent evaluation of air pollution trends for these six pollutants can be found at http://www.epa.gov/airtrends/. General information on the criteria pollutants can be found at http://www.epa.gov/air/urbanair/6poll.html.

2.4.2 Chemicals on the Toxics Release Inventory

In 1984, a cloud of methyl isocyanate released from an accident at a pesticide plant in Bhopal, India, killed thousands of people. Shortly thereafter, there was a serious chemical release at a sister plant in West Virginia. These incidents underscored the needs of industrial workers and communities for more complete information on hazardous materials. Public interest and environmental organizations around the country increased demands for information on toxic chemicals being released "beyond the fence line" – outside of the facility. In response, Congress enacted the Emergency Planning and Community Right-to-Know Act (EPCRA) in 1986. Shortly thereafter, the CAA Amendments of 1990 required EPA to publish regulations and guidance for chemical accident prevention at facilities using extremely hazardous substances (see box below).

Risk Management Planning: Accidental Release Prevention

The CAA Amendments of 1990 required EPA to publish regulations and guidance for chemical accident prevention at facilities using extremely hazardous substances. The Risk Management Program Rule was written to implement section 112(r) of these amendments. The rule, which built upon existing industry codes and standards, requires companies of all sizes that use certain flammable and toxic substances to develop a Risk Management Program, which includes a(n):

- Hazard assessment that details the potential effects of an accidental release, an accident history of the last five years, and an evaluation of worst-case and alternative accidental releases;
- Prevention program that includes safety precautions and maintenance, monitoring, and employee training measures; and
- Emergency response program that spells out emergency health care, employee training measures, and procedures for informing the public and response agencies (e.g., the fire department) should an accident occur.

A summary of each facility's risk management program (known as a "Risk Management Plan" or "RMP") was to be submitted to EPA by 1999 and must be revised and resubmitted every five years.

The List of Regulated Substances under section 112(r) of the Clean Air Act is found in 40 CFR Part 68 and lists the regulated substances, including their synonyms, and threshold quantities (in pounds) to help facilities assess if they are subject to the RMP rule or the general duty clause (see http://www.access.gpo.gov/nara/cfr/cfrhtml_00/Title_40/40cfr68_00.html). Note that pursuant to section 112(r), threshold quantities for RMPs, are of *amounts stored on site* and not emissions. Additional information on the Risk Management Program can be found at: http://yosemite.epa.gov/oswer/ceppoweb.nsf/content/RMPS.htm

EPCRA's primary purpose is to inform communities and citizens of chemical hazards in their areas. Sections 311 and 312 of EPCRA require businesses to report the locations and quantities of chemicals stored on-site as a means of helping communities prepare for chemical spills and similar emergencies. EPCRA section 313 requires EPA and the states to annually collect data on releases and transfers of listed toxic chemicals from certain industrial facilities, and make the data available to the public in the Toxics Release Inventory (TRI). In 1990, Congress passed the Pollution Prevention Act which required that additional data on waste management and source reduction activities also be reported in the TRI. One of the goals of the TRI is to empower citizens, through information, to hold companies and local governments accountable for the management of toxic chemicals.

The TRI program has expanded significantly since its inception in 1987. The Agency has issued rules to roughly double the number of chemicals that the TRI includes to over 650. The TRI has added seven new industry sectors, expanding coverage significantly beyond manufacturing industries. Most recently, the Agency has reduced the reporting thresholds for certain persistent, bioaccumulative, and toxic (PBT) chemicals (discussed in Chapter 4) in order to provide additional information to the public on these chemicals. A full list of the TRI chemicals, along with information on accessing the database and health and environmental effects information, can be found at http://www.epa.gov/tri/.

2.4.3 Toxic Chemicals that Persist and Which Also May Bioaccumulate

Toxic chemicals that persist and which also may bioaccumulate are compounds that can build up in the food chain to levels that are harmful to human and ecosystem health. Such chemicals, commonly called PBT chemicals, may be associated with a range of adverse human health effects, including effects on the nervous system, reproductive and developmental problems, cancer, and genetic impacts. EPA's challenge in reducing risks from these chemicals stems from the pollutant's ability to transfer easily between air, water, and land; to linger for generations in people and the environment; and in some cases to travel long distances. A number of "lists" of these chemicals have been developed through international and EPA efforts (see Chapter 4).

Over the years, much work has been done to reduce the risk associated with these chemicals. However, the nation still finds PBT chemicals in the air, water, land, and, as a result, food. For example, the total number of advisories for eating contaminated fish in the United States increased by 93 percent from 1993 to 2002.[13] Although there are advisories for a total of 39 chemical contaminants, most advisories involve five primary contaminants: mercury, PCBs, dioxins, DDT, and chlordane. Almost 75 percent of the advisories have been issued at least in part because of mercury contamination. The 2,800 advisories issued in 2002 represent approximately 33 percent of the nation's total lake acreage and over 15 percent of the nation's total river miles.

Until the late 1990s, EPA actions to reduce emissions of toxic chemicals that persist and which also may bioaccumulate have been separate regulatory activities aimed at pollutant releases to individual environmental media (air, water, or land). In 1998, EPA developed a **PBT Strategy** to better coordinate these actions and to assure, for example, that regulations removing a pollutant from the air do not inadvertently result in transferring it to the land or water (http://www.epa.gov/opptintr/pbt/). The main goals of the strategy are to:

- Develop and implement national action plans to reduce priority PBT pollutants, utilizing the full range of EPA tools;
- Continue to screen and select more priority PBT pollutants for action;
- Prevent new PBTs from entering the marketplace; and
- Measure progress of these actions against the Government Performance and Results Act (GPRA) goals and national commitments.

Photo from: Charles A. Giannetta

The Agency-wide strategy enables EPA to harness all of its tools – voluntary, regulatory, international, enforcement, compliance, and research – and direct them at a set of priority pollutants of common concern to all EPA program offices. Implementing the strategy will require time and the coordination of many EPA offices as well as other stakeholders, such as industry, other governmental groups, and the international community.

International Transport of Air Pollutants

There is the potential for toxic chemicals that persist and which also may bioaccumulate to be transported from long distances to contaminate distant regions of the globe. An investigation by EPA Region 5 has shown the possibility of long-range transport of certain of these chemicals (identified in an international treaty as "persistent organic pollutants," or POPs – see Chapter 4) which were used in Central America prior to the 1980s to impact the Great Lakes. This is due to several phenomena. The semi-volatility of many POPs, allows them to be volatilized from warmer regions of the globe and redeposited in cooler regions in higher latitudes. Additionally, meteorological patterns during certain times of year can transport air masses and pollutants from the Central American region though the central U.S. into the northern states. Air masses from Central America have an unobstructed path to the Great Lakes (e.g. no physical barriers such as mountain ranges). Satellite photos show the transport of smoke from Central American fires in May of 1998 up through the Great Lakes Region.

This figure illustrates the mean wind flow at 1500 meters of altitude during the months of June, July and August from 1985 to 1996. Although these patterns can be disrupted by climatological events such as El Niño, it is clear that POPs released in the southern areas of this hemisphere can impact areas of the U.S. Studies have shown that long range transport from many regions of the globe is a significant source of POP chemicals to the Great Lakes and that mitigation efforts are going to be needed both in the U.S. and globally to address potential sources. The study of Central American sources has shown that this region is a potential contributor to POPs contamination in the Great Lakes, due to the fact that these chemicals degrade very slowly, and there still exist areas of high contamination and stockpiles of these chemicals that are no longer in use in Cental America.

For more information on International Issues & U.S. Air Quality, see EPA's Air Trends website at http://www.epa.gov/airtrends/international.html

2.4.4 Overlaps and Differences Between Chemical "Lists"

The various lists of chemicals discussed above (e.g., HAPs, criteria air pollutants, TRI chemicals) do not always treat groups of chemicals (or chemical precursors/reaction products) in the same manner. Some examples of the ways in which these lists overlap or differ include:

- "Glycol ethers" are defined differently for the TRI and as HAPs (see box below);
- Ozone is formed by the interaction of NO_X, VOCs, and sunlight. Some of the HAPs are VOCs that may contribute to ozone formation;
- "Particulate matter" that is regulated as a criteria pollutant can be comprised of any number of individual chemicals and may contain various HAPs.

Glycol Ethers in the TRI and as HAPs

The TRI includes certain glycol ethers R-(OCH2CH2)n -OR' where:
n = 1, 2, or 3
R = alkyl C7 or less; phenyl or alkyl substituted phenyl
R' = H, or alkyl C7 or less
OR' consisting of carboxylic acid ester, sulfate, phosphate, nitrate, or sulfonate.

The list of HAPs includes mono- and di- ethers of ethylene glycol, diethylene glycol, and triethylene glycol R-(OCH2CH2)n -OR' where:
n = 1, 2, or 3
R = alkyl or aryl groups
R' = R, H, or groups which, when removed, yield glycol ethers with the structure: R-(OCH2CH)n-OH.
Polymers (surfactant alcohol ethoxylates and their derivatives)are excluded from the glycol category.

It is important to keep these overlaps and differences in mind since they can have important legal, policy, and other practical implications when studying air toxics impact or developing risk reduction alternatives for a particular location. The reader should also remember that the differences among chemical "lists" are based mostly on legal and regulatory considerations, not necessarily on toxicologic properties.

2.5 Reports to Congress on Air Toxics Issues

The CAA requires EPA to study and produce reports on several specific topics relevant to our understanding of air toxics and the risks they pose to human health. These studies have been critical to our understanding of important air toxics sources and how certain chemicals move through and impact our environment. A synopsis of several of these studies is presented below. Links to all of the various reports can be found at http://www.epa.gov/ttn/oarpg/t3rc.html.

2.5.1 Air Toxics Deposition to the Great Waters

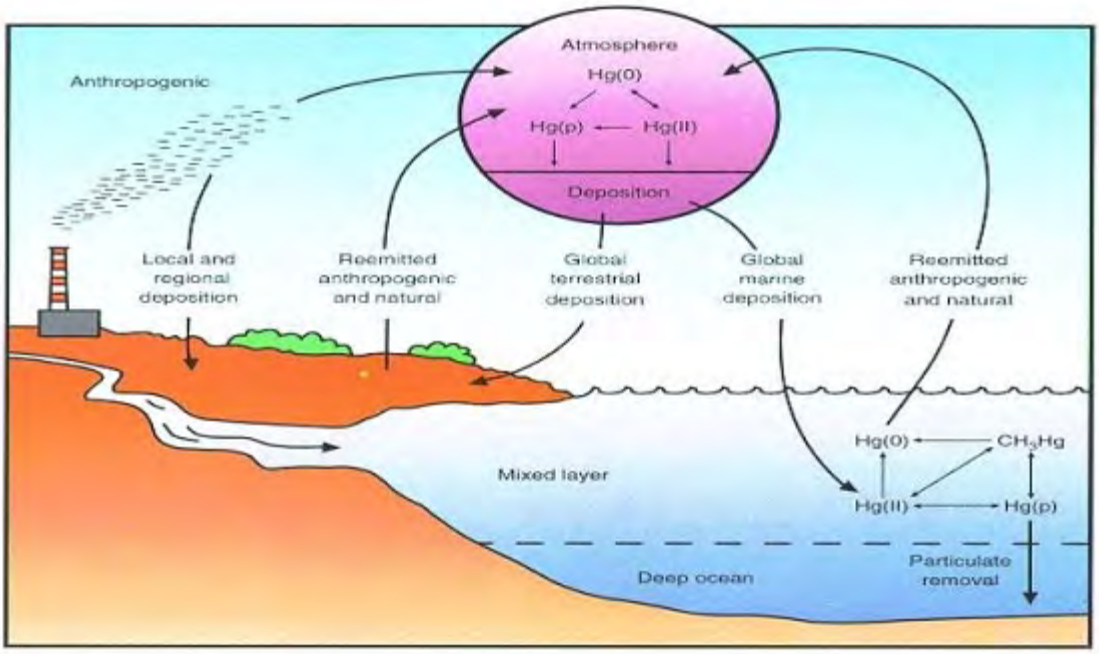
Pursuant to section 112(m) of the CAA, EPA, in conjunction with the National Oceanic and Atmospheric Administration (NOAA), has issued three reports to Congress on the deposition of air toxics and the resulting effects on the Great Lakes, Chesapeake Bay, Lake Champlain, and certain other coastal waters, collectively known as the **Great Waters**. In addition to EPA and NOAA, other international, national, regional, and local organizations also contribute to the body of science relevant to the Great Waters program and are engaged in activities that seek to reduce sources and quantities of pollution to the Great Waters. These activities focus on 15 **pollutants of concern**, including certain pesticides, metal compounds, chlorinated organic compounds, and nitrogen compounds. These pollutants enter the air in a variety of ways, including direct emission from industries and natural sources, and "re-emission" from soil and water. The Agency selected pollutants of concern due to their persistence, potential to bioaccumulate, and/or potential for adverse impacts to the Great Waters. Some of these pollutants are also likely **endocrine disruptors**, meaning they may interfere with the action of hormones in wildlife and humans. EPA will work to increase public awareness of risks of exposure to Great Waters pollutants as well as continue to support the development of modeling tools that address the transport and fate of pollutants in ecosystems and characterize risk, including research to clarify mechanisms of mercury methylation so as to better predict and manage ecosystems at risk. The most recent Great Waters Report to Congress is available at http://www.epa.gov/airprogm/oar/oaqps/gr8water/.

2.5.2 Mercury Study Report to Congress

Mercury compounds are one of the 188 HAPs. They are of concern because they persist in the environment, and bioaccumulate in food, and are associated with serious health and environmental effects, including neurological impacts in infants. Coal-fired electric utility plants are the largest air emission sources of mercury in the U.S. (responsible for approximately 40 percent of 1999 emissions). Resultant mercury concentrations in air are usually low and of little direct concern. However, when mercury enters surface waters, biological processes transform it to a highly toxic form that accumulates in fish, which can result in large exposures to fish consumers (including people). (See following graphic.)

EPA prepared the 1997 Mercury Study as a Report to Congress pursuant to the requirements of section 112(n)(1)(B) of the CAA to provide an assessment of the magnitude of U.S. mercury emissions by source, the health and environmental implications of those emissions, and the availability and cost of control technologies. As the state-of-the-science for mercury is continuously and rapidly evolving, this Report represents a "snapshot" of our understanding of mercury. This Report does not quantify the risk from mercury exposure because of scientific uncertainty in a number of important areas. The Report identifies areas where further research is needed to provide a quantitative risk assessment. The full Report can be accessed at http://www.epa.gov/ttn/atw/112nmerc/mercury.html.

Mercury Cycling in the Environment



2.5.3 Utility Report to Congress

Section 112(n)(1)(A) of the 1990 CAA Amendments required EPA to conduct a study of the public health impacts of emissions of air toxics from electric utilities that burn fossil fuel. Utility emissions include 67 HAPs, including arsenic compounds, nickel compounds, chromium compounds, radionuclides, and mercury compounds. EPA has presented the results of these studies in two key documents, a 1998 Report to Congress and a 1999 analysis of emissions reduction options. The key findings of the report to Congress include:

- **Air Toxics Emissions of Concern**. The report indicates that, although uncertainties in the analysis exist, on balance, mercury from coal-fired utilities is the hazardous air pollutant of greatest potential public health concern. Three other air toxics are identified, for which there are some potential concerns and uncertainties that may need further study: dioxins, arsenic, and nickel.

- **Risk Assessment of Exposure Pathways Other Than Inhalation**. The assessment determined that exposures due to non-inhalation routes (i.e., dermal, ingestion) are by far the most important routes of exposure for mercury and dioxins. For arsenic and radionuclides, both inhalation and ingestion appear to be important exposure routes. However, there are uncertainties and limitations in the data that indicate a need for further evaluation to more fully characterize the public health impacts of these pollutant emissions from utilities.

- **Inhalation Exposure Assessment**. The modeling assessment suggests that a substantial fraction of the utility emissions are dispersed well beyond the local area due to the nature of the emissions (mostly fine particulate substances) and the height of the tall stacks. Assessment of inhalation exposure for the 67 air toxics emitted by utilities indicate that the cancer risk from inhalation exposure is estimated to be less than one in a million for the majority of utility plants, with a few plants perhaps with slightly greater risks. Further research and evaluation may be needed to more comprehensively assess the inhalation cancer risks.

- **Mercury**. The results of the investigation indicate that mercury from coal-fired utilities is the air pollutant of greatest potential concern to public health from utilities. Coal-fired utilities are estimated to emit about one-third (52 tons) of U.S. anthropogenic (manmade) mercury emissions per year. The risk assessment indicates that ingestion of contaminated fish is the most important route of exposure to mercury. The modeling assessment, in conjunction with available scientific data, provides evidence for a plausible link between emissions of mercury from utilities and the methylmercury found in soil, water, air, and fish. Consequently, mercury emissions from coal-fired utilities may contribute to the potential exposures to mercury through consumption of contaminated fish. However, there remain uncertainties about the extent of impacts directly attributable to mercury emissions from utilities.

- **Alternative Control Strategies**. There are numerous potential alternative control technologies and strategies for air toxics control, although the feasibility and effectiveness of potential control technologies vary.

2.5.4 Residual Risk Report to Congress

The Residual Risk Report to Congress responds to section 112(f)(1) of the Clean Air Act, which requires EPA to investigate and report to Congress on a variety of topics pertaining to the assessment of residual risks associated with air toxics emissions from stationary sources remaining after the implementation of technology based standards per section 112(d) (i.e., MACT standards).[g]

While the main purpose of the Report is to describe the methods and the framework that EPA will use to make residual risk determinations, the Report also discusses, in general terms, the available methods of reducing residual risks - including pollution prevention, add-on controls, and voluntary approaches - and factors relevant to costs of these methods; the current state of knowledge regarding health effects of air toxics on humans; and EPA's current methods for collecting and assessing health effects data.

---

[g]As touched on in Section 2.2.3.2, section 112(f) of the CAA requires the Agency to consider the need for additional standards following regulation under section 112(d) to protect public health and the environment. Section 112(f) of the CAA specifies that such residual risk standards "provide an ample margin of safety to protect public health." Section 112(f) also requires EPA to determine whether residual risk standards are necessary to prevent "an adverse environmental effect" taking into consideration "costs, energy, safety, and other relevant factors" in deciding what level is protective.

While developed in response to Clean Air Act provisions particular to "residual risk," the report describes methodologies intended for EPA's use more broadly in assessing risk from toxic air pollutants. The Report does not specify a particular method for conducting risk assessments, stressing that EPA has the flexibility to use current techniques along with new methods as they are developed. The full report is available at http://www.epa.gov/ttncaaa1/t3/reports/risk_rep.pdf.

Specifically, the *Residual Risk Report to Congress*[2] identifies two objectives for residual risk activities:

- Assess any risks remaining after MACT standard compliance; and
- Set standards for the identified source categories, if additional HAP emission reductions are necessary to provide an ample margin of safety to protect public health or, taking into account cost, energy, safety, and other relevant factors, to prevent an adverse environmental effect.

2.5.5 Integrated Urban Strategy Report to Congress

The Strategy addresses the need to reduce emissions of air toxics in urban areas and looks collectively at large and small industrial and commercial operations, as well as mobile sources of pollution. The Strategy also includes plans for improving current understanding of the health risks posed by toxics in urban areas. This Report to Congress provides the following: a more detailed examination of the methodologies used for selecting the 33 initial urban air toxics identified in the Strategy; a summary of recent risk assessments conducted in several urban areas; and a detailed discussion of research needs to achieve the goals outlined in the Strategy. These needs were identified in the following areas: exposure assessment, health effects, dose-response assessment, risk assessment, risk characterization, and risk management. The report is available at http://www.epa.gov/ttnatw01/urban/natprpt.pdf.

2.5.6 Other Reports

Finally, EPA prepared two other reports that were called for in the Clean Air Act (http://www.epa.gov/ttn/atw/112npg.html).

First, section 112(n)(5) of the CAA required EPA to assess the public health hazards associated with emissions of hydrogen sulfide from oil and gas extraction. This report, *Hydrogen Sulfide Air Emissions Associated with the Extraction of Oil and Natural Gas* (EPA-453/R-93-045), is available from the National Technical Information Services (NTIS) as publication number PB94-131224.

Second, section 112(n)(6) of the CAA required EPA to assess the public health hazards associated with emissions of hydrofluoric acid in areas that do not have comprehensive health and safety regulations addressing hydrofluoric acid. The *Hydrogen Fluoride Study: Report to Congress* (EPA 550-R-93-001) was published in September 1993 and is available from NTIS as publication number PB 94-121308.

References

1. U.S. Environmental Protection Agency. 2004. Technology Transfer Network Air Toxics Website *The Original List of Hazardous Air Pollutants*. Updated January 9, 2004. Available at: http://www.epa.gov/ttn/atw/188polls.html (Last accessed March 2004).

2. U.S. Environmental Protection Agency. 1999. *Residual Risk Report to Congress*. Office of Air Quality Planning and Standards, Research Triangle Park, NC. March 1999. EPA/453/R99/001. Available at http://www.epa.gov/ttnatw01/risk/risk_rep.pdf.

3. U.S. Environmental Protection Agency. 2001. *Final Rule to Control Emissions of Hazardous Air Pollutants from Mobile Sources*. *Final Rule. Federal Register* 66:17230, March 29, 2001. Available at http://www.epa.gov/otaq/regs/toxics/toxicfrm.pdf.

4. U.S. Environmental Protection Agency. 2000. *Technical Support Document: Control of Emissions of Hazardous Air Pollutants from Motor Vehicles and Motor Vehicle Fuels.* Office of Transportation and Air Quality, December 2000. EPA/420/R00/023. Available at: http://www.epa.gov/otaq/toxics.htm.

5. U.S. Environmental Protection Agency. 2004. Air - Indoor Air Quality (IAQ). *Indoor Air Quality*. Updated March 2, 2004. Available at http://www.epa.gov/iaq/. (Last accessed March 2004).

6. U.S. Environmental Protection Agency. 2004. Indoor Air. *Publications list.* Updated February 17, 2004. Available at:http://www.epa.gov/iaq/pubs/index.html. (Last accessed March 2004).

7. U.S. Environmental Protection Agency. 2001. *Healthy Buildings, Healthy People: A Vision for the 21st Century.* Office of Air and Radiation, Washington, D.C. EPA/402/K01/003. Available at http://www.epa.gov/iaq/hbhp/index.html.

8. U.S. Environmental Protection Agency. 1991. *Building Air Quality: A Guide for Building Owners and Facility Managers.* Office of Air and Radiation, Washington, D.C. EPA/402/F91/102. Available at http://www.epa.gov/iaq/largebldgs/baqtoc.html.

9. U.S. Environmental Protection Agency. 2003. *Indoor Air Quality Tools for Schools Communication Guide*. Office of Air and Radiation, Washington, D.C. EPA/402/K02/008. Available at http://www.epa.gov/iaq/schools/images/communication_guide.pdf.

10. National Cancer Institute. 1999. *Health Effects of Exposure to Environmental Tobacco Smoke: The Report of the California Environmental Protection Agency*. Smoking and Tobacco Control Monograph No.10. Bethesda, MD: National Cancer Institute, National Institutes of Health, U.S. Department of Health and Human Services. NIH Publication No. 99-4645.

11. Johnston PK, Hadwen G, McCarthy J, Girman GR. 2002. A screening-level ranking of toxic chemicals at levels typically found in indoor air. In: Levin H, ed., *Proceedings: Indoor Air 2002, 9th International Conference on Indoor Air Quality and Climate*, Monterey, CA, June 30-July 5, 2002. Vol. 4, pp. 930-935.

12. U.S. Environmental Protection Agency. 2003. *Latest Findings on National Air Quality; 2002 Status and Trends.* Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina. EPA 454/ K-03-001. Available at: http://www.epa.gov/air/airtrends/2002_airtrends_final.pdf

13. U.S. Environmental Protection Agency. 2003. *Updated Listing of National Fish and Wildlife Advisories*. Office of Water, Washington, D.C. June 4, 2003. Available at: http://www.epa.gov/waterscience/fish/.

Chapter 3 EPA's Risk Assessment Process for Air Toxics: History and Overview

Table of Contents

3.1 Introduction 1

3.2 A Short History of the Development of Human Health Risk Assessment and Risk Management Approaches for Air Toxics 1
- 3.2.1 The 1983 National Academy of Sciences Report 1
- 3.2.2 The 1994 National Research Council Report 3
- 3.2.3 The CRARM 4
- 3.2.4 Development of Human Health Risk Assessment at EPA 6

3.3 Air Toxics Human Health Risk Assessment: Overview of the Process 9
- 3.3.1 Air Toxics Risk Assessment: What Is the Question? 10
- 3.3.2 Air Toxics Risk Assessment: The Process 12
  - 3.3.2.1 Planning, Scoping, and Problem Formulation 13
  - 3.3.2.2 Analysis Phase 14
  - 3.3.2.3 Risk Characterization 15
- 3.3.3 Tiered Assessment Approaches 16

3.4 Uncertainty and Variability in Air Toxics Risk Assessment 18
- 3.4.1 Distinguishing Uncertainty and Variability 19
- 3.4.2 Sources of Uncertainty in Air Toxics Risk Assessment 20
- 3.4.3 Sources of Variability in Air Toxics Risk Assessment 22
- 3.4.4 Characterizing Uncertainty and Variability 23
- 3.4.5 Tiered Approach to Uncertainty and Variability 24
- 3.4.6 Assessment and Presentation of Uncertainty 26

References 29

3.1 Introduction

This chapter provides the historical backdrop to the air toxics risk assessment process that is in use at EPA today. It examines the overall framework of the risk assessment process and how the various elements of the process relate to one another, including resource and timing considerations. Subsequent chapters of this reference manual describe each of the specific elements of the risk assessment process in detail.

3.2 A Short History of the Development of Human Health Risk Assessment and Risk Management Approaches for Air Toxics

Risk assessment is not new. However, only recently have some attempted to formalize the process into a coherent framework. This section briefly describes the chronology and important events in the development of those risk assessment methodologies outlined in this document.

3.2.1 The 1983 National Academy of Sciences Report

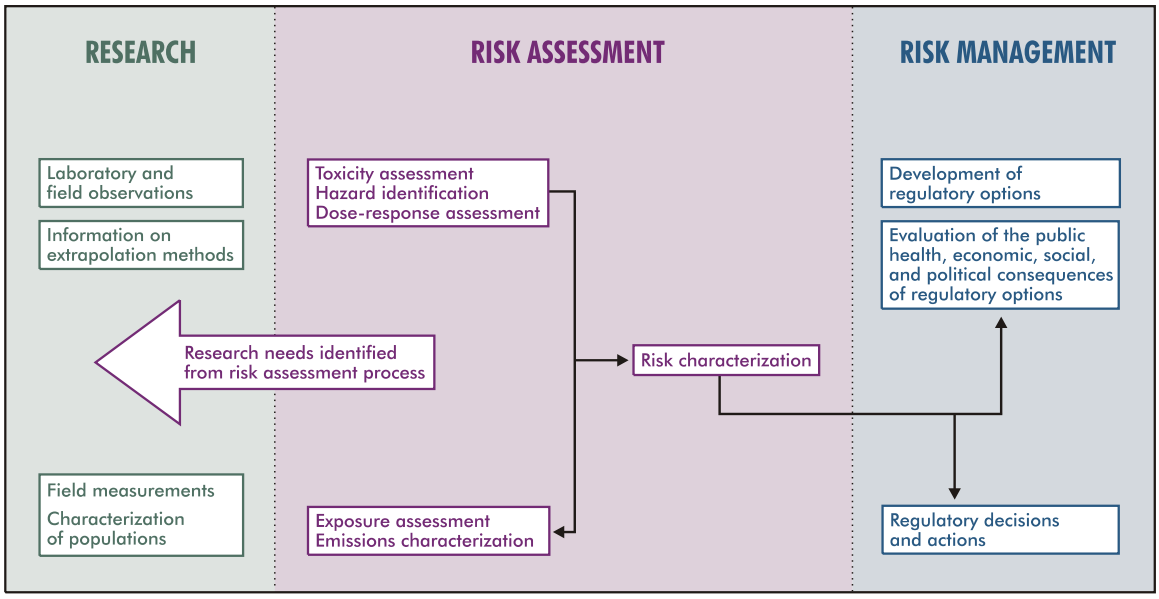
In the 1980s, the emerging practice of federal-level risk assessment spurred Congress to commission a report from the National Research Council (NRC) of the National Academy of Sciences (NAS) on how the process was being used. The result was the landmark 1983 study entitled *Risk Assessment in the Federal Government: Managing the Process*.[1] The document is often referred to as "The Red Book" because of its distinctive red cover. The Red Book acknowledged that regulatory agencies have differing statutory obligations that require some flexibility in both the risk assessment and risk management processes. The Red Book also clarified what risk assessment and risk management are by giving them the definitions that are still commonly used today (see Exhibit 3-1):

Purpose of the 1983 NRC Report

- Assess the merits of separating the analytic functions of developing risk assessments from the regulatory functions of making policy decisions.
- Consider the feasibility of designating a single organization to do risk assessments for all regulatory agencies.
- Consider the feasibility of developing uniform risk assessment guidelines for use by all regulatory agencies.

- "We use **risk assessment** to mean the characterization of the potential adverse health effects of human exposures to environmental hazards" (p. 18).
- "The Committee uses the term **risk management** to describe the process of evaluating alternative regulatory actions and selecting among them" (p. 18).

Exhibit 3-1. Risk Assessment and Risk Management Paradigm



Source: Adapted from the 1983 "Red Book"

The Red Book did not recommend "bright line analysis" because it gives too much weight to risk numbers that are, by their very nature, uncertain. The NRC also made two important recommendations regarding the risk assessment and risk management processes used by federal agencies:

- First, the scientific finding and policy judgments embodied in risk assessments should be explicitly distinguished from the political, economic, and technical considerations that influence the design and choice of regulatory strategies.
- Second, uniform guidelines should be developed for use by federal regulatory agencies in the risk assessment process.

Bright Line Analysis

"Bright line analysis" is the process of comparing a risk assessment result (the estimated numerical value of risk) to a preestablished acceptable level of risk (the "bright line") and making risk management decisions solely on whether the estimated risk is above or below the acceptable level. The NRC emphasized that risk assessment results are only one component of the risk management decision process and that assessment results should not be the only information risk managers consider.

The Red Book had a significant impact on risk assessment and management processes throughout the federal government, and it continues to be an influential reference at EPA. For example, in response to its recommendations, EPA established the Risk Assessment Council (RAC) and began publishing Agency-wide risk assessment guidelines (see Section 3.1.4 below). (Note that the recommendation to develop uniform risk assessment guidelines for use by all regulatory agencies did not happen – each agency is still free to develop their own approaches and guidelines.)

3.2.2 The 1994 National Research Council Report

Recognizing the growing importance of quantitative risk assessment in the regulatory process, Congress in section 112(o) of the Clean Air Act (CAA) amendments required EPA to enter into a contract with the NRC to evaluate the risk assessment methods EPA was using at the time. The NRC's 1994 report, *Science and Judgment in Risk Assessment*,[2] was prepared by the NRC's Committee on Risk Assessment of Hazardous Air Pollutants in the Board on Environmental Studies and Toxicology. In a sense, the "Blue Book" was a follow-up to the 1983 Red Book, but with a specific emphasis on EPA's scientific methods.

Purpose of the 1994 NRC Report

Congress asked the NRC to answer the following questions:

- Given that quantitative risk assessment is essential for EPA's implementation of the CAA, is EPA conducting risk assessments in the best possible manner?
- Has EPA developed mechanisms for keeping its risk assessment procedures current in the face of new developments in science?
- Are adequate risk-related data being collected to permit EPA to carry out its mandates?
- What, if anything, should be done to improve EPA's development and use of risk assessments?

The NRC committee observed that several themes were common to all elements of the risk assessment process and noted that these themes were usually the focal points for criticisms of individual risk assessments:

- The use of default assumptions;
- Available data;
- Uncertainty and variability;
- Assessment of multiple chemical exposures, multiple routes of exposure, the potential for multiple adverse effects; and
- Steps taken to validate the methodologies used throughout the risk assessment process.

In the Blue Book, the NRC updated the risk assessment/risk management paradigm and presented several recommendations for increasing the effectiveness and accuracy of EPA's risk assessment and risk management process, particularly as it pertained to air toxics:

- EPA should generally retain its conservative, default-based approach to risk assessment for screening analysis in standard-setting.
- EPA should use iterative approaches that incorporate improvements in both the models and data used in each successive iteration of analysis. For example, EPA should start with relatively inexpensive screening techniques and move to a more resource-intensive level of data-gathering, model construction, and model application as the particular situation warrants. This method avoids costly case-by-case evaluations of individual chemicals at every facility in every source category.

- EPA should explicitly identify each use of a default option in a risk assessment, should clearly state the scientific and policy basis for each default option, and should consider attempting to give greater formality to its criteria for departure from default options.
- EPA should establish regulatory priorities based on initial assessments of each chemical's possible impact on human health and welfare.
- EPA should present not only point estimates of risk, but also the sources and magnitudes of uncertainty associated with these estimates.

EPA has progressively worked to adopt the report's recommendations as it transitions the Agency into the risk-based phase of the CAA legislative strategy for HAPs.

3.2.3 The CRARM

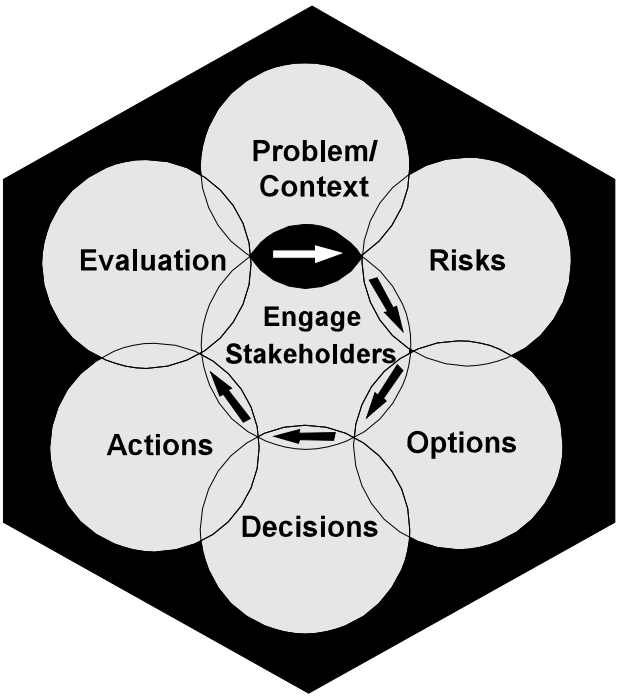
Section 303 of the 1990 CAA Amendments mandated the formation of a Presidential Commission on Risk Assessment and Risk Management (CRARM) in response to unresolved questions about EPA's approach to assessing public health risks remaining after implementation of the maximum achievable control technology (MACT) program (i.e., technology based control). CRARM released its report, *Risk Assessment and Risk Management in Regulatory Decision-Making*, or the White Book, in two volumes in 1997. Volume I focuses on the framework for environmental health risk management. Volume II addresses a variety of technical issues related to risk assessment and risk management, including a common metric for assessment of cancer and other effects, management of residual risks from air toxics, comparative risk, decision criteria, uncertainty analysis, and recommendations to specific agencies.[3]

Purpose of the 1997 White Book

Investigate "the policy implications and appropriate uses of risk assessment and risk management in regulatory programs under various Federal laws to prevent cancer and other chronic health effects which may result from exposure to hazardous substances."

The CRARM developed a risk management framework that fosters an integrated approach to addressing complex, real-world issues that affect multiple environmental media and involve exposures to mixtures of chemicals (Exhibit 3-2). Note that risk assessment (here "risk") is one of several steps in risk management. The framework aims to encourage integrated approaches to environmental risk management.

Exhibit 3-2. The CRARM Framework for Risk Management



The central element of the framework is encouraging stakeholder participation throughout the six stages of risk management. In addition, the framework intends to be iterative – if appropriate, risk assessors can redefine and reassess the risk problem as they develop new data. Another key principle of the framework is that risk management should explicitly consider the comprehensive, real-world context of a risk problem and not limit the context to one that considers only one type of risk associated with a single chemical in a single environmental medium. The CRARM made several additional recommendations:

- **Conduct Comparative Risk Assessment**. Federal agencies should try a comparative risk analysis approach on an experimental or demonstration basis to seek consensus on priorities for managing environmental risks. The results of such efforts should influence agency resource allocation.

- **Harmonize Cancer and Non-Cancer Methodologies**. Assessment techniques for carcinogens and non-carcinogens should be harmonized. This would aid in risk communication, risk management decisions, and comparative risk assessment.

- **Devise Realistic Exposure Scenarios**. Risk management decisions should be based on realistic exposure scenarios, rather than on the hypothetical **maximum exposed individual** (MEI). Distributions of the varied exposures within a population should be evaluated with

explicit attention to specific segments of the population (e.g., individuals with unusually high exposures, infants, children, pregnant women, low-income groups, and minority communities with exposures influenced by social or cultural practices).

- **Place Cost-Benefit Analysis in its Appropriate Context**. Economic analysis is a relevant consideration in risk management decisions, but should not be the overriding factor in a decision. Explicit descriptions of assumptions, data sources, sources of uncertainty, and costs across society should be presented in parallel with descriptions associated with risk assessments.

- **Ensure Interagency Consistency**. Agencies should coordinate their risk assessment methods and assumptions unless there is a specific statutory requirement that allows for different choices. Scientific disagreements should be explained.

- **Conduct Tiered Residual Risk Assessments**. EPA should implement a tiered approach to managing residual risks after implementation of the CAA's technology-based (MACT) standards.

Similar to the recommendations outlined in the Blue Book, EPA has continued to modify its risk assessment guidelines and approaches in response to these recommendations. Other documents, such as the National Research Council's 1996 document entitled *Understanding Risk: Information Decisions in a Democratic Society*,[(4)] also play a role in informing the continued development of the risk assessment and risk management process.[(a)]

3.2.4 Development of Human Health Risk Assessment at EPA

EPA has conducted human health risk assessments since its inception in 1970. EPA built on this early experience while confronting potential hazards associated with pesticide use. For example, after considering available human and non-human toxicity data, EPA restricted domestic use of DDT and other pesticides, in part due to their cancer risks.

Fundamental References for Air Toxics Risk Assessment

(1) Air Toxics Risk Assessment Reference Library, three volumes

(2) The NAS Red and Blue Books

(3) CRARM White Book

(4) EPA Guidelines for Risk Assessment Series

(Full citations are at the end of this chapter.)

EPA acknowledged that such risk-based regulations needed an appropriate scientific basis and began collecting cancer toxicity information on pesticides through administrative hearings and testimony. Summary documents from these hearings became known as the "Cancer Principles." Criticism of these documents, which many inadvertently perceived to be formal Agency cancer risk assessment policy, led the Agency to develop interim guidelines in 1976. Three years later, the

[a] *Understanding Risk* "...illustrates that making risks understandable to the public involves more than translating scientific knowledge. The volume also draws conclusions about what society should expect from risk characterization and offers guidelines and principles for informing the wide variety of risk decisions that face our increasingly technological society." (See http://books.nap.edu/catalog/5138.html.)

Interagency Regulatory Liaison Group (a conglomeration of several federal agencies, including EPA) published additional cancer risk assessment guidelines. Concurrently, EPA used cancer risk assessment techniques in its toxic chemicals regulation under the 1976 Toxic Substances Control Act. By the end of EPA's first decade in existence, the Agency used risk assessment techniques to develop water quality criteria protective of human health.

Throughout the 1980s, EPA increasingly utilized risk assessment to evaluate the potential for chemicals to cause non-cancer health effects in addition to cancer risks. During the 1980s, the Agency used cancer risk assessment techniques in the development of national emission standards for air toxics such as vinyl chloride and benzene.

As EPA increased its use of risk assessment throughout the 1980s, the Agency's inconsistent approach to risk assessment became apparent, largely due to a lack of standard guidance on the topic. To correct this problem, the Agency undertook administrative reforms and published several key guidelines and other policy documents.

First, the Agency published *Risk Assessment and Management: Framework for Decision Making.*[5] EPA intended this reference manual to conform EPA practices with NRC Red Book recommendations and to help the Agency make better and more rapid decisions about environmental toxic chemical problems.

Next, in 1986, EPA established the *Risk Assessment Council* (RAC) to oversee virtually all aspects of the Agency's risk assessment process. EPA appointed Senior Agency officials with experience and responsibilities in the area of science policy and risk assessment to the RAC. This group established EPA's fundamental policies for conducting risk assessments and evaluating risk information. These officials also oversaw the activities of the Risk Assessment Forum.

EPA Risk Assessment Forum

The Risk Assessment Forum is a standing committee of senior EPA scientists established to promote Agency-wide consensus on difficult and controversial risk assessment issues and to ensure that this consensus is incorporated into appropriate Agency risk assessment guidance. To fulfill this purpose, the Forum assembles Agency risk assessment experts in a formal process to study and report on issues from an Agency-wide scientific perspective. Major Forum guidance documents are developed in accordance with the Agency's regulatory and policy development process and become Agency policy upon approval by the Administrator or the Deputy Administrator. Risk Assessment Forum products include: risk assessment guidelines, technical panel reports on special risk assessment issues, and peer consultation and peer review workshops addressing controversial risk assessment topics (http://cfpub.epa.gov/ncea/raf/index.cfm).

Subsequently, EPA began publishing an influential series of Agency-wide guidelines in the *Federal Register* identifying the recommended methods for assessing human health risks from environmental pollution. EPA did not intend for these guidelines, which cover both cancer risks and non-cancer hazards, to be static, and the Agency has revised the guidelines as new information and methods become available (for example, EPA began a process in 1996 to revise and update its guidelines for carcinogenicity).

EPA established the Science Policy Council (SPC) in 1993 with a broader mission and as a replacement for the RAC; specifically, the SPC aims to integrate policies that guide Agency decision-makers in their use of scientific and technical information. To accomplish this goal, the SPC works to implement and ensure the success of selected initiatives that external advisory bodies (such as the National Research Council and the Science Advisory Board, as well as others such as the Congress, industry and environmental groups, and Agency staff), recommend. In this way, the SPC provides guidance for selected EPA regulatory and enforcement policies and decisions. The 1995 Guidance for Risk Characterization was an important part of the SPC's risk characterization program. Standing groups such as the Risk Assessment Forum, a Steering Committee, and interim working groups continue to support the SPC. For more information on the SPC, see http://www.epa.gov/osp/spc/2about.htm.

EPA Human Health Risk Assessment Guidelines[a]

Carcinogenicity
- 1999 Draft Revised Guidelines for Carcinogen Risk Assessment[b]
- 1986 Guidelines for Carcinogen Risk Assessment

Chemical mixtures
- 2000 Supplementary Guidance for Conducting Health Risk Assessment of Chemical Mixtures
- 1986 Guidelines for the Health Risk Assessment of Chemical Mixtures

Developmental toxicity
- 1991 Guidelines for Developmental Toxicity Risk Assessment

Exposure assessment
- 1992 Guidelines for Exposure Assessment

Mutagenicity
- 1986 Guidelines for Mutagenicity Risk Assessment

Neurotoxicity
- 1998 Guidelines for Neurotoxicity Risk Assessment

Probabilistic analysis
- 1997 Guiding Principles for Monte Carlo Analysis

Reproductive toxicity
- 1996 Guidelines for Reproductive Toxicity Risk Assessment

Risk characterization
- 2000 Handbook for Risk Characterization
- 1997 Guidance on Cumulative Risk Assessment. Part 1, Planning and Scoping
- 1995 Guidance for Risk Characterization

(a) A current list is available at http://cfpub.epa.gov/ncea/raf/recordisplay.cfm?deid=55907.
(b) These guidelines are interim final drafts. Check above website for a final version.

Another important group within EPA with a risk assessment focus is the National Center for Environmental Assessment (NCEA). NCEA is a major component of the EPA's Office of Research and Development and acts as EPA's national resource center for human health and ecological risk assessment. NCEA conducts risk assessments, carries out research to improve the state-of-the-science of risk assessment, and provides guidance and support to risk assessors. Many of the critical Agency documents on risk assessment science and policy, as well as risk related databases such as the Integrated Risk Information System (IRIS), can be accessed through the NCEA website (www.epa.gov/ncea).

EPA's use and development of human health risk assessment continued to grow through the 1980s and 1990s with establishment of the IRIS toxicity database, the Agency's repository of chemical-specific toxicity data. IRIS is a critical resource for risk assessors because the database contains toxicity information that reflects a consensus among EPA program offices about a chemical's toxic properties.

EPA's Office of Solid Waste and Emergency Response's Superfund Program also has developed a series of very detailed guidance documents to help risk assessors understand the actual nuts-and-bolts of performing human and ecological risk assessments under the Superfund program. These "how to" documents are called the ***Risk Assessment Guidance for Superfund*** series, or the RAGS series for short. RAGS provides in-depth discussions and guidance for risk assessors to use in their day-to-day work and is an important reference for those working in the field of risk assessment.[b] A full set of RAGS documents is available online.[6]

Risk Assessor. The individual or team of individuals who organizes and analyzes air toxics data, develops exposure and risk calculations, and prepares the human health risk assessment reports. Risk assessors for air toxics can be industry, EPA, an S/L/T air agency, or contractor personnel. The larger **risk assessment team** will often be made up of people with a variety of expertise, including health scientists, monitoring or modeling personnel, and laboratory analysts.

Risk Manager. The individual or group of individuals who serve as the primary decision maker(s) for an area subject to the risk analysis process. The risk managers may base their decisions about the need for risk reduction on a variety of data, including the results of the risk assessment, economic considerations, technical feasibility of risk reduction options, community acceptance, and a number of other factors.

3.3 Air Toxics Human Health Risk Assessment: Overview of the Process

The reports and guidance documents discussed above tend to distill the risk assessment process down to the following five questions:

- Who is exposed to environmental pollutants?
- What pollutants are they exposed to?
- How are they exposed?
- How toxic are the chemicals they are exposed to?
- What is the likelihood that harm will occur because of the exposures?

The role of the risk assessor is to answer these questions. The main product of the risk assessment is a set of qualitative and quantitative statements about the likelihood that people will experience adverse health outcomes because of the exposures. The statements also should discuss how certain the assessor is about these statements. **Risk managers** then use the risk assessment results and other relevant information (including the cost or technical feasibility of resolving a problem) to decide what (if anything) should be done to reduce risk.

[b] Although the information provided in RAGS is primarily geared towards Superfund sites, some of these procedures are generally relevant and compatible to risk assessments developed by other Program Offices, including the Office of Air and Radiation. As such, the information provided in RAGS was taken into consideration in the development of this reference library.

The following sections briefly describe the overall risk assessment process for releases of air toxics to the ambient air. Subsequent chapters of this reference manual revisit each of these subjects in detail and provide contacts and references for more information.

3.3.1 Air Toxics Risk Assessment: What Is the Question?

The overall purpose of a human health air toxics risk assessment is to attempt to understand public health risks potentially associated with exposures to particular pollutants emitted into the air from sources of interest. Exhibit 3-3 presents a simple illustration of the overall real-world process that is investigated through the use of risk assessment.

As Exhibit 3-3 illustrates, air toxics risk assessments usually focuses, at a minimum, on the inhalation of contaminated air. However, for a small subset of air toxics (discussed in Chapter 4), the risk assessment also may need to address ingestion of or dermal contact with soils, water, or food that have become contaminated with chemicals that have deposited out of the air. (Dermal exposures are included here for completeness, but usually they are less of a risk factor for air toxics than ingestion or inhalation exposures.)

The following simple mathematical formula describes the basis for human health risk assessment. Specifically, the likelihood that injury or disease may occur from exposure to air toxics can be described as a function of two separate, but related, things – an estimate of exposure to a chemical and an estimate of the toxic properties of the chemical:

$$\text{Potential for Injury or Disease (i.e., the “Risk”)} = f\ (\text{metric of exposure, metric of toxicity})^{(c)} \qquad \text{(Equation 3-1)}$$

Two key principles emerge from this formula and Exhibit 3-3:

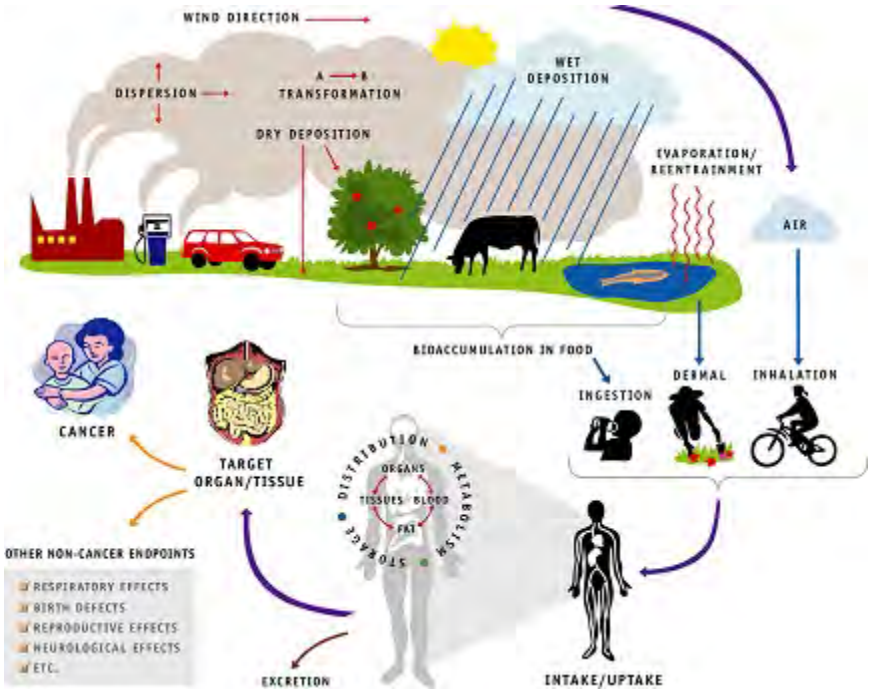
- **There is no risk if there is no exposure**. If a person has no chance of coming in contact with an air toxic, the risk posed to that person is zero.
- **The level of risk associated with an exposure depends on the toxic properties of the chemical**. These properties determine whether the exposure is of great or little concern. Some chemicals can cause severe health effects (even death) when a person receives exposure even to extremely small quantities at a single point in time. Conversely, other chemicals cause essentially no effect even after repeated exposure to high levels over long periods of time.

The general Equation 3-1 is important to understand and keep in mind since the exact equations used to develop risk estimates are derived from it. In other words, the risk equations that will be detailed in later chapters all include both a estimate of exposure and an estimate of toxicity.

---

[c] The symbol “*f*” means “is a function of”

Exhibit 3-3. Generic Conceptual Model of How Air Toxics Releases May Result in Injury or Disease



Starting at the upper left hand side of this diagram, air toxics are released from one or more sources (e.g., factories, cars/trucks, small businesses, forest fires) to the air and begin to disperse by the wind away from the point of release. Once released, the chemical may remain airborne; convert into a different substance; and/or deposit out of the air onto soils, water, or plants. People may be exposed to air toxics by breathing contaminated air (inhalation) or through ingestion of chemicals that can accumulate in soils, sediments, and foods (the latter process is called **bioaccumulation)**. People also can be exposed to deposited chemicals via skin (dermal) contact, however, this tends to be a less important risk factor than ingestion or inhalation. Inhalation, ingestion, and dermal absorption are called the **routes of exposure**.

This description of what happens to an air toxic once it is released into the air is called **fate and transport** analysis. "Transport" evaluates how an air toxic physically moves (i.e., is transported) through the environment. "Fate" describes what ultimately happens to the chemical after it is released to the air (i.e., what is the "fate" of the chemical in the environment). The results of a fate and transport analysis is an estimate of the concentration of the air toxic in the air, soil, water, and/or food at the point where it is contacted by a person. The **exposure assessment** is the process of evaluating how human contact with the contaminated media occurs.

In the case of an air pathway analysis, the metric representing the inhalation exposure is called the **exposure concentration** (EC). For example, if benzene is released from a factory and blows into a nearby neighborhood where people breath it, the EC is the concentration of benzene in the air that they breath.

Once an exposure occurs, the air toxics can enter the body and exert an effect at the point of entry (the "portal of entry") or move via the bloodstream to other target organs or tissues. The action of a pollutant on a target organ can result in a variety of harmful effects, including cancer, respiratory effects, birth defects, and reproductive and neurological disorders. An overall risk assessment process evaluates what people are exposed to, how the exposure occurs, and, when combined with information about the toxic properties of the chemicals in question, estimates the likelihood that the exposure will result in injury or disease.

Air toxics risk assessments commonly look at two types of exposures and their associated toxic outcomes:

- Repeated or extended exposure to relatively low concentrations of air toxics over long periods of time (**chronic exposures**) that may result in **chronic health effects** (e.g., diseases like cancer or recurring respiratory ailments); and
- Infrequent exposure to relatively high concentrations of air toxics over short periods of time (**acute exposures**) that may result in the expression of either near term **acute health effects** (which can range from mild effects, such as reversible eye irritation, to extreme effects, such as loss of consciousness or sudden death), or long term effects (chronic effects).

3.3.2 Air Toxics Risk Assessment: The Process

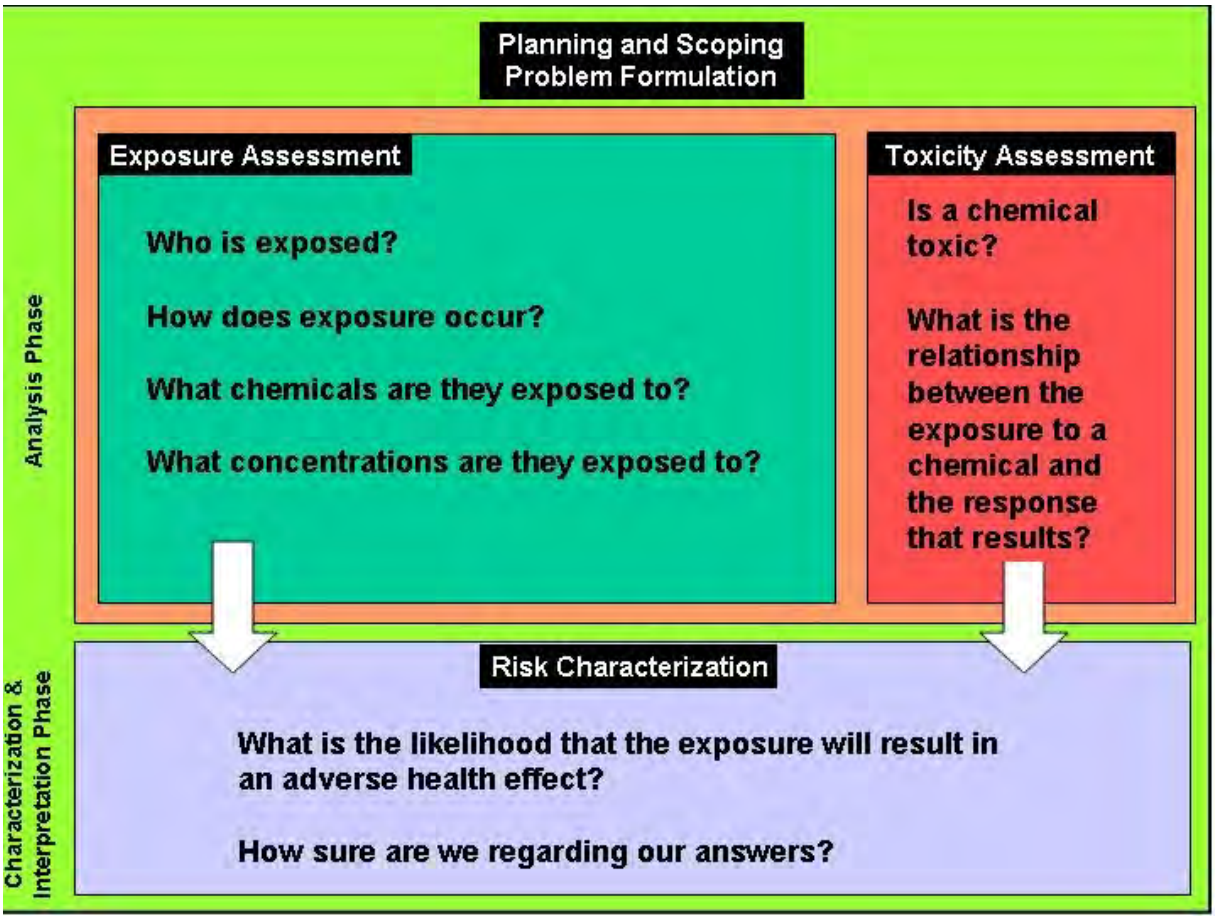
The illustration and narrative overview in the previous section (Exhibit 3-3) describes what may happen when toxic chemicals are released to the air and how those releases can result in adverse health outcomes in people. This picture and narrative description comprise a **conceptual model** of how releases of air toxics may pose risks to people. It is a conceptual model because it provides a picture (or "model") of our "concept" of what may happen in the real world when toxic chemicals are released to the air. The conceptual model provides a starting point for estimating risks posed by those releases. However, in addition to a conceptual model (in this case, a simple picture), there is a need for a defined process to quantify relationships among the conceptual model components in order to generate numeric risk estimates. Exhibit 3-4 outlines the major steps in the process that EPA uses to perform a risk assessment:

- Planning, scoping, and problem formulation;
- Analysis, which includes exposure assessment and toxicity assessment; and
- Risk characterization.

With the addition of an explicit planning and scoping step (which should always be done for any systematic investigation), Exhibit 3-4 encompasses the same features as espoused by the National Academies in the Red and Blue books described previously. The National Academies' process has been redrawn in Exhibit 3-4 to better clarify how the risk assessment is actually done in the air toxics arena.

It is useful to think of this figure as a "roadmap" to how air toxics risk assessments are performed. The roadmap breaks air toxics risk assessment down into four manageable elements, each of which are described briefly below and in detail in subsequent chapters. Note, however, that all of these steps are inter-related and usually require refinement throughout the risk assessment process. A helpful starting place is to think of these as "separate steps."

Exhibit 3-4. The General Air Toxics Risk Assessment Process



3.3.2.1 Planning, Scoping, and Problem Formulation

Any human health risk assessment should begin with **planning and scoping**. Properly planning and scoping the risk assessment at the beginning of the project is critical to the success of the overall effort. Good planning and scoping clearly articulates the assessment questions; states the quantity and quality of data needed to answer those questions; provides in-depth discussion of how assessors will do the analysis; outlines timing and resource considerations, as well as product and documentation requirements; and identifies who will participate in the overall process from start to finish and what their roles will be. Poor planning and scoping will almost certainly lead to an assessment that does not answer the correct questions, does not provide a supportable basis for risk management decision-making, and wastes significant amounts of time, resources, and good will. The planning and scoping process needs to recognize, to the extent possible, important data gaps and uncertainties and the measures needed to address these problems. Where the extent of data gaps and their potential impacts on the risk assessment are not fully understood, the planning process may be iterative, with decision points specified during the analytical phase (see below) that are contingent on the results of data gathering efforts or sensitivity/uncertainty analyses.

During **problem formulation**, the planning and scoping team generally makes initial decisions about the scope of the risk assessment (e.g., size of the study area, what emission sources and chemicals are to be considered); the appropriate level of detail and documentation; trade-offs between depth and breadth in the analysis; quality assurance and quality control requirements; analytical approaches to be used (modeling vs. monitoring); and the staff and monetary resources to commit. Problem formulation results in two important products: the conceptual model and the analysis plan.

- The **study-specific conceptual model** is similar to the generic conceptual model (Exhibit 3-3); however, for an actual assessment the conceptual model explicitly identifies the physical boundaries of the study area; the potential emission sources and air toxics they are emitting that the risk assessment will consider; the location and composition of potentially exposed populations; the fate/transport mechanisms by which those populations may be exposed; the routes of exposures that may be occurring; and the expected health outcomes to be evaluated. The study-specific conceptual model is developed as both a picture and a written description of how air toxics emissions may be affecting the study area. As the assessment moves forward, the assessment team members will use the model as a guide, but they also routinely refine the model as they learn more about the study area. For example, the initial study-specific conceptual model may include a deposition element. If subsequent modeling or monitoring suggests this fate and transport mechanism is unimportant, the assessors will revise the conceptual model.
- The **analysis plan** will guide the remainder of the assessment. It lays out in detail how the elements of the conceptual model are going to be studied. In developing the analysis plan, it is important to include provisions for tiered or iterative analyses, as discussed in Sections 3.2.3 and 3.3.5.

3.3.2.2 Analysis Phase

The analysis phase is the process in which analysts apply risk assessment approaches to evaluate the problem at hand. It consists of two main components: exposure assessment and toxicity assessment.

An **exposure assessment** is conducted to characterize the potentially exposed population, the chemicals of potential concern, identify exposure pathways and routes of exposure, and estimate the exposure. This includes estimating or measuring concentrations of air toxics in the environment and evaluating how nearby populations interact with the contaminated media.

In the exposure assessment, the risk assessment team will refine the initial conceptual model by providing detailed information about the study area (e.g., physical description, meteorology, source locations and detailed characteristics, population demographics and locations, the

What is "Study-Specific?"

Air toxics risk assessments can be designed to evaluate a wide range of air toxics release scenarios. For example, a risk assessment might look at the impact of one emission point at a factory on a nearby population or it might look at the combined impact of hundreds of sources on a large urban area.

This reference manual uses the term "study-specific" to mean the specific geographic area and populations under study, along with the emission sources included in the scope of the study.

exposure pathways under study). The exposure assessment also is the analytic step in which the magnitude, frequency, and duration of human exposures are quantified. For example, one of the main outcomes of an air toxics exposure assessment is an estimate of the concentration of air toxics in the air at the point where human contact occurs (the EC). Assessors usually estimate this value with either a computer program (a **model**) or by physically taking samples of air and measuring air toxics concentrations in a laboratory (a **monitor**). When there are concerns about exposure pathways other than inhalation, assessors may use different models or monitoring strategies to estimate or measure concentrations of air toxics in soil, water, or foods.

The **toxicity assessment** component of the risk assessment process considers: (1) the types of adverse health effects associated with exposure to the chemicals in question, and (2) the relationship between the amount of exposure and resulting response. Toxicity assessment for air toxics generally consists of two steps:

- **Hazard identification** is the process of determining whether exposure to a chemical can cause an adverse health effect (e.g., cancer, birth defect, etc.), as well as the nature and strength of the evidence of causation and circumstances in which these effects occur (e.g., inhalation/ingestion, repeated exposure over a long period/single exposure over a short period, etc,).
- **Dose-response assessment** is the process of quantitatively characterizing the relationship between the dose of the contaminant and the incidence of adverse health effects in the exposed population. As information on dose at the site in the body where the response occurs is rarely available, various factors and models are used to predict the dose metric from estimates of exposure (the inhalation exposure concentration or oral intake). From this quantitative dose-response relationship, toxicity values are derived for use in risk characterization.[(d)] Most toxicity assessments are based on studies in which toxicologists expose animals to chemicals in a laboratory and extrapolate the results to humans. For some chemicals, information from actual human exposures is available (usually from workplace exposure studies).

Although air toxics risk assessors need to understand the underlying scientific basis and uncertainties associated with toxicity values, they will usually rely on toxicity values already developed and available in the literature. A list of default screening level toxicity values that EPA recommends for the 188 HAPs is in Appendix C. The most up-to-date list is at http://www.epa.gov/ttn/atw/toxsource/summary.html.

3.3.2.3 Risk Characterization

The **risk characterization** summarizes and combines outputs of the exposure and toxicity assessments to characterize risk, both in quantitative (numerical) expressions and qualitative (descriptive) statements. Chemical-specific exposure-response information is mathematically combined with modeled or monitored contaminant levels and other information regarding how exposure occurs to give numbers that represent the likelihood that the exposure may cause an

[d]Toxicity values are numerical expressions of the relationship between a given level of exposure to an air toxic and adverse health impacts. The two most common toxicity values for inhalation exposures are the upper-bound inhalation unit risk estimates (IURs) for cancer effects and reference concentrations (RfCs) for non-cancer effects (which include uncertainty factors). Chapter 12 provides a more detailed discussion of toxicity values.

adverse health outcome. Per the Agency's *Policy for Risk Characterization*,[7] this likelihood is evaluated both with regard to a "central tendency" of exposure estimates and "high end" estimates. The risk characterization also includes a thorough **uncertainty analysis** for each step of the entire risk assessment process in order to provide the risk manager with an understanding of which elements of the assessment are most uncertain, the magnitude and direction of the effect (higher or lower) that the various uncertainties have on the risk estimates and in some cases, a quantitative analysis of uncertainty. Often the uncertainty analysis is a narrative that reflects the assessor's best professional judgment. Other analyses, however, may require a more quantitative approach to evaluating uncertainty.

The product of the risk assessment is a written report that provides all of the analyses performed to assess exposure, identify toxicity values, characterize risk, and assess and present uncertainty. It is critical that the risk assessment only provide the factual basis of why the assessment was done, how it was done, what the answers are, and the uncertainties associated with those answers. That is not to say that the risk assessment should not provide an analysis of differing scientific opinions on any number of the elements of the risk assessment. It does, however, preclude the assessment from discussing items more appropriately considered under risk management (e.g., cost or technical feasibility of mitigation alternatives). The presentation also must be clear and provide enough details so future readers will find the overall assessment process, including critical assumptions, to be fully transparent.

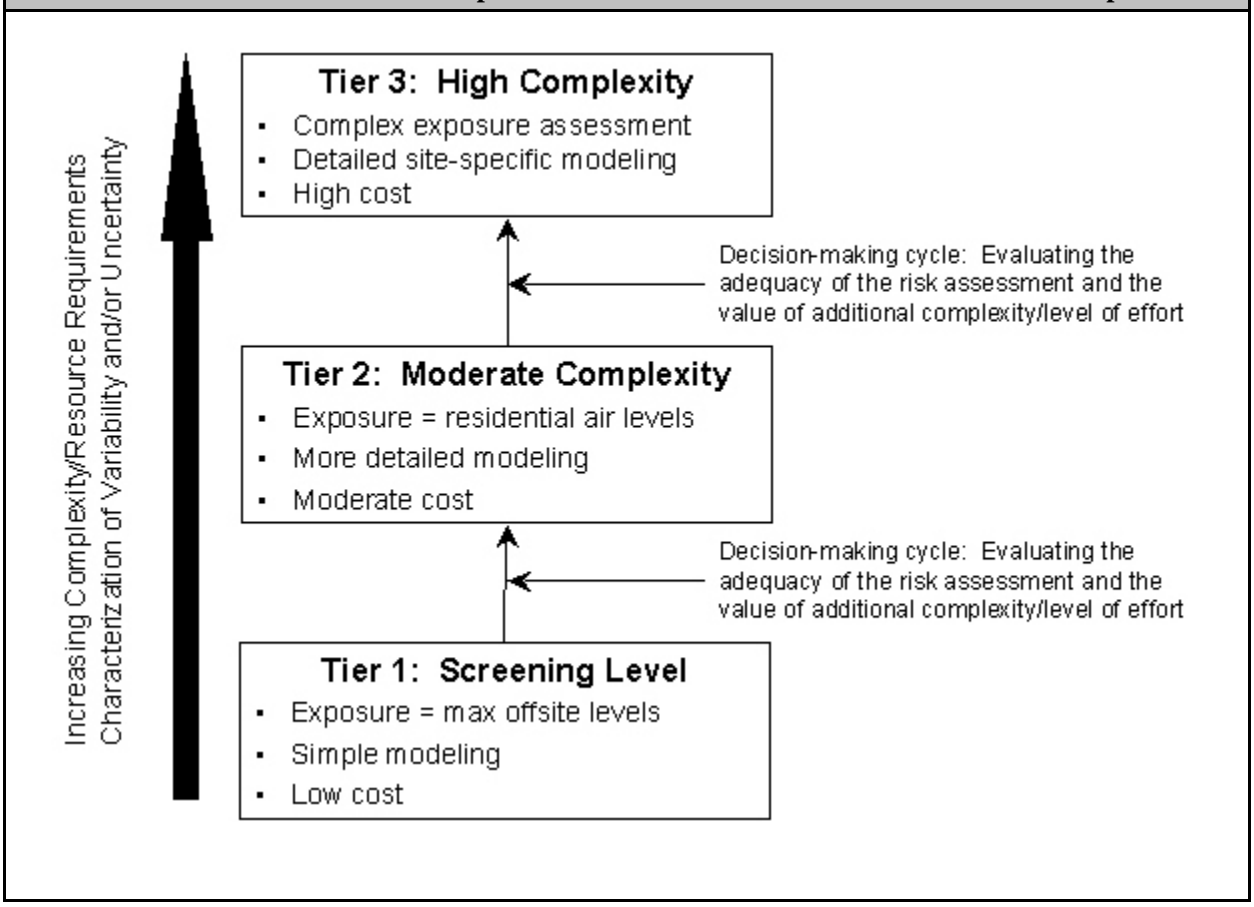
3.3.3 Tiered Assessment Approaches

Various EPA guidance documents and the Air Program's *Residual Risk Report to Congress* have recommended tiered approaches to risk assessments.[8] A tiered approach is a process for a systematic, informed progression from a relatively simple to a more complex risk assessment approach. Essentially, the approach begins with an analysis that includes few study-specific data and many conservative assumptions. This process generally results in a very conservative answer (and is likely to be fairly uncertain), but may demonstrate, with relatively little effort, that the sources being assessed pose insignificant risk. If such an approach indicates that the risk appears to be relatively high, assessors pursue a higher tier of analysis to determine if the risk is a realistic concern or an artifact of the lower tier's conservative assumptions. The higher level of analysis reflects increasing complexity and, in many cases, will require more time and resources. Higher tiers also reflect increasing characterization of variability and/or uncertainty in the risk estimate, which may be important for making risk management decisions.

Exhibit 3-5 illustrates a generalized representation of the tiered risk assessment concept. Central to the concept of the tiered approach is an iterative process of evaluation, deliberation, data collection, work planning, and communication aimed at deciding:

- Whether or not the risk assessment, in its current state, is sufficient to support the risk management decision(s); and
- If the assessment is determined to be insufficient, whether or not progression to a higher tier of complexity (or refinement of the current tier) would provide a sufficient benefit to warrant the additional effort.

Exhibit 3-5. Generalized Representation of the Tiered Risk Assessment Concept



The deliberation cycle also provides an opportunity to evaluate the direction and goals of the assessment as new information becomes available. It may include evaluations of both scientific and policy information.

This representation, which provides an example of a tiered assessment process consistent with that described in the *Residual Risk Report to Congress*,[8] depicts three tiers of analysis. Each successive tier represents more complete characterization of variability and/or uncertainty as well as a corresponding increase in complexity and resource requirements.

- **Tier 1** is represented as a relatively simple, screening-level analysis using conservative exposure assumptions (e.g., receptors are located in the area with the highest estimated concentrations) and relatively simple modeling (e.g., a model that requires few inputs, most of which can be "generic," yet conservative).
- **Tier 2** is represented as an intermediate-level analysis using more realistic exposure assumptions (e.g., use of actual receptor locations) and more detailed modeling (e.g., a model that requires additional site-specific inputs).
- **Tier 3** is represented as an advanced analysis using probabilistic techniques such as Monte Carlo analysis (see Part VII of this reference manual for a discussion of these techniques) and more detailed and/or intensive modeling.

This representation does not imply that there is a clear distinction between Tiers 1, 2, and 3. For example, a series of refinements in a Tier 1 analysis might be indistinguishable from a Tier 2 analysis, or a Tier 2 analysis could incorporate probabilistic techniques.

This representation also notes the decision-making cycle that occurs between each tier. In this cycle, the existing risk assessment results are evaluated to determine whether they are sufficient for the risk management decision, and if not, what refinements to the risk assessment are needed (including moving up to the next tier).

While the tiered risk assessment concept usually contains three tiers of complexity (as in Exhibit 3-5), these three tiers are best thought of as points along a spectrum of increasing complexity and detail in the risk assessment. The important focus is the specific ways in which a given risk assessment is refined in successive iterations, rather than whether or not it would be considered Tier 1, 2, or 3.

3.4 Uncertainty and Variability in Air Toxics Risk Assessment

Risk assessment is based on a series of questions that the assessor asks about available scientific information that is relevant to human health and/or ecological risk. Each question calls for analysis and interpretation of the studies, selection of the concepts and data that are most scientifically reliable and most relevant to the problem at hand, and conclusions regarding the question presented. For example, in the exposure assessment, through the use of modeling and/or monitoring, the risk assessor asks what is known about the principal environmental fate and transport of contaminants and the patterns and magnitudes of human or ecosystem exposures. The toxicity assessment asks what is known about the ability of an air toxic to cause cancer or other adverse health effects in humans, laboratory animals, or wildlife species and what is known about the biological mechanisms and dose-response relationships underlying any effects observed in the laboratory or in epidemiology studies. The risk characterization integrates information from the preceding components of the risk assessment and synthesizes an overall conclusion about estimated risk that is complete, informative, and useful for risk managers.[7]

Air toxics risk assessments make use of many different kinds of scientific concepts and data (e.g., exposure, toxicity, epidemiology, ecology), all of which are used to characterize the estimated risk in a particular environmental context. Informed use of scientific information from many different sources is a central feature of the risk assessment process. Highly accurate information is often not available for many aspects of a risk assessment. However, since **scientific uncertainty** is inherent in the risk assessment process, and risk managers often must make decisions using assessments that are not as definitive in all important areas as would be desirable, it is important that the most current and complete information that is available be used to support decision making. Risk assessors and decision makers must understand that it may be necessary to revise risk estimates and to alter decisions in light of new information.

Risk assessments also incorporate a variety of professional judgements (e.g., which models to use, where to locate monitors, which toxicity studies to use as the basis of developing dose-response values). Risk managers therefore need to understand the strengths and the limitations of each assessment and to communicate this information to all participants and the public.

This section provides an overview of **uncertainty** and **variability**, two critically important characteristics of risk assessment that need to be understood and described at some level in every air toxics risk assessment. It describes several sources of uncertainty and variability in air toxics risk assessments, discusses approaches for describing and analyzing uncertainty and variability, and describes how uncertainty and variability are often addressed at different tiers of the risk assessment process.

A full discussion of this subject, including quantitative techniques for uncertainty analysis, is beyond the scope of this reference manual. Risk assessment is an evolving discipline, and improvements in scientific understanding and techniques will continue to provide new avenues and insights into uncertainty and variability analysis. Because this manual is intended as an introduction to risk assessment approaches and tools, our discussion focuses on relatively simplistic, deterministic risk assessment techniques (i.e., Tier 1 approaches to risk characterization that lead to single value estimates of risk). Readers are encouraged to consult the references at the end of this Chapter for additional information about uncertainty analysis in the risk assessment process.

3.4.1 Distinguishing Uncertainty and Variability

Variability refers to true heterogeneity or diversity. For example, among a local community that is exposed to an air toxic originating from the same source, and with all people breathing the same contaminant concentration in ambient air, the risks from inhalation of the contaminated air will still vary among the people in the population. This may be due to differences in exposure (i.e., different people have different exposure frequencies and exposure durations), as well as differences in response (e.g., differences in metabolic processes of chemical uptake into target organs). Differences among individuals in a population are referred to as inter-individual variability, while differences for one individual over time (e.g., change in sensitivity to air toxics with aging, illness) are referred to as intra-individual variability.

Uncertainty occurs because of a lack of knowledge. For example, we can be very certain that different people are exposed to contaminated air for different time periods, but we may be uncertain about how much variability there is in these exposure durations among the people in the population. Data may not be available concerning the amount of time specific people spend indoors at home, outdoors near home, or in other "microenvironments."

Uncertainty can often be reduced by collecting more and better data, while variability is an inherent property of the population being evaluated. Variability can be better characterized with more data, but it cannot be reduced or eliminated. Often, however, it is difficult to distinguish between uncertainty and variability in a risk assessment, particularly if available data are limited. For that reason, in many cases variability can be treated as a type of uncertainty in the risk assessment.

Uncertainty is an inherent characteristic of each step of the risk assessment process. Assessing uncertainty in risk assessment is an involved process because of the complex nature of the risk assessment process itself (i.e., risk assessment is a combination of a variety of data gathering and analytical processes, each with their own associated uncertainties). Specifically, risk assessment requires the integration of the following:

- Information on emissions of air toxics into the environment;
- Information on the fate and transport of air toxics, in a variety of different and variable environments, by processes that are often poorly understood or too complex to quantify completely;
- Information on the potential for adverse health effects in humans and/or ecosystems, often extrapolated from surrogate animal studies; and
- Information on the likelihood of adverse effects in a human population that is highly variable genetically, as well as factors such as age, activity level, lifestyle, and underlying disease.

Uncertainty, when applied to the process of risk assessment, is defined as "a lack of knowledge about specific factors, parameters, or models."[9] Such uncertainties affect the confidence of any risk estimates that were developed for individuals exposed to the substances in question.[10] It is important to keep in mind that many parameter values (e.g., emissions rates) may be *both* uncertain and variable. Also, the presence of uncertainty in risk assessment does not imply that the results of the risk assessment are wrong, but rather that the risks cannot be estimated beyond a certain degree of confidence.

The relatively simple, deterministic (i.e., single value estimate) approach outlined in this reference manual generally relies on a combination of point values – some which may be set at protective (i.e., high end) levels and some which may be set at typical (i.e., central tendency) levels. The result is a point estimate of exposure, and risk that falls at some percentile within the full distributions of exposure and risk. The degree of conservatism in high end risk estimates depends on the combination of input values selected.[11]

One of the key purposes of the uncertainty analysis is to provide an understanding of where the estimate of exposure, dose, or risk is likely to fall within the range of possible values. Often this is expressed as a subjective confidence interval (one based on incomplete data supplemented by professional judgment) within which there is a high probability that the estimate will fall. A related analysis, termed "sensitivity analysis" or "analysis of uncertainty importance," is often performed to identify the relative contribution of the uncertainty in a given parameter value (e.g., emission rate, ingestion rate) or model component to the total uncertainty in the exposure or risk estimate. This is often used either to identify which parameter values should be varied to provide high-end vs. central-tendency risk estimates, or to identify parameter values where additional data collection (or modeling effort) can increase the confidence in the resulting risk estimate.

3.4.2 Sources of Uncertainty in Air Toxics Risk Assessment

Although other taxonomies are sometimes used, sources of uncertainty in risk assessment are often divided into four categories (variability is sometimes included as a fifth category).[12]

- **Scenario uncertainty** occurs when information to fully define exposure and/or risk is missing or incomplete. This may include descriptive errors regarding the magnitude and extent of chemical exposure or toxicity, temporal and spatial aggregation errors, incomplete analysis (i.e., missing exposure pathways), and potential mis-specification of the exposed population or exposure scenario.

- **Model uncertainty** is associated with all models used in all phases of a risk assessment, including (1) animal models used as surrogates for evaluating human carcinogenicity, (2)

dose-response models, (3) computer models used to predict the fate and transport of chemicals in the environment, and (4) models used to estimate exposures for populations of concern. Model uncertainty also is sometimes referred to as specification uncertainty.

Computer models are simplifications of reality that use mathematical approximations to describe the most important processes governing the modeled relationships, while excluding what are believed to be less important processes, or processes that are too complex to be easily approximated. The risk assessor needs to consider the potential importance, in consultation with the modeler, of the level of detail and comprehensiveness of the models being used, because specific processes may have important impacts on uncertainty in some instances and not in others. A similar problem can occur when a model that is applicable under average conditions is used for a case in which conditions differ from the average. In tiered analyses, resource considerations and the level of precision required to support decision making may enter into considerations of model selection. Model uncertainty may be particularly important in multipathway analyses, because the modeling effort is much more complex (as compared to inhalation analyses). In addition to air quality modeling, multipathway analyses involve analysis of the transfer of air toxics from the air to other media (e.g., soil, sediment, water); the subsequent movement of the air toxics between these media (e.g., soil runoff to surface water); uptake and metabolism by biota; and subsequent ingestion by humans and wildlife. Uncertainties are associated with all of these analytical steps.

Model uncertainty is often difficult to deal with quantitatively. It is rarely possible to directly evaluate the merits of competing models, either due to resource constraints, or because direct comparisons are inherently complex (e.g., the models may take different input parameters, and produce outputs that are not directly comparable). Statistical methods (Bayesian analyses) can sometimes be used to combine the results of different models, but these approaches are often complex, and generalizability to specific cases is hard to predict. Thus, model selection tends to be based primarily on profession judgement and cost/complexity considerations.

- **Parameter Uncertainty** refers to the limitations in the modelers' ability to estimate precise values for certain parameters (variables) in the chosen models. It is a generic term that in common usage can refer either to variability or uncertainty, and generally indicates a situation where a given variable may take a range of values, rather than a single point estimate. Parameter uncertainty is generally addressed in risk assessment through gathering additional data, sensitivity analysis, or probabilistic modeling (discussed in Section 3.3.4).

- **Decision-rule uncertainty** is a type of uncertainty associated with policy and other choices made during the risk assessment. For example, the number of chemicals of potential concern (COPC) evaluated at a given tier of assessment may be reduced through use of a toxicity-weighted or risk-based screening analysis. In this example, the decision rule could be something like "Calculate the toxicity weighted emission for each chemical in the emissions inventory, rank the scores from highest to lowest and, starting with the highest score and working down, select as COPCs those chemicals that contribute to 99 percent of the cumulative toxicity weighted sum." This type of judgment introduces uncertainties about the contribution of the omitted air toxics to overall exposure or risk. As another example, risk managers may decide to select as chemicals for risk reduction efforts (i.e., the Chemicals

of Concern or COCs) only those COPCs that, individually, pose a risk above some specified level (e.g., one per million general population lifetime cancer risks). In this case, the decision rule would be "COCs are those COPCs which have a risk, on an individual chemical basis, of one in one million or greater." For any given risk assessment, some or all of these practices may be questioned, either on technical grounds (e.g., a risk number has been generated, but it is highly uncertain) or for policy reasons. The risk assessor needs to be sensitive these considerations when planning, conducting, and reporting the results of the risk assessment.

3.4.3 Sources of Variability in Air Toxics Risk Assessment

As noted previously, variability refers to true heterogeneity or diversity that occurs within a population or sample. Factors that lead to variability in exposure and risk include variability in contaminant concentrations in an environmental medium (e.g., air, water, soil) and differences in other exposure parameters such as ingestion rates and exposure frequencies.

Temporal and spatial variability in contaminant concentrations is often a very important aspect to consider in air toxics risk assessments. Spatial variability arises from many factors, including the release forms, physical and chemical dilution and transformation processes, and physical characteristics of the source or surrounding environment. Ecological receptors and humans may exhibit spatial variability in their contact with an exposure medium. Likewise, temporal variability can result from a variety of factors. For example, a source may only emit a chemical at specific times during the year (e.g., during the processing of a batch of product). Meteorological changes between seasons also can cause variable exposure (even though source emissions remain relatively constant). Because variability is an intrinsic property of the quantities being evaluated, it cannot be reduced by data gathering or refinements in models. However, understanding and/or analysis of variability are still important, especially during problem formulation. For example, it may be thought that certain air toxic emission source characteristics or potentially exposed populations are very heterogeneous and that a more robust description of the numbers and types of people at different risk levels is necessary to meet risk management decision goals

Confusion often arises about whether data are describing variability or uncertainty. For example, consider a group of 10,000 office workers who spend part of the time indoors at home and part of the time indoors at work. To assess the fraction of time spent indoors at a home or the office, a randomly chosen group of 100 office workers are asked to fill out a survey (resources preclude surveying all 10,000 people). Once we have our data, we draw a frequency diagram of the number of workers who spend specified amounts of time indoors at home and at the office. The picture we get clearly shows that different people spend different amounts of time inside at home and at the office – there is variability in the parameter for this population.

However, is our picture of variability correct (i.e., how certain are we that we have a good picture of the true variability of all 10,000 people)? Since we did not survey every possible worker and because some of the workers may have given incorrect responses, we have to admit to ourselves that there is probably some amount of uncertainty as to whether our frequency diagram is an accurate representation of variability in full worker population. In other words, we have developed an expression of variability that we think is uncertain. But only having a sense that our picture of variability may not be an accurate representation may not be enough (knowing just how uncertain our estimate of variability is may be important in our risk assessment).

Fortunately we have a variety of methods to look at the uncertainty in just one parameter (e.g., how variable is time spent indoors versus outdoors) and in the combination of parameters to provide estimates of exposure and risk. We can, for example, look at our data to see if patterns of time use vary for different subgroups of workers, or we can look for "outliers" (individuals with unusual patterns of indoor/outdoor time use). Alternatively, we could gather data from a larger sample of workers. Any of these would decrease the level of uncertainty in worker behavior, by providing more accurate representations of the variability of time usage for more clearly defined categories of workers. The newly developed worker categories would then be included in the exposure modeling.

3.4.4 Characterizing Uncertainty and Variability

Ideally, one would like to carry through the risk assessment, in a quantitative fashion, the uncertainty associated with each element in order to characterize the overall uncertainty associated with the final risk estimates. However, this is not always possible (because data are extremely limited) and, in some cases, may not be necessary (when all reasonable modeling assumptions and parameter values lead to the same recommendation). Nevertheless, it is always a good idea to provide some level of uncertainty analysis (be it qualitative, semi-quantitative, or quantitative). For example, one important use of uncertainty characterization can be to identify areas where a moderate amount of additional data collection might significantly improve the risk assessment, and hence the decision on the need for risk reduction or the risk reduction strategy to be used.

- **Qualitative characterization**. In a qualitative uncertainty analysis, a description of the uncertainties in each of the major elements of the risk analysis is provided, often with a statement of the estimated magnitude of the uncertainty (e.g., small, medium, large) and the impact the uncertainty might have on the risk element (e.g., the uncertainty is large and risk estimate is likely underestimated due to this element).

- **Quantitative characterization**. When appropriate, quantitative approaches to the uncertainty analysis are used to better characterize the uncertainty associated with the risk assessment. In this case, the first step is usually to characterize the probability distributions for key input parameter values (either using measured or assumed distributions). The second step would be to propagate parameter value uncertainties through the analysis using analytic (e.g., first-order Taylor series approximation) or numerical (e.g., Monte Carlo simulation) methods, as appropriate. Analytic methods might be feasible if there are a few parameters with known distributions and linear relationships. Numerical methods (e.g., Monte Carlo simulation) can be suitable for more complex relationships. "Two-dimensional" Monte Carlo analyses may be used where separate estimates of uncertainty and variability are available for some or all variables. Specific approaches are likely to be highly variable depending on the nature of the assessments being performed. Examples of approaches applied to a variety of assessments are provided in the reference list at the end of this chapter in Exhibit 3-8 (Hope, 1999; Moore et al., 1999; Smith, 1994).

- Both qualitative and quantitative uncertainty characterization is subject to scope-related limitations and uncertainty. For example, ecological risk assessments that are limited to primary effects evaluation for organisms or populations are uncertain with regard to secondary effects for communities or ecosystems. Similarly, human health assessments that

are restricted to the HAPs may ignore exposures and potential effects from other chemicals in the same emissions. Such uncertainties persist regardless of the assessment's refinement level (Tier). Their communication provides important contextual information for decision making.

Guidance developed by the National Council on Radiation Protection and Measurements[13] provides useful insights as to when to perform a quantitative uncertainty analysis in environmental risk assessments (Exhibit 3-6).

Exhibit 3-6. When to Perform a Quantitative Uncertainty Analysis
Quantitative uncertainty analysis is NOT recommended when: • Conservative, screening-level calculations indicate that the risk from potential exposure is clearly below regulatory or other risk levels of concern; • The cost of an action to reduce exposure is low; and/or • Data for characterizing the nature and extent of contamination or exposure are inadequate to permit even a bounding estimate (an upper and lower estimate of the expected value).
Quantitative uncertainty analysis IS recommended when: • An erroneous result in the exposure or risk estimate may lead to large or unacceptable consequences; • Whenever a realistic rather than a conservative estimate is needed; and/or • When it is important to identify those assessment components for which additional information will likely lead to improved confidence in the estimate of exposure or risk.
Source: NCRP. 1996. [13]

3.4.5 Tiered Approach to Uncertainty and Variability

Building on the approach outlined in Exhibit 3-6, the following description provides one possible tiered approach to deciding when and how to perform an uncertainty analysis.[14]

Single-Value Estimates of High-End and Central Tendency Risk. This approach starts with simple risk estimates using both representative and more conservative scenarios, models, and input values, using point estimates to represent each of the major parameters. This "deterministic" approach, which is described extensively in this document, may provide sufficient information for the risk management question being addressed. For example, if risks for a suitably defined high-end receptor are below levels of concern, then no additional uncertainty analysis (or risk analysis) may be needed to support a risk management decision. It is important to recall, however, that using single values for inputs, essentially ignores uncertainty and variability – information that may be very important for risk managers and the public.

Despite some limitations, single-value estimates or point estimates are an important tool in the risk assessment process. Single-value estimates are particularly useful as a screening tool to identify situations in which even highly conservative assumptions about exposure and other model parameters indicate low risk. (Note that EPA risk assessors are directed to provide, in Agency risk assessments, information about the range of exposures derived from exposure

scenarios and on the use of multiple risk descriptors [e.g., central tendency, high end of individual risk, population risk, important subgroups, if known] consistent with terminology in the Guidance on Risk Characterization, Agency risk assessment guidelines, and program-specific guidance.[7])

Qualitative Evaluation of Model and Scenario Sensitivity. Where the single-value high-end and central tendency point estimates do not provide sufficient information to make a risk management decision, qualitative analyses can be conducted to determine the range of values within which the risk estimate is likely to fall and the major factors that contribute to uncertainty. The sensitivity of the high-end and central tendency estimates to the plausible range of values for various parameters can usually be evaluated by conducting a manageable number of case studies using different parameter values and observing the resulting changes in risks. If scenario or model specification turns out to strongly affect risk estimates, a more refined analysis (see below) may be necessary. These may include Bayesian or decision-tree models.

Quantitative Sensitivity Analysis of High-End or Central Tendency Estimates. The risk assessor may want to evaluate the sensitivity of the point estimates of risks to variability and uncertainty in model input parameters. This may be done through sensitivity analysis or through the use of more detailed probabilistic methods (see Chapter 31). If sensitivity analyses are used, care must be taken to ensure that the combinations of parameter values that have the greatest impact on risks are identified.

Full Quantitative Characterization of Uncertainty and Uncertainty Importance. For many risk assessments, the systematic sensitivity analyses can provide sufficient information to provide reasonable confidence in the risk estimate. If they do not, the next step is explicit probability modeling, which is described in Chapter 31. Using such approaches, uncertainty and variability distributions can be defined for the major parameter values used in the derivation of the risk estimates. This approach is referred to as **parameter uncertainty analysis** and includes the following steps:[15]

- **Define the assessment endpoint** (i.e., the specific measure being evaluated). Examples would include an estimate of exposure concentration, hazard index, or a quantitative estimate of individual cancer risk.

- **List all potentially important uncertain parameters**. Include additional parameters, if necessary, to represent uncertainty in the assessment approach itself.

- **Specify the maximum conceivable range of possibly applicable values for each parameter with respect to the endpoint being assessed**.

- **For this range, specify a probability distribution for the parameter**. The probability distribution quantitatively expresses the state of knowledge about alternative values for the parameter (i.e., defines the probability that the true value of the parameter is located in various sub-intervals of the indicated range). These may include statistical distributions (e.g., "normal" or other distributions derived from data) or simpler approximations (triangular distributions defined by high, medium, and low values).

- **Determine and account for dependencies that are suspected to exist among parameters**. For example intake rate may not be independent of age or body weight.

- **Using either analytical or numerical procedures, propagate the uncertainty in the model parameters to produce a probability distribution for the assessment endpoint**. This results in the development of a probability distribution function (PDF) representing the state of knowledge for the endpoint.

- **Derive quantitative statements of uncertainty in terms of a probability or confidence interval about the assessment endpoint.**

- **Identify parameters according to their relative contribution to the overall uncertainty in the prediction of the value of the assessment endpoint.**

- **Present and interpret the results of the analysis.**

A full quantitative characterization of uncertainty requires a number of assumptions, including:

- The most important sources of uncertainty and variability are identified;
- The assumed probability distributions are correct; and
- The assumed dependence structure for different sources of uncertainty or variability is correct.

A comprehensive quantitative analysis may be a daunting task, particularly if a large number of sources, chemicals, receptors, exposure pathways, and endpoints, are of concern. Furthermore, the difficulty in justifying a large number of distributional assumptions (often based on professional judgement) needed for an uncertainty analysis might make such an analysis in itself unreliable.

In practice, the number of "tiers" available to the risk assessor may be limited. Often the practical choice is between using simple "screening" models (e.g., SCREEN3), and highly refined, fully parameterized modeling packages (e.g., ISCST3). In such cases, it may be easier to do a highly refined analysis with the state-of-the art models than to incrementally improve on the screening methods.

3.4.6 Assessment and Presentation of Uncertainty

The assessment and presentation of uncertainty is a very important component of the risk characterization. Based on the amount of information about sources and emissions and the degree of uncertainty associated with estimates of risk, decision-makers will weigh the importance of the risk estimates in the eventual decision. As noted previously, when the uncertainty analysis is qualitative in nature, a description of the uncertainties in each of the major elements of the risk analysis is usually described, often with a statement of the estimated magnitude of the uncertainty (e.g., small, medium, large) and the impact the uncertainty might have on the risk element (e.g., the uncertainty is large and risk estimate is likely underestimated). Important uncertainties to discuss include, but are not limited to:

- Scope issues such as the choice of air toxics, receptors, or endpoints that are evaluated in the assessment and the choice of air quality or multimedia models used to characterize exposure;
- Data quality issues, such as the quality of available sampling, emissions inventory, or toxicity data;
- Uncertainties inherent in the toxicity values for each substance used to characterize risk; and
- Uncertainties that are incorporated in the risk assessment when exposures to several substances across multiple pathways are summed.

When the analysis is more quantitative in nature, the description of uncertainty generally is separated into two parts:

- The first part is a summary of the values used to estimate exposure and risk (including model inputs), the range of these values, the midpoint or other descriptive values, and the value used to estimate exposure.
- The second part is a narrative discussion that identifies which variables or assumptions used in the risk assessment have the greatest potential to affect the overall uncertainty in the exposure assessment.

Chapter 13 provides additional discussion of how to assess and present uncertainty in an air toxics risk assessment. Exhibit 3-7 provides additional references on uncertainty analysis.

Example of a Six-step Process for Producing a Quantitative Uncertainty Estimate

Finkel (1990)[12] presents another example of a quantitative uncertainty analysis process:

1. Define the measure of risk (such as deaths, life-years lost, maximum individual risk [MIR], or population above an "unacceptable" level of risk). More than one measure of risk may result from a particular risk assessment; however, the uncertainty may be quantified or reached individually.
2. Specify "risk equations" that present mathematical relationships that express the risk measure in terms of its components. This step is used to identify the important variables in the risk estimation process.
3. Generate an uncertainty distribution for each variable or equation component. These uncertainty distributions may be generated by using analogy, statistical inference techniques, expert opinion, or a combination of these.
4. Combine the individual distributions into a composite uncertainty distribution.
5. Re-calibrate the uncertainty distributions. Inferential analysis could be used to "tighten" or "broaden" particular distributions to account for dependencies among the variables and to truncate the distributions to exclude extreme values.
6. Summarize the output clearly, highlighting the important risk management implications. Address specific critical factors, including (1) the implication of supporting a point estimate produced without considering uncertainty; (2) balance the costs of under- or over-estimating risks; and (3) unresolved scientific controversies, and their implications for research.

Exhibit 3-7. Additional References on Uncertainty Analysis
Burmaster, D.E. and Anderson, P.D. 1994. Principles of good practice for the use of Monte Carlo techniques in human health and ecological risk assessments. *Risk Analysis* 14: 477-481. Cullen, A.C. and Frey, H.C. 1999. *Probabilistic Techniques in Exposure Assessment.* New York: Plenum Press. Fayerweather, W.E., Collins, J.J., Schnatter, A.R., Hearne, F.T., Menning, R.A., and Reyner, D.P. 1999. Quantifying uncertainty in a risk assessment using human data. *Risk Analysis* 19: 1077-1090 Finkel, A.M. and Evans, J.S. 1987. Evaluating the benefits of uncertainty reduction in environmental health risk management. *Journal of the Air Pollution Control Association.* 37: 1164-1171. Frey, H.C. 1992. Quantitative analysis of uncertainty and variability in environmental policy making. Pittsburgh: Carnegie Mellon University. Hattis, D. and Burmaster, D.E. 1994. Assessment of variability and uncertainty distributions for practical risk assessments. *Risk Analysis* 14: 713-730. Hope, B. K. 1999. Assessment of risk to terrestrial receptors using uncertainty analysis - A case study. *Human and Ecological Risk Assessment* 5(1): 145-170. Moore, D.R.J., Sample, B.E., Suter, G.W., Parkhurst, B.R., and Teed, R.S. 1999. A probabilistic risk assessment of the effects of methylmercury and PCBs on mink and kingfishers along East Fork Poplar Creek, Oak Ridge, Tennessee, USA. *Environmental Toxicology and Chemistry* 18: 2941-2953. National Research Council (NRC). 1991. *Human Exposure Assessment for Airborne Pollutants.* Washington DC: National Academy Press. Roberts, S.M. 1999. Practical issues in the use of probabilistic risk assessment and its applications to hazardous waste sites. *Human and Ecological Risk Assessment.* 5(4): 729-868. Special Issue. Smith, R.L.. 1994. Use of Monte Carlo simulation for human exposure assessment at a Superfund site. *Risk Analysis* 14(4): 433-439. U.S. Environmental Protection Agency. 1985. *Methodology for Characterization of Uncertainty in Exposure Assessments.* Washington DC, EPA-600/8-85-009).

References

1. National Research Council (NRC). 1983. *Risk Assessment in the Federal Government: Managing the Process* (The "Red Book"). National Academy Press, Washington, D.C.

2. National Research Council (NRC). 1994. *Science and Judgment in Risk Assessment* (The "Blue Book"). National Academy Press, Washington, D.C.

3. Commission on Risk Assessment and Risk Management (CRARM). 1996. *Risk Assessment and Risk Management in Regulatory Decision-Making* (The "White Book"). Draft Report, Washington, D.C.

   CRARM. 1997. *Framework for Environmental Health Risk Management.* Final Report, Volume 1, Washington, D.C.

   CRARM. 1997. *Risk Assessment and Risk Management in Regulatory Decision-Making.* Final Report, Volume 2, Washington, D.C.

4. National Research Council (NRC). 1996. *Understanding Risk: Informing Decisions in a Democratic Society*. National Academy Press, Washington, D.C.

5. U.S. Environmental Protection Agency. 1984. *Risk Assessment and Management: Framework for Decision Making*. Office of Policy, Planning and Evaluation, Washington, D.C. EPA/600/985/002.

6. U.S. Environmental Protection Agency. *Superfund Risk Assessment.* Updated January 29, 2004. Available at: http://www.epa.gov/superfund/programs/risk/tooltrad.htm#gdhh. (Last accessed March 2004).

7. U.S. Environmental Protection Agency. 1995. *Policy for Risk Characterization at the U.S. Environmental Protection Agency*. Science Policy Council, Washington, D.C., March 1995. Available at: http://64.2.134.196/committees/aqph/rcpolicy.pdf.

8. U.S. Environmental Protection Agency. 1999. *Residual Risk Report to Congress*. Office of Air Quality Planning and Standards, Research Triangle Park, NC. March 1999. EPA/453/R99/001. Available at: http://www.epa.gov/ttnatw01/risk/risk_rep.pdf.

9. U.S. Environmental Protection Agency. 1997. *Guiding Principles for Monte Carlo Analysis*. Risk Assessment Forum, Washington, DC. March 1997. EPA/630/R97/001.

10. U.S. Environmental Protection Agency. 1988. *National Emission Standards for Hazardous Air Pollutants*. *Federal Register* 53(145):28496-28056, Proposed Rule and Notice of Public Hearing. July 28, 1988.

11. Hattis, D.B. and D.E. Burmaster. 1994. Assessment of variability and uncertainty distributions for practical risk analyses. *Risk Analysis* 14(5):713-730.

12. Finkel, A.M. 1990. *Confronting Uncertainty in Risk Management: A Guide for Decision-Makers.* Center for Risk Management, Resources for the Future. Washington, DC.

13. National Council on Radiation Protection and Measurements. 1996. *A Guide for Uncertainty Analysis in Dose and Risk Assessments Related to Environmental Contamination*. Bethesda, MD, May 1996. NCRP Commentary No. 14.

14. U.S. Environmental Protection Agency. 1997. *Policy for Use of Probabilistic Analysis in Risk Assessment*. Office of the Administrator, Washington, DC. May 15, 1997.

15. International Atomic Energy Agency. 1989. *Evaluating the Reliability of Predictions Made Using Environmental Transfer Models*. Vienna, Austria. IAEA Safety Series 100.

Chapter 4 Air Toxics: Chemicals, Sources, and Emissions Inventories

Table of Contents

4.1 Introduction 1

4.2 Air Toxics 1
- 4.2.1 Introduction to Air Toxics Chemical Lists 1
- 4.2.2 Hazardous Air Pollutants (HAPs) 4
- 4.2.3 Criteria Air Pollutants 5
- 4.2.4 Toxics Release Inventory (TRI) Chemicals 6
- 4.2.5 Toxic Chemicals That Persist and Which Also May Bioaccumulate 6
- 4.2.6 Other Chemicals 11

4.3 Sources of Air Toxics 11
- 4.3.1 Point Sources 12
- 4.3.2 Nonpoint Sources 13
- 4.3.3 On-Road and Nonroad Mobile Sources 14
- 4.3.4 Sources Not Included in the NEI or TRI 15
  - 4.3.4.1 Indoor Sources 15
  - 4.3.4.2 Natural Sources 18
  - 4.3.4.3 Formation of Secondary Pollutants 19
  - 4.3.4.4 Other Sources Not Included in NEI or TRI 20

4.4 Emissions Inventories 20
- 4.4.1 National Emissions Inventory (NEI) 21
- 4.4.2 Toxics Release Inventory (TRI) 25

References 29

4.1 Introduction

This chapter identifies the set of chemicals broadly, and most commonly, considered "air toxics." This section also describes the general categories of air toxics sources that emit these chemicals as well as the primary places where air toxics emissions information (e.g., databases that contain information on the location and nature of emissions released from various types of sources) used in air toxics risk assessments can be found. Section 4.2 discusses air toxics; Section 4.3 describes air toxics sources; and Section 4.4 describes air toxics emissions data sources.

The exhaustive lists of air toxics discussed in this section do not include all of the hazardous chemicals of public health concern. Note also that other forms of air pollution (e.g., odors) are not addressed in this Reference Library.

4.2 Air Toxics

Chapter 2 of this Volume introduced Hazardous Air Pollutants (HAPs), criteria air pollutants, Toxic Release Inventory (TRI) chemicals, and persistent, bioaccumulative, and toxic (PBT) chemicals and discussed the relationships among these various groupings. This section will revisit each of these groups to provide more detailed information related to the chemicals on each of those lists. A thorough understanding of the different types of chemicals that may be of interest for an assessment, as well as the nuances of the various ways chemicals are written into those lists, will be important for the risk assessment team to comprehend before the assessment begins in earnest.

The term "air toxics" is a generic term that could conceivably encompass literally anything in the air that poses harm to people or the environment. This Volume uses the term "air toxics" in this general sense. Thus, while the focus of most air toxics risk assessments will be on the 188 chemicals and chemical compounds listed as HAPs in the Clean Air Act (CAA) section 112(b), some assessment teams may wish to have a broader focus. The use of the term "air toxics" in this general sense is meant to provide for this flexibility. Ultimately, the scope of any assessment must clearly identify the chemicals that will be evaluated and the reason for their inclusion or exclusion in the evaluation.

4.2.1 Introduction to Air Toxics Chemical Lists

The various lists that are the focus of this volume were all derived directly from the Clean Air Act, the Emergency Planning and Community Right to Know Act, or a specific EPA initiative (e.g., the PBT initiative list of chemicals). It is important to understand that there is not always consistency among these various lists in either the naming of chemicals or the meaning of the names. For example, as noted in Chapter 2, "glycol ethers" are defined differently for the TRI and as HAPs (see box in Section 2.4.4).

Lists of toxic chemicals commonly provide the chemical identity by both a name and a unique identifying number, called a **Chemical Abstracts Registry Number**.[(a)] However, most

---

[a]CAS (Chemical Abstracts Service) is a division of the American Chemical Society. A CAS Registry Number (CAS number or CASRN) is assigned in sequential order to unique, new substances identified by CAS scientists for inclusion in the CAS REGISTRY database. Each CAS Registry Number is a unique numeric identifier; designates only one substance; and has no chemical significance. A CAS Registry Number is a numeric

chemicals have multiple synonyms (sometime dozens). Fortunately, every unique chemical has only one unique CAS number and one can always refer to this unique number to identify the compound in question. For example, toluene and methylbenzene are synonyms for the same compound (which is normally referred to as toluene). However, there is only one CAS number for the compound: 108-88-3. No matter where one is in the world or what name is attached to a chemical, there is unanimity of identity through the CAS numbering system.

When there is any question about what a particular chemical name means, it is always advisable to try to pinpoint the identity through use of the CAS number. For example, a risk assessment team may ask for air sampling analysis for the HAP acetaldehyde (CAS number 75-07-0); however, when they receive the analytical lab report, acetaldehyde is not reported. A quick scan of the CAS numbers reported by the lab lists the CAS number 75-07-0 next to the name "ethanal." Ethanal is a synonym for acetaldehyde and, hence, has the same CAS number. EPA's *Handbook for Air Toxics Emission Inventory Development* includes a list (Appendix C) of synonyms and CAS numbers for HAPs that is helpful in overcoming the nomenclature obstacle.[1] (Note, however, that there are nuances even beyond this simplistic description. For example, some chemicals have one CAS number for their pure form and a different CAS number for a technical grade. A knowledgeable chemist can usually identify and clarify these issues.)

Some of the entries on chemical lists are for large groups of compounds and not just one single substance. For example, one of the HAPs is listed in the CAA as "polychlorinated biphenyls (aroclors)" and is most commonly referred to as PCBs. This listing is not for one single substance but, rather, for any one or a mixture of any of the 209 possible chemicals that are themselves PCBs. As another example, the pesticide "2,4-D" is written into the list of HAPs as "2,4-D (salts and esters)." This listing includes any possible salt of 2,4-D and any possible ester of 2,4-D. In our earlier lead example, the lead compound listing includes any compound known to exist in or be emitted to the environment that contains a lead molecule as part of the compound's molecular structure (a potentially huge number of possibilities). Another important group of chemicals is called "POM" for polycyclic organic matter. This includes organic compounds with more than one benzene ring, and which have a boiling point greater than or equal to 100° C (e.g., polycyclic aromatic hydrocarbons (PAHs) such as benzo(a)pyrene).

In reality, most risk assessments will deal with a relatively small number of chemicals because either the sources in a given place are releasing only a limited number of chemicals or the ability to model or monitor the numerous chemicals present is limited by the available inventories or monitoring/analytical methods, respectively.

In the initial stages of the assessment, risk assessors often sort the chemicals of interest into groups that, generally, have similar physical and/or chemical properties. This is a helpful thing to do as a way of making some educated guesses about how chemicals are likely to behave in the environment. The groupings also help an assessment team to plan for the types of sampling and analysis methods that will be needed, because the sampling and analytical methods tend to be broken out along these same lines. In general, all air toxics can be broadly categorized into three main groups, organic chemicals, inorganic chemicals, and organometallic compounds as follows:

---

identifier that can contain up to nine digits, sometimes divided by hyphens into three parts. See http://www.cas.org/faq.html for more information.

Organic Chemicals

Organic chemical compounds are composed of carbon in combination with other elements such as hydrogen, oxygen, nitrogen, phosphorous, chlorine, and sulfur (not including carbonic acid or ammonium carbonate). Organic compounds can generally be split into two different groups, based on their propensity to evaporate. The following such groupings are commonly employed by analytical chemistry laboratories for purposes of sample analysis.

- **Volatile Organic Compounds (VOCs)**. These are organic chemicals that have a high vapor pressure and tend to have low water solubility.[b] Simply put, VOCs have a high propensity to evaporate and remain airborne. Many VOCs are human-made chemicals that are used and produced in the manufacture of paints, pharmaceuticals, and refrigerants, as industrial solvents, such as trichloroethylene, or produced as by-products, such as chloroform produced by chlorination in water treatment. VOCs are often also components of petroleum fuels (e.g., benzene), hydraulic fluids, paint thinners, and dry cleaning agents.[c]

  A subgroup of VOCs is termed **Carbonyl Compounds** and includes chemicals such as formaldehyde and acetaldehyde. While such chemicals are themselves VOCs due to their high vapor pressure, they are often grouped as a separate class from the VOCs because of the special sampling and analytical methods necessary to measure them in air.

- **Semivolatile Organic Compounds (SVOCs)**. SVOCs are organic chemicals that have a lower vapor pressure than VOCs and, thus, have a lower propensity to evaporate from the liquid or solid form. Once airborne, they also tend to more readily condense out of the gas phase. Examples of SVOCs include most organic pesticides (e.g., chlordane), and certain components of petroleum, such as polycyclic aromatic hydrocarbons. Note that the demarcation between SVOCs and VOCs is not exact. For example, the two separate air sampling and analytical methods for VOCs and SVOCs will both usually detect naphthalene when present, indicating that this chemical is on the lower end of the VOC scale of volatility and on the higher end of the SVOC scale of volatility. In general, as chemicals increase in molecular weight and/or polarity, they become more SVOC-like.

Inorganic Chemicals

This group includes all substances that do not contain carbon and includes a wide array of substances such as:

- Metals (e.g., mercury, lead, and cadmium) and their various salts (e.g., mercury chloride);
- Halogens (e.g, chlorine and bromine);
- Inorganic bases (e.g., ammonia); and
- Inorganic acids (e.g., hydrogen chloride, sulfuric acid).

---

[b] The regulatory definition of VOC does not identify vapor pressure as a consideration. See 40 CFR 51.100(s).

[c] "VOC" refers to volatile organic compounds that contribute to ozone formation as defined by 40 CFR 50.100(s) as ozone precursors. VOC is a subset of VOCs. VOC emissions inventory information is sometimes used to derive estimates for specific chemicals; when this is done, the VOC number is said to have been speciated.

Organometallic Compounds

This group is comprised of compounds that are both organic and metallic in nature. The alkyl lead compounds that were added to gasoline to enhance its properties can be used for illustration. "Alkyl" refers to the organic portion of a compound which is attached to the inorganic metal lead. The result is a so-called "organometallic" material, a hybrid of both metallic and organic. (Note that salts, such as sodium benzoate, are usually classified as an organic chemical, rather than an organometallic compound.)

An understanding of the general characteristics of organic chemicals, inorganic chemicals and organometallic compounds will aid in planning a risk assessment and developing an appropriate analysis strategy. For example, most VOCs tend to remain airborne and also do not tend to bioaccumulate to the same extent as some of the non-volatile chemicals. Thus, if an assessment were being planned to evaluate the impact of a source from which only VOCs were released, it becomes less likely that a multipathway risk analysis will be necessary (since VOCs do not tend to migrate into soil or water and do not tend to bioaccumulate as strongly in living tissue).

In addition, the sampling and analytical methods available to test for chemicals in environmental media are generally broken out along the same chemical groupings noted above. Thus, if one were interested in testing for airborne chlordane (an SVOC), a VOC monitoring method would not be used. Detailed information on available monitoring methods and the chemicals for which they have been validated is provided in Chapter 10.

In air toxics studies, both individual substances and mixtures of substances are of interest. Particulate matter (PM), for example, is almost never comprised of just one substance; instead, PM is usually made up of numerous individual substances (sometimes in the hundreds). Both the physical and chemical nature of a mixture will influence the fate and transport of the chemicals in the environment as well as the potential for the mixture to cause harm. For example, a toxic chemical adsorbed onto the surface of a relatively large particle (> 10 microns in diameter) will usually be trapped in the upper portion of the respiratory system and either coughed/sneezed out of the body or swallowed. The same chemical adsorbed onto a very small particle (< 2.5 microns in diameter) has a much higher likelihood of being inhaled into the deep lung. As we will see in later chapters, both the route of exposure (in this example, ingestion or inhalation) as well as the toxic properties of the chemical in question are important determinants of potential harm.

4.2.2 Hazardous Air Pollutants (HAPs)

The HAPs are a group of 188 specific chemicals and chemical compounds and are identified in Section 112(b) of the CAA. The Agency provides additional information on the HAPs online.[2] HAPs are pollutants known to cause or suspected of causing cancer or other serious human health effects or ecosystem damage. They include individual organic and inorganic compounds and pollutant groups closely related by chemical structure (e.g., arsenic compounds, cyanide compounds, glycol ethers, polycyclic organic matter) or emission sources (e.g., coke oven emissions). EPA may add or remove pollutants from the HAP list as new information becomes available. A full list of the HAPs is provided in Appendix A.

When people talk about "air toxics risk assessment," they generally mean assessments of risks associated with one or more of the HAPs. This is largely because of the CAA listing of 188

HAPs and its requirement under Section 112(f)(2) (Residual Risk) that EPA assess the risks associated with HAPs that remain after the application of the Maximum Achievable Control Technology (MACT) standards (Section 112(d) of the Act). However, given that this is a relatively short list of chemicals, many communities may want to go beyond this list when assessing risk. It is for this reason, that assessors and other stakeholders must clearly identify why they are conducting an "air toxics" risk assessment and what they want to include in that assessment.

In its Integrated Urban Air Toxics Strategy, EPA identified a subset of 33 HAPs as those posing the greatest risk in urban areas (see Section 2.2.1). These 33 HAPs were selected based on a number of factors, including toxicity-weighted emissions, monitoring data, past air quality modeling analysis, and a review of existing risk assessment literature.

The national-scale assessment for 1996 (see Section 2.3.2) focused on 32 of these 33 Urban HAPs (dioxin was omitted) and also includes diesel particulate matter, which is used as a surrogate measure of diesel exhaust. EPA recently concluded that diesel exhaust is likely to be carcinogenic to humans by inhalation at environmental levels of exposure. Diesel exhaust is addressed in several regulatory actions and diesel particulate matter plus diesel organic gases are listed by EPA as a mobile source air toxic (see Section 4.3.3 below).

4.2.3 Criteria Air Pollutants

The "criteria air pollutants" are six substances regulated pursuant to Title I of the CAA, for which "criteria documents" are developed by the Agency prior to national standard setting decisions. There are already national ambient air quality standards (NAAQS) in place for each of these pollutants as well as established regulatory programs and activities in place to meet those standards. However, they are discussed here because there is some crossover between the realm of HAPs and criteria pollutants. The more important crossover issues are discussed below.

- **Particulate matter**. NAAQS have been established for particles with an aerodynamic diameter less than or equal to 10 microns (called PM_{10}) and particulate matter with an aerodynamic diameter less than or equal to 2.5 microns (called $PM_{2.5}$). As noted above, however, PM can be made up of as little as one or a few, or many hundreds of individual chemicals. In many cases (and depending on the source of the PM), any number of specifically listed HAPs may be a part of the PM mix. It is for this reason that risk assessors may opt to evaluate the composition of PM and to include the identified chemicals in risk calculations.

  For example, it is possible to collect samples of PM_{10} for purposes of determining the types and amounts of individual substances contained in the particles. The risks posed by those individual chemicals may then be estimated for the inhalation route of exposure. Because particles with diameters greater than 10 microns are not generally respirable, analysts usually select a PM_{10} monitor to capture samples for risk assessment purposes rather than a total suspended particulate (TSP) sampler, because TSP would capture larger particles that do not penetrate very far into the respiratory tract (thus leading to an overestimate in inhalation risk associated with the specific pollutants studied). Note that this would not be true for particle-bound chemicals that exert their toxic effects on the nasal passages.

- **Ozone and other criteria pollutants**. Certain other criteria pollutants are not specifically listed as HAPs, but HAPs may lead to their formation or they may lead to HAP formation. For example, ozone is produced by the interaction of certain VOCs, oxides of nitrogen (called NO_x), and sunlight. As noted previously, many of the HAPs are VOCs and may play a role in ozone formation. In contrast, sulfur dioxide is a criteria pollutant that can be transformed in the environment into sulfuric acid which, in turn, may become part of a listed HAP (e.g., cadmium sulfate). In general, the criteria pollutants ozone, nitrogen dioxide, sulfur dioxide, carbon monoxide are not usually considered in air toxics risk assessments.

4.2.4 Toxics Release Inventory (TRI) Chemicals

Data on TRI chemicals are reported pursuant to Section 313 of the Emergency Planning and Community Right-To-Know Act (EPCRA) of 1986 and Section 6607 of the Pollution Prevention Act of 1990 (PPA). EPCRA and the PPA are intended to inform communities and citizens about chemical hazards in their areas. EPA and states are required to collect data annually on releases (to each environmental medium) and waste management methods (e.g., recycling) of certain toxic chemicals from industrial facilities, and to make the data available to the public in the TRI.[3] EPCRA Section 313(d) permits EPA to list or delist chemicals based on certain criteria. In a 1994 rulemaking, EPA added 286 chemical categories to the TRI chemical list. The TRI chemicals are listed in 40 CFR Section 372.65, and information about the 667 currently-listed TRI chemicals is provided online.[4]

The current TRI chemical list contains 582 individually listed chemicals and 30 chemical categories (including three delimited categories containing 58 chemicals), for a total of 612 separate chemicals. If the members of the three delimited categories are counted as separate chemicals then the total number of chemicals and chemical categories is 667 (i.e., 582 + 27 + 58). The TRI list of toxic chemicals includes most (180) of the HAPs. Similar to the HAPs, the TRI chemicals include VOCs, SVOCs, inorganic compounds, and organometallic compounds.

The utility of the TRI for air toxics risk assessment is two fold. First, it provides a broader perspective of industrial emissions than the HAP list because it includes information on air releases of many hundreds of additional chemicals. Second, accessing TRI information is extremely quick and easy. Using the TRI Explorer search engine (http://www.epa.gov/tri/tridata/index.htm), one may quickly identify the location of emissions sources and the identity and quantity of chemicals released to the air. The data is also updated annually (as opposed to the National Emissions Inventory (NEI), a nationwide inventory of emissions developed by EPA, which is only updated triennially). However, other characteristics of the TRI data may limit their use for risk assessments (see Section 4.4.2).

4.2.5 Toxic Chemicals That Persist and Which Also May Bioaccumulate

Some toxic compounds have the ability to persist in the environment for long periods of time and may also have the ability to build up in the food chain to levels that are harmful to human health and the environment. For example, releases of metals from a source may deposit out of the air onto the ground where they remain in surface soils for long periods of time. Children playing in the area may ingest this contaminated dirt through hand-to-mouth behaviors. The chemicals in the dirt may also be taken up into plants through the roots and accumulate in foraging animals.

EPA's challenge in reducing risks from this category of toxic air pollutants stems from this ability to transfer from air, to sediments, water, land, and food; to linger for long periods of time in the environment; and for some substances, their ability to travel long distances. Many of these chemicals (e.g., DDT) have been banned for use in the U.S. As such, there should be no active air emissions of these chemicals (although releases into the air are still possible, e.g., by resuspension of previously contaminated soil). However some, such as mercury, are still in use today. A number of lists of these persistent and bioaccumulative chemicals have been developed through international and internal EPA efforts (see Exhibit 4-1). A number of the HAPs appear on one or more of these lists.

Exposure to persistent and bioaccumulative air toxics through a pathway other than inhalation of contaminated air is termed **an indirect exposure pathway** because contact with the chemical occurs in a medium that is not the original medium to which the chemical was released (i.e., air). In contrast, a **direct exposure pathway** is one in which contact occurs with the chemical in the medium to which it was originally released. When exposure of a person to a chemical (or chemicals) occurs through more than one pathway, a **multipathway analysis** may be considered.

In air toxics risk assessment, the inhalation pathway is commonly assessed (i.e., the release of a chemical to air and human exposure through breathing that air). However, indirect exposure pathways are usually assessed for a limited set of chemicals released to the air. EPA has identified a preliminary set of HAPs for which indirect exposure pathway analyses should generally be conducted for situations involving significant emissions of these chemicals in a study area. This new list of chemicals is termed **Persistent Bioaccumulative HAP Compounds (PB-HAP Compounds)** (Exhibit 4-2); however, all of the PB-HAP compounds occur on one or more of EPA's existing lists of PBT chemicals. The designation "PB-HAP" was developed to distinguish this list from the existing lists of PBT chemicals (Exhibit 4-1) and specifically to clarify that chemicals on this new list are:

- HAPs;
- Relatively persistent in the environment; and
- For some chemicals, have a strong propensity to bioaccumulate and/or biomagnify.[(d)]

This preliminary list of PB-HAPs was derived primarily on the basis of human health concerns. It does not consider direct contact by plants or inhalation by animals. Additional HAPs may be identified as EPA gains more familiarity with ecological risk assessments for air toxics. Appendix D describes the process by which EPA identified the list of PB-HAPs.

---

[d]Biomagnification is the process whereby certain substances transfer up the food chain and increase in concentration. Chemicals that biomagnify tend to accumulates to higher concentration levels with each successive food chain level. Biomagnification is a particular concern for ecological risk assessment.

Exhibit 4-1. "Lists" of Toxic Chemicals that Persist and Which Also May Bioaccumulate
LRTAP chemicals – The United States signed protocols on Persistent Organic Pollutants (POPs) and heavy metals pursuant to the Convention on Long-Range Transboundary Air Pollution (LRTAP) in June 1998 at a ministerial meeting in Aarhus, Denmark. Sixteen POPs and three metals are regulated (http://www.epa.gov/oppfead1/international/lrtap2pg.htm):
• aldrin • cadmium • chlordane • dieldrin • endrin • hexabromobiphenyl • kepone (chlordecone) • mirex • toxaphene • hexachlorobenzene • heptachlor • lead • mercury • polychlorinated biphenyls (PCBs) • dichlorodiphenyltrichloroethane (DDT) • lindanedioxins (polychlorinated dibenzo-p-dioxins) • furans (polychlorinated dibenzofurans) • hexachlorobenzene • polycyclic aromatic hydrocarbons
PBT Chemicals – EPA has identified the following priority persistent, bioaccumulative, and toxic (PBT) chemicals and has developed the PBT program to address the cross-media issues associated with these chemicals (http://www.epa.gov/opptintr/pbt/):
• aldrin/dieldrin • mercury and its compounds • benzo(a)pyrene • mirex • chlordane • octachlorostyrene • DDT • dichlorodiphenyldichloroethane (DDD) • dichlorodiphenyldichloroethylene (DDE) • PCBs • hexachlorobenzene • dioxins and furans • alkyl-lead • toxaphene
Great Lakes Priority Substances. In keeping with the obligations of the Great Lakes Water Quality Agreement, Canada and the United States on April 7, 1997, signed the "Great Lakes Binational Toxics Strategy: Canada-United States Strategy for the Virtual Elimination of Persistent Toxic Substances in the Great Lakes" (http://www.epa.gov/glnpo/p2/bns.html). This Strategy seeks percentage reductions in targeted persistent toxic substances so as to protect and ensure the health and integrity of the Great Lakes ecosystem. The list of "Level 1" substances is identical to EPA's priority PBT pollutants.
Great Waters Pollutants of Concern. The 1990 Clean Air Act Amendments established research and reporting requirements related to the deposition of hazardous air pollutants to the Great Lakes, Lake Champlain, Chesapeake Bay, and certain other "Great Waters." The Program has identified the following pollutants of concern (http://www.epa.gov/airprogm/oar/oaqps/gr8water/index.html):
• cadmium and cadmium compounds • chlordane • DDT/DDE • dieldrin • hexachlorobenzene • α-hexachlorocyclohexane • lindane (γ-hexachlorocyclohexane) • lead and lead compounds • mercury and mercury compounds • PCBs • polycyclic organic matter • tetrachlorodibenzo-p-dioxin (dioxins) • tetrachlorodibenzofuran (furans) • toxaphene • nitrogen compounds

Exhibit 4-1 (continued)

TRI PBT chemicals. EPA has published two final rules that lowered the Toxics Release Inventory (TRI) reporting thresholds for certain persistent bioaccumulative and toxic (PBT) chemicals and added certain other PBT chemicals to the TRI list of toxic chemicals (http://www.epa.gov/tri/lawsandregs/pbt/pbtrule.htm). The following PBT chemicals are subject to reporting at lowered thresholds:

- dioxin and dioxin-like compounds
- lead compounds
- mercury compounds
- polycyclic aromatic compounds
- aldrin
- benzo(g,h,i)perylene
- chlordane
- heptachlor
- hexachlorobenzene
- isodrin
- lead
- mercury
- methoxychlor
- octachlorostyrene
- pendimethalin
- pentachlorobenzene
- PCBs
- tetrabromobisphenol A
- toxaphene
- trifluralin

Waste Minimization Priority Chemicals. EPA's National Waste Minimization Partnership Program focuses on reducing or eliminating the generation of hazardous waste containing any of 30 Waste Minimization Priority Chemicals (WMPCs). This list replaces the list of 53 chemicals EPA identified in 1998 (*Notice of Availability: Draft RCRA Waste Minimization Persistent, Bioaccumulative and Toxic (PBT) Chemical List*, Federal Register 63(216): 60332-60343, November 9, 1998). Twenty six of the chemicals in the current list were also in the draft list published in 1998. The remaining four chemicals on the current list were added in response to comments and new information EPA received from the public regarding the Agency's methodology for selecting the 53 chemicals in the draft list (http://www.epa.gov/epaoswer/hazwaste/minimize/chemlist.htm).

- 1,2,4-trichlorobenzene
- 1,2,4,5-tetrachlorobenzene
- 2,4,5-trichlorophenol
- 4-bromophenyl phenyl ether
- acenaphthene
- acenaphthylene
- anthracene
- benzo(g,h,i)perylene
- dibenzofuran
- dioxins/furans
- endosulfan, alpha and endosulfan, beta
- fluorene
- heptachlor and heptachlor epoxide
- hexachlorobenzene
- hexachlorobutadiene
- hexachlorocyclohexane, gamma-
- hexachloroethane
- methoxychlor
- naphthalene
- PAH group (as defined in TRI)
- pendimethalin
- pentachlorobenzene
- pentachloronitrobenzene
- pentachlorophenol
- phenanthrene
- pyrene
- trifluralin
- cadmium and cadmium compounds
- lead and lead compounds
- mercury and mercury compounds

Exhibit 4-2. PB-HAP Compounds			
PB-HAP Compound	**Pollution Prevention Priority PBTs**	**Great Waters Pollutants of Concern**	**TRI PBT Chemicals**
Cadmium compounds		X	
Chlordane	X	X	X
Chlorinated dibenzodioxins and furans	X[(a)]	X	X[(b)]
DDE	X	X	
Heptachlor			X
Hexachlorobenzene	X	X	X
Hexachlorocyclohexane (all isomers)		X	
Lead compounds	X[(c)]	X	X
Mercury compounds	X	X	X
Methoxychlor			X
Polychlorinated biphenyls	X	X	X
Polycyclic organic matter	X[(d)]	X	X[(e)]
Toxaphene	X	X	X
Trifluralin			X

[(a)] "Dioxins and furans" ("" denotes the phraseology of the source list)
[(b)] "Dioxin and dioxin-like compounds"
[(c)] Alkyl lead
[(d)] Benzo[a]pyrene
[(e)] "Polycyclic aromatic compounds" and benzo[g,h,i]perylene

4.2.6 Other Chemicals

The chemicals included in the various lists of air toxics described above – HAPs, criteria pollutants, TRI chemicals, and toxic chemicals that persist and which also may bioaccumulate – do *not* represent all of the chemicals potentially emitted to air in a given place. EPA is required to maintain an inventory, known as the "Toxic Substances Control Act (TSCA) Inventory," of each chemical substance which may be legally manufactured, processed, or imported in the U.S. The TSCA inventory currently contains over 75,000 chemicals (see: "enforcement programs" at http://www.epa.gov/compliance/civil/programs/tsca/). At best, we have the capability to assess only a few hundred in detail. As noted previously, this does not imply that risk assessments are always missing important information. To the contrary, the actual number of chemicals used in significant amounts and released to air are relatively small compared to the number of chemicals known. Nevertheless, it is important to keep in mind that the ability to evaluate air toxics releases is limited by current technology, the lack of toxicity information for all but a relatively small number of chemicals and, in some cases, costs (e.g., a single sample for certain analytes such as dioxin can cost upwards of $1,000 per sample, making multiple sampling events cost prohibitive).

The HPV Challenge Program

EPA, in partnership with industry and environmental groups, recently created a voluntary chemical testing effort, the high production volume (HPV) Challenge Program. This program was developed to make publicly available a complete set of baseline health and environmental effects data on HPV chemicals (those manufactured in, or imported into, the United States in amounts equal to or exceeding 1 million pounds per year). Information on HPV chemicals is available at http://www.epa.gov/chemrtk/rtkfacts.htm.

4.3 Sources of Air Toxics

Many anthropogenic and natural activities are sources of air pollutants. Examples of human activities that result in the release of air toxics include:

- Fuel combustion activities in power plants, factories, automobiles, and homes;
- Biomass burning and other agricultural activities;
- Use of consumer products, such as pesticides and cleaning agents;
- Commercial activities, such as dry cleaning; and
- Industrial activities, such as petroleum refining, chemical manufacture, and metal plating.

Sources of air toxics can be categorized in various ways – whether they occur indoors or out, whether they are stationary or mobile, by the amount of chemicals they release, or by other approaches. For the purposes of this discussion, air toxics have been placed into several major groupings that track EPA's programs and emissions inventories. Note that some differences in terminology exist between the CAA and the NEI (Exhibit 4-3).

- Point sources;
- Nonpoint sources;
- On-road mobile sources;
- Nonroad mobile sources;
- Indoor sources;
- Natural sources; and
- Exempt sources.

The first four categories are groupings of emission sources of HAPs and criteria air pollutants in EPA's **National Emissions Inventory (NEI)**. The NEI is a nationwide inventory of emissions that has been developed by EPA with input from numerous state, local, and tribal (S/L/T) air agencies. The NEI is discussed in more detail as a source of quantitative emissions release data in Section 4.4.1 below. For detailed information on NEI, refer to EPA's main NEI web page.[5] NEI summaries were posted in October 2003.

Exhibit 4-3. Terminology Related to Groupings of Source Types

Source Type	How Defined in CAA	How Reported in NEI
Point source – Major	Point source – Major	Point source
Point source – Area	Point source – Area	Point source if location coordinates reported Area source if coordinates not reported
Nonpoint source	Nonpoint source	Area
Mobile source – On-road	Mobile source – On-road	Modeled
Mobile source – Nonroad	Mobile source – Nonroad	Modeled or estimated
Indoor	Not defined	Not reported
Natural	Not defined	Not reported
Exempt	Not defined	Not reported

4.3.1 Point Sources

Point sources of air toxics are stationary sources (i.e., sources that remain in one place) that can be located on a map. A large facility that houses an industrial process is an example of a point source – the facility and its emission release points (e.g., stacks, vents, fugitive emissions from valves) are stationary, and the emission rates of air toxics can be characterized, either through direct measurements, such as stack monitoring, or indirect methods, such as engineering estimates based on throughput, process information, and other data. The CAA divides point sources into two main categories primarily on the basis of annual emissions rates:

- **Major sources** are defined in Section 112(a)(1) as "any source or group of stationary sources located within a contiguous area and under common control that emits or has the potential to emit, considering controls, in the aggregate, 10 tons per year (tpy) or more of any hazardous air pollutant or 25 tpy or more of any combination of hazardous air pollutants."

- **Area sources** are defined in Section 112(a)(2) as "any stationary source of hazardous air pollutants that is not a major source. For purposes of this section, the term 'area source' shall not include motor vehicles or nonroad vehicles subject to regulation under Title II." Examples of area sources include dry cleaners, gas stations, chrome electroplaters, and print shops. Though emissions from individual area sources may be relatively insignificant in human health terms, collectively their emissions can be quite significant, particularly where large numbers of sources are located in heavily populated areas. Note that sources that are classified as "area sources" pursuant to the CAA may be reported in the NEI as "point sources" if they can be located on a map.

Many sources of HAPs are subject to **National Emission Standards for Hazardous Air Pollutants (NESHAPs)** pursuant to Section 112 of the CAA. This Section of the CAA directs EPA to issue regulations listing categories and subcategories (commonly referred to collectively as **source categories**) of major sources and area sources of HAPs and to develop standards for each listed category and subcategory.[6] EPA periodically updates the list of source categories (see Appendix E).[7]

Physical Forms of Emissions	
Gas	Emissions that are distinguished from solid and liquid states
Fume	Tiny particles trapped in vapor in a gas stream
Mist	Liquid particles measuring 40 to 500 micrometers that are formed by condensation of vapor
Particulate Matter (and Aerosols)	Fine liquid or solid particles

Air pollutants can be found in all three physical phases: solid, liquid, or gaseous. The distinct chemical and physical attributes of each phase contribute to the pollutant's transport and fate. For example, as reported in the *Mercury Study Report to Congress*,[8] elemental mercury vapor is not thought to be susceptible to any major process of direct deposition to the earth's surface due to its relatively high vapor pressure and low water solubility. Therefore, it is carried by the wind and subsequently dispersed throughout the atmosphere. However, divalent mercury, in either vapor or particulate phase, is thought to be subject to much faster atmospheric removal, and is expected to be deposited near its source. For further details on fate and transport analysis, see Chapter 8.

As noted in Chapter 2, EPA regulates stationary sources in a two-phase process. First, EPA issues technology-based MACT standards that require sources to meet specific emissions limits. The emission limits are typically expressed as maximum emission rates, or minimum percent emission reductions, for specific pollutants from specific processes. In the second phase, EPA applies a risk-based approach to assess how well MACT emissions limits reduce health and environmental risks. Based on these **residual risk assessments**, EPA may implement additional standards to address any significant remaining, or residual, health or environmental risks (see Chapter 2 for a more detailed discussion of the MACT and residual risk programs).

Area sources may be subject to either MACT or **Generally Available Control Technology (GACT)** standards. GACT standards are generally less stringent than MACT standards. Area sources subject to MACT standards include Commercial Sterilizers using Ethylene Oxide, Chromium Electroplaters and Anodizers, Halogenated Solvents Users, and Asbestos Processors.

4.3.2 Nonpoint Sources

The term nonpoint source refers to smaller and more diffuse sources within a relatively small geographic area. In the context of EPA's NEI, nonpoint sources of air toxics are stationary sources for which emissions estimates are provided as an aggregate amount of emissions for all similar sources within a specific local geographic area, such as counties or cities, rather than on a facility- or source-specific basis. Emission estimates for nonpoint sources are generated using "top-down" methods, when detailed information at the local level is lacking. Instead, the total emissions over a large geographic area (e.g., n tons in the northeastern states) are allocated to the

local level (e.g., x percent is assigned to locality 1, y percent is assigned to locality 2, and so on). Note that for the purposes of this discussion, the nonpoint source category includes only stationary sources and does *not* include mobile sources.

Source-specific information may be available for *some* (but not all) of the specific facilities within a certain nonpoint source type. Area sources may be reported as either point or nonpoint sources in the NEI. If a state or local agency reports an area source emission as a point source, then the NEI retains the area source emission as a point source. The NEI does not aggregate point area sources as nonpoint sources, and **EPA has taken steps to avoid "double-counting" of emissions in the point and nonpoint source inventories**.

To compile nonpoint estimates for a category, the EPA first estimates county level emissions for nonpoint source categories. Then EPA replaces nonpoint EPA generated estimates with state and local agency and tribal estimates. If a state or local agency or tribe includes point source estimates for an EPA generated nonpoint source category, EPA removes the nonpoint estimate that it had generated and the point source inventory contains the S/L/T estimate. For example, in the Denver area, the State of Colorado inventories dry cleaners and service stations as point sources. The NEI contains point sources estimates for these two categories in the six county area of Denver and the NEI does not contain nonpoint estimates for these two categories. Dry cleaners and service station emissions are contained in the NEI nonpoint inventory for the other fifty counties on Colorado.

A variety of sources are categorized as nonpoint sources in the NEI, including some small industrial/commercial processes (e.g., small dry cleaning facilities, hospital sterilization facilities, and dental offices). Additional nonpoint sources that contribute to air pollution are agricultural activities, residential trash and yard-waste burning, wood stoves and fireplaces, releases from spills and other accidents, and volatilization and resuspension of pollutants from contaminated sites. Examples of agricultural activities contributing to air pollution are biomass burning (e.g., for land clearing) and the application of fertilizers and pesticides. The open burning of forests (including wildfires) are also categorized as nonpoint sources. (Note that forest fires are generally considered for the purposes of the NEI to be an anthropogenic source of air toxics because they are assumed to be directly or indirectly, for purposes of the NEI, caused by man.)

Some nonpoint sources emit HAPs and are subject to NESHAPs pursuant to Section 112 of the CAA (see Section 4.3.1 above for more information on NESHAPs). These nonpoint sources are area sources in that they emit less than 10 tpy of a single air toxic or less than 25 tpy of a mixture of air toxics. For example, facilities that perform perchloroethylene dry cleaning belong to a source category that is subject to NESHAPs.

4.3.3 On-Road and Nonroad Mobile Sources

Mobile sources pollute the air with fuel combustion products and evaporated fuel. These sources contribute greatly to air pollution nationwide and are the primary cause of air pollution in many urban areas. Section 202(l) of the CAA gives EPA the authority to regulate air toxics from motor vehicles. Based on 1996 National Toxics Inventory data (the NTI is the former name of the air toxics portion of the current NEI), mobile sources contributed 2.3 million tpy or about half of all air toxics emissions in the U.S. Mobile sources emit hundreds of air pollutants – for example, exhaust and evaporative emissions from mobile sources contain more than 700

compounds. EPA's Final Rule, *Control of Emissions of Hazardous Air Pollutants from Mobile Sources*, commonly known as the "Mobile Source Air Toxics" (MSAT) rule,[9] identified 21 compounds as HAPs emitted by mobile sources (see Chapter 2). All of these compounds except diesel particulate matter and diesel exhaust organic gases (DPM + DEOG) are included on the CAA Section 112 HAPs list. Although some mobile source air toxics are TRI chemicals, mobile sources are not generally subject to TRI reporting. Other mobile source regulations address emissions of criteria pollutants and their precursors, including carbon monoxide (CO), nitrogen dioxide (NO_2), particulate matter (PM), volatile organic compounds (VOCs), and sulfur dioxide (SO_2). These criteria air pollutant control programs for mobile sources have and will continue to result in substantial reduction of HAP releases.

Mobile sources include a wide variety of vehicles, engines, and equipment that generate air pollution and that move, or can be moved, from place to place. In the NEI, EPA divides mobile sources into two broad categories. **On-road mobile sources** include motorized vehicles that are normally operated on public roadways for transportation of passengers or freight. This includes passenger cars, motorcycles, minivans, sport-utility vehicles, light-duty trucks, heavy-duty trucks, and buses. **Nonroad mobile sources**, (sometimes also called "off-road") include aircraft, commercial marine vessels (CMVs), locomotives, and other nonroad engines and equipment. The other nonroad engines and equipment included in NEI comprise a diverse list of portable equipment, such as lawn and garden equipment; construction equipment; engines used in recreational activities; and portable industrial, commercial, and agricultural engines.

EPA's National Air Pollutant Trends Report, 1900–1998[10] indicates that about 60 percent of mobile source air toxics emissions in the U.S. are from on-road sources, and 40 percent of mobile source air toxics emissions are from nonroad sources. The emissions distribution between on- and off-road sources emitting criteria pollutants depends on the chemical. CO comprises the majority of criteria pollutants emitted, with over 100 million tons per year emitted in the U.S. Releases of CO are *primarily* the result of mobile sources – like HAPs, these emissions are split approximately 60/40 between on-road and off-road sources. (The use of CO as a monitoring surrogate for mobile source emissions is discussed in Section 4.4.1.)

Within the two broader categories of mobile sources, EPA further distinguishes on-road and nonroad sources by size, weight, use, horsepower and/or fuel type. For example, categories of on-road vehicles include light-duty gasoline vehicles (i.e., passenger cars), light-duty gasoline trucks, heavy-duty gasoline vehicles, and diesel vehicles. Examples of nonroad sources include nonroad *gasoline* engines and vehicles, (e.g., recreational off-road vehicles, construction equipment, lawn and garden equipment, and recreational marine vessels that use gasoline), nonroad *diesel* engines and vehicles (including the vehicles and equipment listed above, *except* those that use diesel fuel), aircraft, non-recreational marine vessels, and locomotives. An additional category covers all nonroad sources that use liquified petroleum gas or compressed natural gas.

4.3.4 Sources Not Included in the NEI or TRI

In addition to the four primary categories used in compiling the NEI, five other sources of air toxics which are not captured by either the NEI or TRI are described below: Indoor sources, natural sources, secondary formation of air toxics, exempt sources, and international transport.

4.3.4.1 Indoor Sources

Indoor pollution sources that release gases or particles into the air are the primary cause of indoor air quality problems in homes (Exhibit 4-4). Inadequate ventilation can increase indoor pollutant levels by not bringing in enough outdoor air to dilute emissions from indoor sources and by not carrying indoor air pollutants out of the home. High temperature and humidity levels indoors can increase the uptake of some pollutants, thereby magnifying negative health effects.

There are many sources of indoor air pollution in any home. These include combustion sources such as oil, gas, kerosene, coal, wood, and tobacco products; building materials and furnishings as diverse as deteriorated, asbestos-containing insulation, wet or damp carpet, and cabinetry or furniture made of certain pressed wood products; products for household cleaning and maintenance, personal care, or hobbies; central heating and cooling systems and humidification devices; and outdoor sources such as radon, pesticides, and outdoor air pollution.

The relative importance of any single source depends on how much of a given pollutant it emits and how hazardous those emissions are. In some cases, factors such as how old the source is and whether it is properly maintained are significant. For example, an improperly adjusted gas stove can emit significantly more carbon monoxide than one that is properly adjusted.

Some sources, such as building materials, furnishings, and household products like air fresheners, release pollutants more or less continuously. Other sources, related to activities carried out in the home, release pollutants intermittently. These include smoking, the use of unvented or malfunctioning stoves, furnaces, or space heaters, the use of solvents in cleaning and hobby activities, the use of paint strippers in redecorating activities, and the use of cleaning products and pesticides in housekeeping. High pollutant concentrations can remain in the air for long periods after some of these activities.

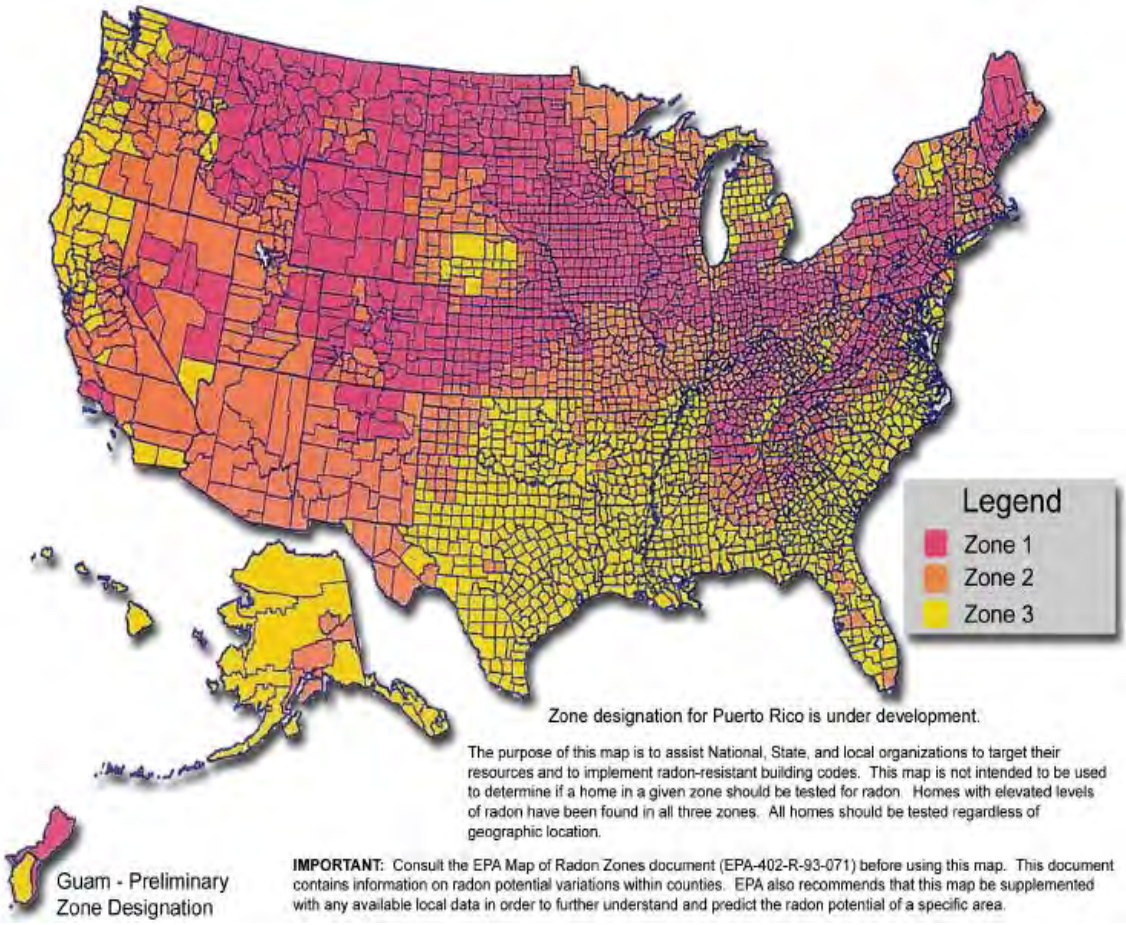
In addition to the same indoor air problems as single-family homes, apartments can have indoor air problems similar to those in offices, which are caused by sources such as contaminated ventilation systems, improperly placed outdoor air intakes, or maintenance activities.

One particularly important indoor air toxics problem actually results from an outdoor natural source. In fact, radon gas, a HAP, is one of the leading causes of lung cancer in the U.S. The most common source of indoor radon is uranium in the soil or rock on which homes are built (thus, a natural source becomes an indoor air quality problem). As uranium naturally breaks down, it releases radon as a colorless, odorless, radioactive gas. Radon gas enters homes through dirt floors, cracks in concrete walls and floors, floor drains, and sumps. When radon becomes trapped in buildings and indoor concentrations build up, exposure to radon becomes a concern.

Sometimes radon enters the home through well water. In a small number of homes, the building materials can give off radon, too. However, building materials alone rarely cause radon levels of concern (see http://www.epa.gov/radon/risk_assessment.html for more information on radon risks). Exhibit 4-5 shows EPA's map of radon zones in the U.S.

Exhibit 4-4. Major Indoor Air Pollutants and their Sources	
Major Indoor Air Pollutants	**Sources**
Radon (Rn)	Earth and rock beneath home; well water; building materials
Environmental Tobacco Smoke (includes carbon monoxide, nitrogen dioxide, and respirable particles)	Cigarette, pipe, and cigar smoking
Biologicals (e.g., pollen, mold, animal dander, and fungi)	Wet or moist walls, ceilings, carpets, and furniture; poorly maintained humidifiers, dehumidifiers, and air conditioners; bedding; household pets
Carbon Monoxide	Unvented kerosene and gas space heaters; leaking chimneys and furnaces; back-drafting from furnaces, gas water heaters, woodstoves, and fireplaces; gas stoves. Automobile exhaust from attached garages
Nitrogen Dioxide (NO_2)	Kerosene heaters, unvented gas stoves and heaters. Environmental tobacco smoke
Volatile Organic Compounds (such as xylene)	Paints, paint strippers, and other solvents; wood preservatives; aerosol sprays; cleansers and disinfectants; moth repellents and air fresheners; stored fuels and automotive products; hobby supplies; dry-cleaned clothing
Respirable Particles	Fireplaces, wood stoves, and kerosene heaters. Environmental tobacco smoke
Formaldehyde	Pressed wood products (hardwood plywood wall paneling, particle board, fiberboard) and furniture made with these pressed wood products. Urea-formaldehyde foam insulation (UFFI). Combustion sources and environmental tobacco smoke. Durable press drapes, other textiles, and glues
Pesticides	Products used to kill household pests (insecticides, termiticides, and disinfectants). Also, products used on lawns and gardens that drift or are tracked inside the house
Asbestos	Deteriorating, damaged, or disturbed insulation, fireproofing, acoustical materials, and floor tiles
Lead	Lead-based paint, contaminated soil, dust, and drinking water
Source: U.S. Environmental Protection Agency and the United States Consumer Product Safety Commission. 1995. Office of Radiation and Indoor Air (6604J) EPA/402/K/93/007, April 1995. Available at: http://www.epa.gov/iaq/pubs/insidest.html.	

Exhibit 4-5. EPA Map of Radon Zones



- **Zone 1** counties have a predicted average indoor radon screening level greater than 4 pCi/L (pico curies per liter)
- **Zone 2** counties have a predicted average indoor radon screening level between 2 and 4 pCi/L
- **Zone 3** counties have a predicted average indoor radon screening level less than 2 pCi/L

4.3.4.2 Natural Sources

Natural processes are significant sources of some air pollutants, including VOCs, NO_x, O_3, PM and other pollutants (Exhibit 4-6). Examples of natural sources of air pollutants that are *not* covered by the four main categories described above include natural processes occurring in vegetation and soils (e.g., emissions from trees), in marine ecosystems, as a result of geological activity in the form of geysers or volcanoes, as a result of meteorological activity such as lightning, and from fauna, such as ruminants and termites. Sources associated with biological activity are called biogenic sources.

Natural pollutants contribute significantly to air pollution. For example, biogenic emission estimates for the United States were 28.2 million tons of VOC and 1.53 million tons of NO_x in 1997.[10]

Exhibit 4-6. Categories of Natural Sources		
Category	**Examples of Emissions**	**Sources**
Geological	• Sulphuric, hydrofluoric and hydrochloric acids • Radon • Nitrogen oxides	• Volcanic gases • Radioactive decay of rock • Soils, lightning
Biogenic	• Ammonia • Methane • VOCs	• Animals wastes • Animal wastes, plant decay • Vegetation
Marine	• Dimethyl sulfide, ammonia, chlorides, sulfates, alkyl halides, nitrous oxides	• Sea spray released by breaking waves
Source: International Fertilizer Industry Association. 2001. Food and Agriculture Organization of the United Nations. *Global estimates of gaseous emissions of NH3, NO and N2O from agricultural land*. ISBN 92-5-104698-1. Available at: www.fao.org/DOCREP/004/Y2780E/y2780e01.htm.		

4.3.4.3 Formation of Secondary Pollutants

Some air pollutants, in addition to being directly emitted to the atmosphere by identifiable sources, are generated in the atmosphere by the chemical transformation of precursor compounds (a process called **secondary formation**). For example, under some meteorological conditions, up to 90 percent of ambient formaldehyde originates from secondary formation from a variety of precursor compounds in the presence of light (i.e., via a **photochemical reaction**). Some of the precursor compounds include isoprene (an organic compound released from trees), isobutene, and propene. The secondary formation of pollutants like formaldehyde and acetaldehyde is a complex process but can be estimated by some photochemical models (e.g., UAM-Tox, a special version of the Urban Airshed Model (UAM)). Other available models also address secondary formation but in a much more limited way (see Chapter 9 for a more detailed discussion of air models).

The NEI and other emission inventories generally do not include estimates of pollutants formed through secondary formation – only the initially emitted species are included. Because the formation of secondary pollutants depends on the meteorological conditions and the presence or absence of other compounds and/or light, a model that incorporates chemical transformation algorithms is required to estimate how much secondary product is formed from precursor compounds once they enter the atmosphere. EPA has in some instances developed estimates of secondarily formed chemicals to better inform the assessment of exposure of people to toxic air pollutants. For example, for the 1996 NATA, National-scale Air Toxics Assessment, risk characterization exercise, EPA developed a special inventory of precursor compounds to supplement the NTI, which was used in conjunction with the Assessment System for Population Exposure Nationwide (ASPEN) model to calculate ambient concentrations (see http://www.epa.gov/ttn/atw/nata/). Formation of secondary pollutants is discussed in greater detail in Chapter 8.

4.3.4.4 Other Sources Not Included in NEI or TRI

Many air toxics sources, usually relatively small ones, may not be covered or are exempt from various emissions control, reporting, and other requirements, and in some cases the number or stringency of requirements is tiered according to source size or other criteria. For example, air pollution regulations for municipal waste combustors (MWCs) promulgated pursuant to Section 129 of the CAA include separate rules for large MWCs (i.e., with capacities greater than 250 tons per day) and small MWCs (i.e., with capacities between 35 and 250 tons per day). However, there are no rules for MWCs with capacities less than 35 tons per day.

Other miscellaneous sources of air pollution (e.g., agricultural and residential burning) are controlled primarily by other S/L/T requirements. However, EPA conducts research, provides information, and pursues other non-regulatory means of addressing some of these pollution sources. For example, EPA, in conjunction with the Consumer Product Safety Commission and the American Lung Association, has published a guide for reducing pollution from residential wood combustion, including design information for less-polluting stoves and fireplaces.[11] Some local areas have ordinances that require new fireplace and wood stove installations to comply with the certification program, and others have ordinances that prohibit the use of a wood stove or fireplace on days that are conducive to the concentration of wood smoke emissions.

International Transport of Air Toxics

As noted earlier in Chapter 2 and Section 4.2.5, certain air toxics may be transported over long distances, sometimes across international borders. International sources may be an important contributor to local pollutant levels in some study areas.

Ultimately, there is no single comprehensive source of information on all sources of air toxics in a given area. The NEI and TRI are good places to start an investigation of what is being released in a study area, but as noted above, in any given place, there are probably a number of air toxics sources that are not accounted for in these inventories. Nonregulated sources, natural sources, and material moving into a study area from distant sources all have an impact on overall air quality. Assessors need to clearly understand what these limitations are as they move into the planning and scoping stage of the risk assessment (see Chapter 6). (A description of how EPA addressed background concentrations for the NATA national-scale assessment is provided at http://www.epa.gov/ttn/atw/nata/natsa2.html.)

4.4 Emissions Inventories

As mentioned previously, information on releases of air toxics is primarily compiled and maintained in **emissions inventories**. The primary emission inventory for HAPs and criteria pollutants is EPA's NEI. EPA's TRI is a second inventory that has some utility for planning and scoping an air toxics risk assessment, but is of limited use for risk assessment because of the nature of the way the data are reported. In addition to the NEI and the TRI, S/L/T air agency permit files and, in some instances, S/L/T inventories that have been developed, but not submitted to the NEI, can also provide information on the location, identity, magnitude, and source characteristics of air toxics releases.

The best inventory data are collected near the ground, literally at the source. For example, an urban scale study might opt to do a "drive by" or "windshield" verification of the number and

location of dry cleaners and gas stations in the study area rather than rely on an aggregate county-level estimate. Ultimately, the needs of the assessment (e.g., screening level or more refined) will determine the level of accuracy needed in the emissions inventory. This section will describe the NEI and the TRI. Other potential sources of air toxics data are described in Chapter 7. The process of developing an emissions inventory is also described in Chapter 7.

4.4.1 National Emissions Inventory (NEI)

EPA's Office of Air and Radiation compiles and maintains the **National Emissions Inventory (NEI)** that includes quantitative data on anthropogenic emissions of criteria pollutants and HAPs and characteristics of the sources of these air toxics.[5] It includes point, non-point, and mobile sources for all 50 states, Washington, D.C., and U.S. territories.

Previously, emissions of criteria pollutants and HAPs were tracked separately by EPA in databases that preceded the NEI. Criteria pollutant emissions data for 1985 through 1998 are available in the National Emission Trends (NET) database. Hazardous air pollutant (HAP) emissions data are available for 1993 and 1996 in the National Toxics Inventory (NTI) database. For 1999 (the most recent year for which data are available), criteria and HAP emissions data have been prepared separately but in a more integrated fashion. The final version of both the criteria and HAP inventories (for 1999) are available at http://www.epa.gov/ttn/chief/net/1999inventory.html. Note that the data collection and processing requirements for this undertaking are significant. As such, EPA plans to update the NEI every three years.

The NEI inventories are developed by EPA's Emission Factors and Inventories Group with input from S/L/T agencies, industry, and a number of EPA offices. In some cases, if a S/L/T agency does not submit data, EPA may use data from an earlier year and "grow" the emissions (e.g., for criteria stationary sources) or use only data available from other sources (e.g., HAP collected by EPA as part of the development of emission standards, or data submitted by sources under the Toxics Release Inventory program). Separate inventory documentation files have been prepared for each part of NEI (i.e., for criteria pollutants and HAPs, and for point, nonpoint, and mobile sources). These detailed documentation files are available online for criteria pollutants[12] and HAPs.[13] The reader should refer to these documentation files for detailed information on NEI. Summaries of data sources for the components of the current version of NEI are also provided below.

An important fact to keep in mind about the NEI is that it includes data on HAPs from both small and large stationary sources and both on- and off-road mobile sources. Equally important, it is much more likely to include the data necessary for modeling (although many of the data fields needed for modeling are not "mandatory," and thus states are not required to provide this information to the NEI). Information such as stack height, emission rate, and temperature are critical to developing reasonably accurate estimates of human exposure in the areas surrounding a source. It is for this reason that the NEI can be of more use than other databases, for example, for getting a better handle on realistic exposure and risk estimates in an actual study.

NEI for HAPs – Point Sources. For the NEI for HAP emissions from point sources, S/L/T agencies are asked to supply HAP emission inventory data to EPA. If they do not provide HAP emission inventory data to EPA, then EPA prepares default emission inventory data (this has been done for the 1993, 1996, and 1999 inventory years). As discussed previously, EPA uses a variety of methods to develop data and fill in gaps, where necessary (for point sources of HAPs,

EPA uses S/L/T data, EPA estimates for MACT and source categories, and TRI data; all the TRI facilities are in the NEI). This is one reason why the NEI provides, for some sources, data that may not accurately reflect actual emissions in any given place. Depending on a study's specific data quality objectives, closer inspection and verification of emissions estimates may be necessary.

The target area for the NEI includes every state and territory in the United States and every county within a State. There are no boundary limitations pertaining to traditional criteria pollutant nonattainment areas or to designated urban areas. If a facility was included in a S/L/T database, it is included in the NEI regardless of where in the state it was located. The pollutants inventoried included all 188 HAPs identified in Section 112(b) of the CAA. Some S/L/T agencies collect information on more than just these HAPs, but only the 188 are included in the HAP NEI. In addition to numerous specific chemical species and compounds, the list of 188 HAPs includes several compound groups (e.g., individual metals and their compounds, polycyclic organic matter [POM], and glycol ethers); the NEI includes emission estimates for the individual compounds within these groups wherever possible. Appendix F lists all of the specific pollutants and compound groups included in the 1999 NEI along with their Chemical Abstract Services (CAS) numbers (for individual compounds).

NEI for Criteria Pollutants – Point Sources. For the NEI for criteria emissions from point sources, EPA solicits point source data from S/L/T governments. EPA uses S/L/T point source data preferentially, except for NO_x and SO_2 emissions from utilities. For utilities, EPA uses NO_x and SO_2 emissions that facilities report to the Emissions Tracking System/Continuous Emissions Monitoring (ETS/CEM) Scorecard database. Some other criteria pollutant emissions data in the most recent version of NEI have been supplemented by EPA based on submissions to other emissions databases. In addition, emissions of ammonia (NH_3) (which is not a criteria pollutant, but is a precursor for PM) have been added to NEI based on reports submitted by S/L/T offices, TRI data, and (for locations where reports were not submitted) also based on EPA estimation methods.

Nonpoint Sources (Both HAPs and Criteria Pollutants). Much of the nonpoint source data in NEI for HAPs was initially compiled as a national-level inventory. National-level emission estimates are spatially allocated to the county-level using a number of allocation factors, such as population and employment within certain industries. For example, aggregate amounts of dry cleaner emissions for a county might be estimated from the number of people living within a county. For HAPs, EPA uses MACT data and S/L/T data, where available.

When S/L/T- or locality-specific emissions data are available, those data are substituted for data that had been allocated from national emission estimates. EPA prepares emissions for several area source categories for the NEI each year using the most current activity and emission factor data available. Emissions for other area source categories for which methodologies were not prepared in a given year are extrapolated (and assumed to increase some percentage each year) from the most recent S/L/T inventory submitted previously to EPA. For example, if an inventory was submitted in the past 3 years to EPA for the 1996 base year, the 1999 NEI emissions are extrapolated from the 1996 inventory. In some cases, criteria air emissions may also be extrapolated from other inventories (e.g., the 1985 National Air Pollutant Assessment Program inventories). A more detailed discussion of emissions estimation routines for source categories with national-level emission estimates are described in the documents referenced above.

EPA uses an emissions estimation model known as the **Biogenic Emissions Inventory System (BEIS)** to predict emissions of VOC and NO_x from forests, crop lands, and fertilized lands. Emission rates are dependent on several meteorological factors. VOC emissions are dependent on temperature and sunlight, and NO_x emissions from fertilized soils are dependent on temperature and soil moisture. The BEIS model is used to predict emissions that are included in the NEI inventory for criteria pollutants. (Keep in mind that VOCs, as a group, are inventoried, but not speciated, to help evaluate an area's potential for ozone production. Non-speciated VOC data are of limited use for performing air toxics risk assessments.)[e]

On-road Mobile Sources. In the final Version (V 3.0) of the 1999 NEI, EPA used the most recent version of the MOBILE6 (Version 6.2) model to calculate emission factors for criteria pollutants and 36 HAPs. On-road emissions inventories for CO, NO_x , VOC, PM_{10}, $PM_{2.5}$, SO_2, NH_3, and the 36 HAPs are calculated by multiplying an appropriate emission factor in grams emitted per mile by the corresponding vehicle miles traveled (VMT) in millions of miles, and then converting the product to units of tons of emissions. Emission estimates include calculations by month, county, road type, and vehicle type, with VOC broken down by exhaust and evaporative emissions and PM_{10} and $PM_{2.5}$ broken down by exhaust, brake wear, and tire wear emissions. The MOBILE6 model used is the publicly available version from EPA's Office of Transportation Air Quality's (OTAQ) Website (http://www.epa.gov/otaq/m6.htm). This model incorporates both MOBILE6.0, which is used to estimate emission factors of VOC, CO, and NO_x , and MOBILE6.1, which is used to calculate emission factors of PM_{10}, $PM_{2.5}$, SO_2 and NH_3 and MOBTOX, which is used to calculate certain HAPs. The particulate and SO_2 emission factors were previously calculated using EPA's PART5 model.

Nonroad Mobile Sources. To develop this component of the NEI, data were compiled on criteria and HAP emissions data for aircraft, commercial marine vessels, and locomotives. HAP emissions for other nonroad engines operating in the United States were estimated using the latest nonroad model. S/L/T data are used when provided. In this effort, national emission estimates were often developed for each of the above types of nonroad sources and allocated to counties based on available Geographic Information System (GIS) data. For some pollutants associated with the nonroad category, county-level (instead of national) data were used to estimate emissions. The methodologies used to estimate emissions and the procedures used to spatially allocate them to the county level vary by source category and pollutant. For some pollutants and categories, the NONROAD model was utilized to estimate emissions (see http://www.epa.gov/otaq/nonrdmdl.htm).

Concurrent with the development of the national emission estimates, S/L/T agencies developed and provided to EPA emissions inventory data for their areas based on local knowledge and activity information. These S/L/T agency data replaced the national emission estimates when the pollutant, source type, and emission type matched with the national estimates. Submitted S/L/T data that did not match the nationally-derived data were retained along with the national estimates. S/L/T data were used as provided and not adjusted to better match the national data. Some S/L/T inventories did not provide estimates for all of the pollutants included in the

---

[e]Volatile Organic Compound means any organic compound which participates in atmospheric photochemical reactions; or which is measured by a reference method, an equivalent method, an alternative method, or which is determined by procedures specified under any subpart (40 CFR Part 60).
.

nationally-derived emission estimates; in these cases, the submitted S/L/T data were used and the national estimates were included only for the missing pollutants.

Although the current NEI data files represent a valuable source of emissions data, there are numerous uncertainties associated with the current versions of these inventories that should be considered when using the data in a risk assessment. Sources of uncertainty include the following:

- The emission data included in NEI are of variable, and in some cases, undocumented derivation. Many of the emission estimates were submitted by the sources of the emissions to S/L/T air agencies, and then to EPA, without full explanations of how the emissions were estimated.

- Not all sources are accounted for. In some S/L/T data sets, very small sources have been reported, while in others only the largest sources in certain types of industry are included.

- Not all S/L/T agencies have submitted data. Specifically, for the NEI for criteria pollutants, 35 out of 50 states submitted data to EPA for the 1999 version of NEI. For the other states, EPA extrapolated the affected portions of the inventory from an earlier year. This omits sources that came online in the target year and erroneously include sources that have shut down. For more information on which S/L/T governments submitted data and for which states the inventory is extrapolated, the user can refer to the documentation for the respective inventory sector and the respective pollutant type (see website addresses above). Some of the states for which 1999 data were not available when these inventory versions were compiled have now provided EPA with their data, and EPA is working to incorporate this data into the next versions. For HAPs, 46 states have participated in the development of the 1999 NEI (with some revisions from states still under way).

- Duplicate facilities may be present, but most of the duplicates have been removed. Facility identification (ID) codes are a potential source of confusion. The NEI Unique Facility ID is the ID for the entire facility, while the state IDs are usually for individual processes; therefore an NEI Unique Facility ID can have multiple state facility IDs.

- The primary source of uncertainty associated with the inventory is the methodology used to generate the emission estimates. The emission estimation methodology is often poorly documented in the NEI Input Format - this data field is not mandatory. Data in the 1999 NEI for HAPs are made using different estimation methods. Future versions of the NEI will include a data quality rating to each emissions record, which should help characterize the quality of the emissions estimate.

Emissions data in the NEI are submitted to EPA according to the **NEI Input Format (NIF) Shell** (see http://www.epa.gov/ttn/chief/nif/index.html). This format consists of data fields grouped into tables that provide the basic structure of NEI. The NIF shell consists of eight tables for point sources, five tables for area sources, three tables for mobile sources, and two tables for biogenic sources. EPA has developed data element descriptions and data element validation rules to enforce mandatory data fields and relationships between the various tables and records of the NIF. As the NEI has evolved (and continues to be improved and developed), the NIF shell has evolved as well. Version 3.0 of the NIF shell was released in May 2003 and updated in November 2003.

In June 2002, EPA promulgated the final Consolidated Emissions Reporting Rule,[14] which simplifies and consolidates emission inventory reporting requirements (for criteria air pollutants only) to a single location within the *Code of Federal Regulations* (CFR), establishes new reporting requirements related to $PM_{2.5}$ and regional haze, and establishes new requirements for the statewide reporting of area source and mobile source emissions. Many state and local agencies asked EPA to take this action to consolidate reporting requirements; improve reporting efficiency; provide flexibility for data gathering and reporting; and better explain to program managers and the public the need for a consistent inventory program. Consolidated reporting should increase the efficiency of the emission inventory program and provide more consistent and uniform data.

In conjunction with the NIF shell, EPA has developed an automated software program to help NIF users perform **quality assurance/quality control (QA/QC) checks** on their files to ensure correct format specification. This software, available for download from the NIF shell web page, separates QA/QC checks into format and content. Format checks ensure that the submitted information includes the minimum data elements required for Emission Factor and Inventory Group (EFIG) to accept the submitted data. Content checks are provided for the user as a way to highlight possible errors in the submitted data. The latest version of the software allows the user to choose whether to perform QA/QC checks on the data for format, the minimum standards required to put the data in the database, or the more resource intensive content or reasonableness checks. When checking for content, the format is also checked as the format must be correct in order for content checks to be performed at all.

4.4.2 Toxics Release Inventory (TRI)

The **Toxics Release Inventory (TRI)** is a publicly available EPA database that contains information about releases and other waste management activities reported annually by certain covered industry groups as well as federal facilities for over 650 toxic chemicals (see http://www.epa.gov/tri/). This inventory was established under the Emergency Planning and Community Right-to-Know Act of 1986 (EPCRA) and expanded by the Pollution Prevention Act of 1990.

TRI reporting is required only for facilities that meet all of the following three criteria:

- They have ten or more full-time employees or the equivalent (i.e., a total of 20,000 hours or greater; see 40 CFR 372.3);
- They are included in specified industrial sectors (see Exhibit 4-7); and
- They exceed any one reporting threshold for manufacturing, processing, or otherwise using a TRI chemical (see Exhibit 4-8).

If a facility meets these criteria, then it must report releases to environmental media as well as waste management data. In 2001 (the latest year for which data are publicly available), air emissions of toxic chemicals totaled 1.7 billion pounds (over a quarter of all releases of TRI chemicals to the environment).

Exhibit 4-7. Standard Industrial Classification (SIC) Codes in TRI Reporting			
Original Industries			
SIC Code	**Industry Group**	**SIC Code**	**Industry Group**
20	Food	30	Rubber and Plastics
21	Tobacco	31	Leather
22	Textiles	32	Stone, Clay, and Glass
23	Apparel	33	Primary Metal
24	Lumber and Wood	34	Fabricated Metals
25	Furniture	35	Machinery (excluding electrical)
26	Paper	36	Electrical and Electronic Equipment
27	Printing and Publishing	37	Transportation Equipment
28	Chemicals	38	Instruments
29	Petroleum and Coal	39	Miscellaneous Manufacturing
New Industries Reporting to TRI as of the 1998 Reporting Year			
SIC Code	**Industry Group**		
10	Metal mining (except for SIC codes 1011,1081, and 1094)		
12	Coal mining (except for 1241 and extraction activities)		
4911, 4931, and 4939	Limited to electrical utilities that combust coal and/or oil for distribution in commerce (SIC codes 4911, 4931, and 4939)		
4953	Limited to hazardous waste treatment and disposal facilities regulated under the Resource Conservation and Recovery Act (RCRA) Subtitle C		
5169	Chemicals and allied products wholesale distributors		
5171	Petroleum bulk plants and terminals		
7389	Solvent recovery services primarily engaged on a contract or fee basis		
Source: U.S. Environmental Protection Agency. 2004. Toxic Release Inventory (TRI) Program. *Standard Industrial Classification (SIC) Codes in TRI Reporting.* Updated March 2, 2004. Available at: http://www.epa.gov/tri/report/siccode.htm. (Last accessed April 2004.)			

Exhibit 4-8. Thresholds for Reporting to the TRI

EPCRA Section 313 non-PBT chemicals (Section 372.25). A facility meeting the SIC code (or Federal facility) and employee criteria must file a TRI report for a non-PBT Section 313 chemical if the facility:

- Manufactured (including imported) more than 25,000 pounds per year; or
- Processed more than 25,000 pounds per year; or
- Otherwise used more than 10,000 pounds per year.

EPCRA Section 313 PBT chemicals (40 CFR372.28). If a facility manufactures, processes, or otherwise uses any chemicals that are listed as persistent, bioaccumulative, and toxic (PBT), the threshold quantity is one of the following (per Section 313 chemical or category per year):

Type of Chemical	Reporting Threshold by Activity		
	Manufacture	**Process**	**Otherwise Used**
Highly persistent and bioaccumulative compounds	10 pounds	10 pounds	10 pounds
Dioxin and dioxin-like compounds	0.1 grams	0.1 grams	0.1 grams
Other persistent and bioaccumulative compounds (lead and lead compounds)	100 pounds	100 pounds	100 pounds

Activity thresholds are calculated independently of each other based on cumulative quantities per Section 313 chemical over the reporting year.

Current list of Section 313 PBT Chemicals

- aldrin
- benzo(g,h,i)perylene
- chlordane
- dioxin and dioxin-like compounds
- heptachlor
- hexachlorobenzene
- isodrin
- lead
- lead compounds
- mercury
- mercury compounds
- methoxychlor
- octachlorostyrene
- pendimethalin
- pentachlorobenzene
- polychlorinated biphenyl
- polycyclic aromatic compounds
- tetrabromobisphenol A
- toxaphene
- trifluralin

Note in Exhibit 4-7 that additional industries have been added to the TRI over time. Thus, some industries were not required to report in the past and, as such, no records will exist for these facilities in the historical TRI files. The list of covered chemicals has also grown over time. Thus, the ability to track trends for more recently added industries and chemicals is more limited than for industries and chemicals that have been covered throughout the history of the TRI.

Industrial sectors subject to TRI reporting are identified by Standard Industrial Classification (SIC) codes. SIC codes are numerical codes developed by the U.S. government as a means of consistently classifying the primary business of business establishments. A full list of the industry groups that are required to report can be found at http://www.epa.gov/tri/report/siccode.htm.

Although most of the existing emissions data in the TRI system are organized according to SIC codes, EPA has proposed regulations that would result in the use of the North American Industry Classification System (NAICS) rather than SIC codes (see http://www.census.gov/epcd/www/naics.html). Rather than classifying industries on the basis of several different economic concepts (as the SIC structure does), NAICS classifies establishments according to similarities in the processes used to produce goods and services. The TRI program issued a proposed rule to implement the NAICS system classification on March 21, 2003. (See 66 *Fed. Reg.* 13872.) It is expected that the use of NAICS in the TRI system will allow EPA to more accurately characterize the current state of the national economy (including new and emerging industries not adequately covered by SIC codes). The existing SIC structure will not be updated in the future because the Office of Management and Budget has adopted NAICS as the United States' new industry classification system. In addition, using NAICS for TRI reporting purposes will enable more efficient database integration and will promote public access to commonly defined data from disparate sources. This change will not affect the universe of facilities that is currently required to report to TRI.

EPCRA requires only that facilities report their releases of the listed chemicals. There are no additional control or mitigation actions required. The information collected through the TRI program is made public, however, and pressure from local citizen groups has been an incentive to many industries to reduce the quantity of pollutants they release.

While the TRI data have utility for the scoping out of an air toxics risk assessment project, they have several limitations that assessors must understand. Importantly for risk assessors, the TRI program requires only that one single annual value representing total releases to the air (segregated only by stack releases and fugitive releases) be reported by the individual affected facilities. So while annual average emissions may be useful in screening-level assessments for chronic exposures, it may be difficult to assess acute noncancer hazard associated with short-term, peak emission levels. Source-specific information within the facility is not routinely reported through the TRI. Likewise, no information is reported on release parameters critical to air dispersion modeling (e.g., location of release on the facility property, release rates, stack height, stack diameter, release temperature). (See Chapter 9, for more information on modeling parameters used in air quality and exposure modeling.)

As discussed in Section 4.2, the list of TRI pollutants is organized differently than the list of HAPs in CAA Section 112, causing some complications in interpreting emission data. It is difficult to correctly relate some of the SIC codes (under which TRI emissions are grouped) to specific air emission processes. Because quantities are only reported if a statutory threshold is

met, a facility may report emissions for one year but not the next, even though the facility is still in operation. Similarly, individual pollutants may not be reported consistently from year to year due to the thresholds that apply to individual pollutants (e.g., a facility may report releases of 10 pollutants one year and releases of only five pollutants the next year because the others dropped below the reporting threshold).

Furthermore, for some facilities, it is possible that, for a variety of releases, the data included for a facility's emissions in the TRI do not match the same data reported to the NEI, indicating a potential problem with either or both data sets. The risk assessor should apply care and discretion when using TRI information to estimate exposures and risk from individual facilities. Ultimately, the TRI provides information about the location, identity, and amount of air toxics emissions in a community. However, due to the nature of the way the data are developed and reported, TRI data should generally be considered a source of limited information about a facility and should not be used in risk assessments involving modeling (as noted above, S/L/T and NEI data are more likely to be useful for modeling). For robust analysis, it should generally be considered a starting point, not an end.

References

1. United States Environmental Protection Agency. 1998. *Handbook for Air Toxics Emission Inventory Development, Volume 1: Stationary Sources* (Appendix C). Office of Air Quality Planning and Standards, Research Triangle Park, NC, November 1998. EPA/454/B98/002. Available at: http://www.epa.gov/ttn/chief/eidocs/airtoxic.pdf.

2. U.S. Environmental Protection Agency. 2003. Technology Transfer Network Air Toxics Website. *The Original List of Hazardous Air Pollutants.* Updated January 9, 2004. Available at: http://www.epa.gov/ttn/atw/188polls.html. (Last accessed March 2004.)

3. U.S. Environmental Protection Agency. 2001. *The Emergency Planning and Community Right-to-Know Act: Section 313 Release and Other Waste Management Reporting Requirements*. Washington, D.C. EPA/260/K01/001. Available at: http://www.epa.gov/tri/guide_docs/2001/brochure2000.pdf.

4. U.S. Environmental Protection Agency. 2001. *Emergency Planning and Community Right-to-Know Section 313; List of Toxic Chemicals*. Office of Environmental Information. Washington, DC, March 2001. EPA/ 260/B01/001. Available at: http://www.epa.gov/tri/chemical/chemlist2001.pdf.

5. U.S. Environmental Protection Agency. 2004. Technology Transfer Network Clearinghouse for Inventories & Emission Factors. *National Emissions Inventories for the U.S.* Updated February 4, 2004. Available at: http://www.epa.gov/ttn/chief/net/. (Last accessed March 2004.)

6. U.S. Environmental Protection Agency. 1992. *Notice Initial Source Category List*. *Federal Register* 57:31576, July 16, 1992.

7. U.S. Environmental Protection Agency. 2002. *National Emission Standards for Hazardous Air Pollutants: Revision of Source Category List Under Section 112 of the Clean Air Act. Federal Register* 67:6521, February 12, 2002.

8. U.S. Environmental Protection Agency. 1997. *Mercury Study Report to Congress*. Office of Air Quality Planning and Standards. Washington D.C. EPA/452/R97/0003. Available at: http://www.epa.gov/ttn/atw/112nmerc/mercury.html.

9. U.S. Environmental Protection Agency. 2001. *Control of Emissions of Hazardous Air Pollutants From Mobile Sources. Final Rule*. *Federal Register* 66:17230, March 29, 2001. Available at: http://www.epa.gov/fedrgstr/EPA-AIR/2001/March/Day-29/a37.htm.

10. U.S. Environmental Protection Agency. 2000. *National Air Pollutant Emission Trends: 1900-1998*. Office of Air Quality Planning and Standards, Research Triangle Park, NC. EPA/454/R00/002. Available at: (http://www.epa.gov/ttn/chief/trends/trends98/index.html).

11. U.S. Environmental Protection Agency, Consumer Product Safety Commission (CPSC), and American Lung Association. 1998. *What You Should Know About Combustion Appliances and Indoor Air Pollution*. CPSC Document #452. Available at: http://www.cpsc.gov/cpscpub/pubs/452.html. (Last accessed March 2004.)

12. U.S. Environmental Protection Agency. 2004. Technology Transfer Network Air Toxics Website. *Area Source Standards*. Updated January 15, 2004. Available at: ftp://ftp.epa.gov/EmisInventory/finalnei99ver2/criteria/documentation/. (Last accessed March 2004.)

13. U.S. Environmental Protection Agency. 2003. *Documentation for the Final 1999 Nonpoint Area Source National Emission Inventory for Hazardous Air Pollutants (Version 3).* Emissions, Monitoring and Analysis Division, Research Triangle Park, NC, August 2003. Available at: ftp://ftp.epa.gov/EmisInventory/finalnei99ver3/haps/documentation/.

14. U.S. Environmental Protection Agency. 2002. *Consolidated Emissions Reporting. Final Rule*. *Federal Register* 67:39602, June 10, 2002. Available at: http://www.epa.gov/ttn/chief/cerr/index.html.

PART II

HUMAN HEALTH RISK ASSESSMENT: INHALATION

Chapter 5 Getting Started: Planning and Scoping the Inhalation Risk Assessment

Table of Contents

5.1 Introduction 1

5.2 Framework and Process for Air Toxics Risk Assessments 1
- 5.2.1 Framework for Cumulative Risk Assessment 1
- 5.2.2 General Framework for Residual Risk Assessment 3
- 5.2.3 The Air Toxics Risk Assessment Process 3
- 5.2.4 Overview of Inhalation Exposure Assessment 6
  - 5.2.4.1 Exposure and Exposure Assessment: What's the Difference? 6
  - 5.2.4.2 Components of an Exposure Assessment 7

5.3 Planning and Scoping 8
- 5.3.1 Why is Planning and Scoping Important? 9
- 5.3.2 The Planning and Scoping Process 9
  - 5.3.2.1 What is the Concern? 9
  - 5.3.2.2 Who Needs to be Involved? 10
  - 5.3.2.3 What is the Scope? 12
  - 5.3.2.4 Why is There a Problem? 13
  - 5.3.2.5 How will Risk Managers Evaluate the Concern? 13
  - 5.3.2.6 Lessons Learned on Planning and Scoping 13

References 15

5.1 Introduction

The background discussion in Part I of this manual introduced the general air toxics risk assessment process (see Exhibit 3-4). Part II describes the tools and approaches risk assessors use to evaluate human health risks associated with inhalation exposures to air toxics. Section 5.2 below describes the framework used for air toxics risk assessment, including its three phases: (1) planning, scoping, and problem formulation; (2) analysis (which includes exposure assessment and toxicity assessment); and (3) risk characterization. Part II includes nine chapters that describe these three phases in detail.

- The remainder of the current chapter describes planning and scoping (Section 5.3).

- Chapter 6 describes problem formulation.

- Because exposure assessment is generally the most labor and financially-intensive step in the analysis phase, and because it involves a variety of related (but heterogeneous activities), the discussion of exposure assessment includes five chapters:
  - Chapter 7 describes how to characterize sources and quantify emissions;
  - Chapter 8 explores the fate and transport of air toxics in the atmosphere;
  - Chapter 9 discusses air quality modeling;
  - Chapter 10 discusses monitoring; and
  - Chapter 11 discusses quantifying exposure, including exposure modeling.

- Chapter 12 describes the remainder of the analysis phase, toxicity assessment.

- Chapter 13 describes the risk characterization phase for inhalation assessments.

5.2 Framework and Process for Air Toxics Risk Assessments

The original risk assessment framework developed in 1983 by the NRC (see Chapter 3) has been refined based on the risk assessment experience gained by EPA and other agencies. Two descriptions of this refined framework are particularly useful for air toxics risk assessments: EPA's framework for cumulative risk assessment, and EPA's general framework for assessing residual risks.

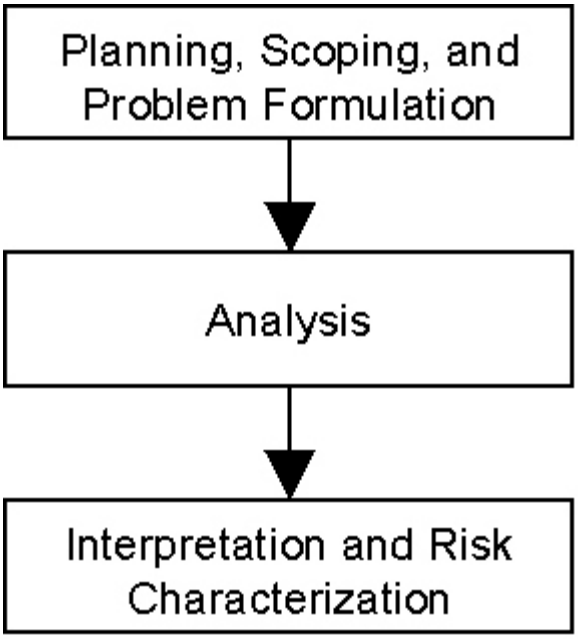
Cumulative Risk Assessment

An analysis, characterization, and possible quantification of the combined risks to health or the environment from simultaneous exposure to multiple agents or stressors.

5.2.1 Framework for Cumulative Risk Assessment

EPA's *Framework for Cumulative Risk Assessment*[1] describes three main phases to a risk assessment: (1) planning, scoping, and problem formulation; (2) analysis; and (3) risk characterization (Exhibit 5-1).

Exhibit 5-1. Three-Phase Framework for Cumulative Risk Assessment



Source: EPA *Framework for Cumulative Risk Assessment*[1]

- In the **planning, scoping, and problem formulation phase**, a team of risk managers, risk assessors, and other stakeholders identify the problem to be assessed and establish the goals, breadth, depth, and focus of the assessment. The end products of this phase are a conceptual model and an analysis plan. The conceptual model establishes the air toxics, exposure pathways, and health and ecological effects to be evaluated. The analysis plan lays out how the elements of the conceptual model are going to be studied.

- The **analysis phase** (the elements of which are described by the analysis plan) is primarily an analytic process in which risk experts apply risk assessment approaches to evaluate the problem at hand. Specifically, the analysis plan specifies how data, modeling, or assumptions will be obtained, performed, or defined for all aspects of the exposure evaluation. Additionally, the analysis plan specifies the strategy for obtaining and considering hazard and dose-response information for these stressors and the method for combining the exposure information with the hazard and dose-response information to generate risk estimates. As the risk analysis is refined, it may be appropriate to revisit and refine the exposure, hazard, and dose-response information in an iterative fashion.

- The **risk characterization** phase integrates and interprets the results of the analysis phase and addresses the problem(s) formulated in the planning, scoping, and problem formulation phase. It describes the qualitative and/or quantitative risk assessment results and lists the important assumptions, limitations, and uncertainties associated with those results; and discusses the ultimate use of the analytic-deliberative outcomes.

5.2.2 General Framework for Residual Risk Assessment

EPA's *Residual Risk Report to Congress*[2] outlines a general framework for assessing residual risks to implement the requirements of CAA sections 112(f)(2) through (6). Those sections require EPA to promulgate standards beyond MACT when necessary to provide "an ample margin of safety to protect public health" and to "prevent, considering costs, energy, safety, and other relevant factors, an adverse environmental effect." EPA developed the general framework using knowledge gained from past risk assessments and guidance gained from reports such as the NRC and CRARM reports (see Chapter 3). The framework calls for an iterative, tiered assessments of the risks to humans and ecological receptors through inhalation and, where appropriate, non-inhalation exposures to HAPs.

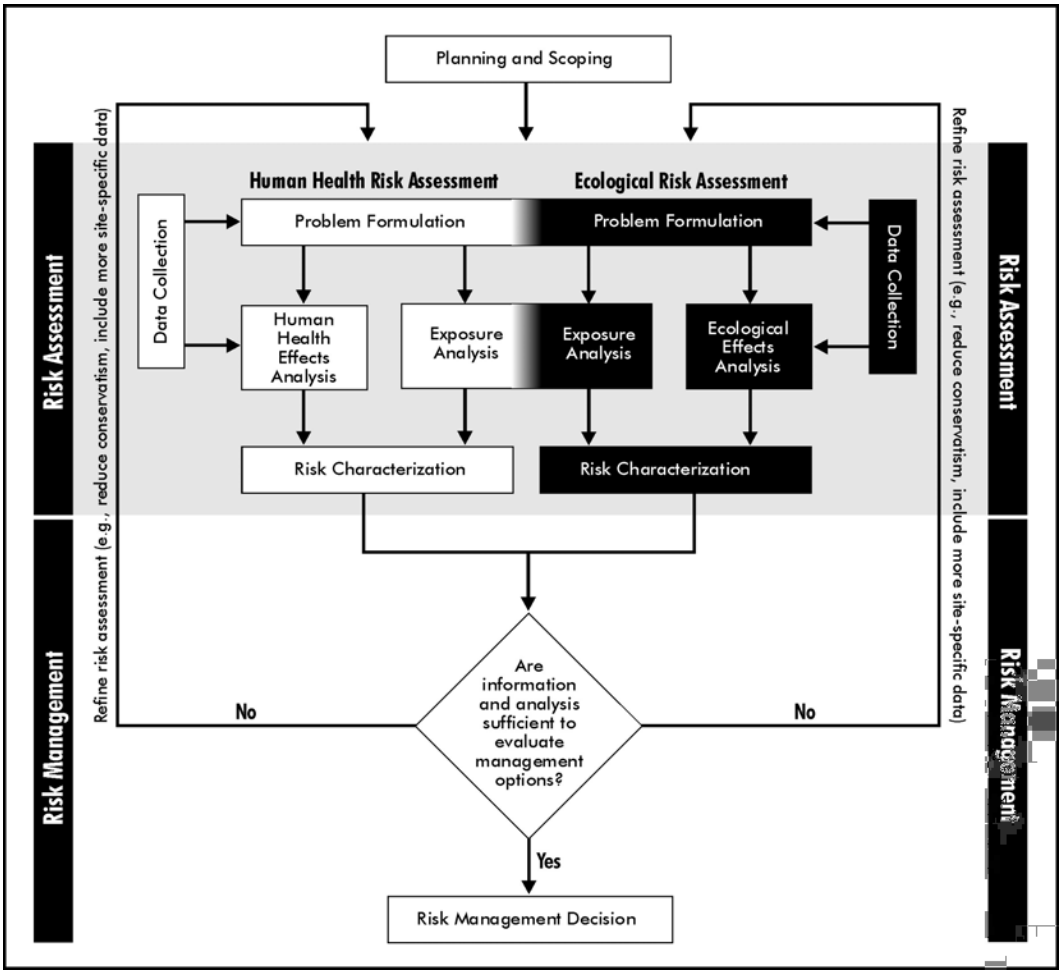
As shown in Exhibit 5-2, each human health and ecological risk assessment is organized into three phases: (1) the problem formulation phase, in which the context and scope of the assessments are specified (this phase also includes planning and scoping activities); (2) the analysis phase, in which the toxicity of HAPs and exposures to humans or ecological receptors are evaluated; and (3) the risk characterization phase, in which the toxicity and exposure analyses are integrated to determine the level of risk that may exist. The problem formulation and analysis phases of the human health and ecological risk assessments will partially "overlap" in that some pathway of concern for humans (e.g., consumption of contaminated fish) may also be pathways of concern for ecological receptors (e.g., fish-eating wildlife). Consequently, exposure analyses for some air toxics may be designed to provide information for both ecological and human health assessments.

In both human health and ecological risk assessments, there is essentially a continuum of possible levels of analysis from the most basic screening approach to a highly refined, detailed assessment. The screening level or tier of analysis is designed, through the use of simplifying assumptions and conservative inputs, to identify for no further action or analysis, exposure pathways and air toxics for which risks are unlikely to be of concern. Screening tier analyses are designed to be relatively simple, inexpensive, and quick, using existing data, defined decision criteria, and models with simplifying conservative assumptions as inputs. More refined levels of analysis include the refinement of aspects of the analysis that are thought to influence risk most or may contain the greatest uncertainty. They may also allow a more quantitative analysis of uncertainty and variability. Refined analysis requires more effort, but produces results that are hopefully less uncertain and less conservative (i.e., less likely to overestimate risk).

5.2.3 The Air Toxics Risk Assessment Process

Building on the Cumulative and Residual Risk frameworks discussed above, the human health portions of this reference manual describe the risk assessment process for air toxics in three general phases (Exhibit 5-3; the process for ecological risk assessment is provided in Part IV). [Note that Exhibit 5-3 is consistent with both the Cumulative and Residual Risk frameworks discussed above. The benefit of Exhibit 5-3 is that it helps to better visualize the *detailed elements* that are usually performed in an air toxics risk assessment.]

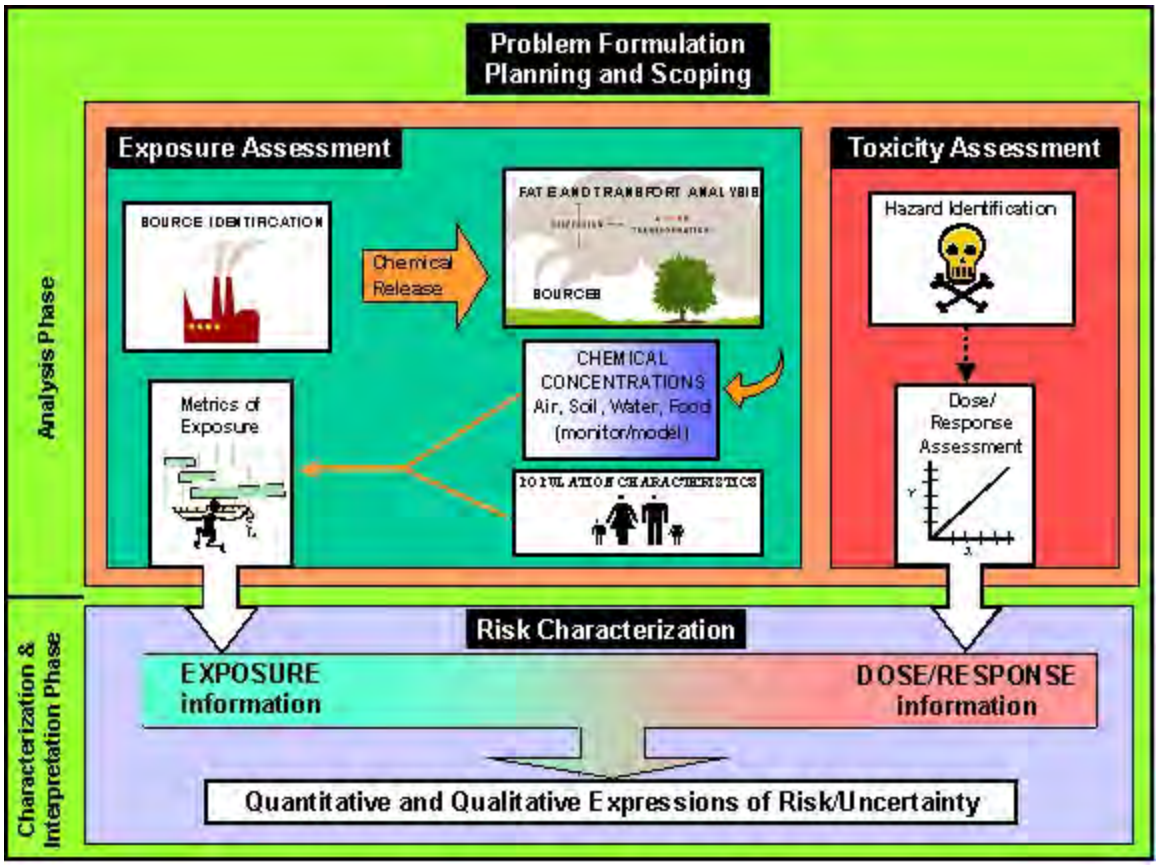
Exhibit 5-2. General Framework for Residual Risk Assessment



Source: Modified from *EPA's Residual Risk Report to Congress*[2]

- The **planning, scoping, and problem formulation** phase is divided into two general steps: planning and scoping, and problem formulation. These two steps consist of the activities described above in the cumulative risk assessment framework. The end products of this phase are a conceptual model and an analysis plan. As shown in the Exhibit 5-3, planning, scoping, and problem formulation encompass the entire risk assessment process because stakeholders aim to understand and state the problem they want to study using the risk assessment process and plan how they are going to study the problem *before* the risk assessment is performed. They also must recognize that they may need to refine the problem statement and study methodology as new information is gained during the assessment.

Exhibit 5-3. The Detailed Air Toxics Risk Assessment Process



- The **analysis** phase is divided into two general steps: **exposure assessment** and **toxicity assessment** (the general process for ecological risk assessments is described in Part IV). Exposure assessment is a relatively complex process involving source identification; development of an emissions inventory; fate and transport analysis (through modeling and/or monitoring) to estimate chemical concentrations in air (and soil, food, and water for multimedia assessments); and combining information on chemical concentrations with population characteristics to obtain one or more metric(s) of exposure. Toxicity assessment includes hazard identification and dose-response assessment.

- The **risk characterization** phase integrates the information from the exposure assessment and the toxicity assessment to provide both quantitative and qualitative expressions of risk. The risk characterization also includes a thorough discussion of uncertainty associated with each of the major elements of the risk assessment.

The remainder of Parts I, II, and III of this Volume will rely on the general approach outlined in Exhibit 5-3 as a roadmap for describing the air toxics risk assessment process.

Risk Assessment: Is it a Linear Process?

It may be useful to think of the risk assessment process as a set of steps that proceed in a linear fashion. But it does not always work out that way. For example, through good planning, scoping, and problem formulation (e.g., a thorough identification of sources and chemicals while developing the conceptual model), much of the preliminary exposure assessment work may be accomplished. A prior basic knowledge and discussion of toxic and chemical/physical properties of the chemicals of potential concern (COPCs) (information often developed during the toxicity and exposure assessments, respectively) may help the risk assessment team rule out certain pathways for consideration during the planning, scoping, and problem formulation phase. Of course, a good analysis plan will include mechanisms to confirm and document all these decisions, but the fact still remains that the risk assessment process is actually a combination of a variety of steps, many of which may occur simultaneously.

5.2.4 Overview of Inhalation Exposure Assessment

Because exposure assessment is generally the most multifaceted and time-consuming part of an air toxics risk assessment, it cannot be discussed in a single chapter. This subsection provides an overview of exposure assessment and identifies where each step of the process is described in more detail in subsequent chapters (i.e., Chapters 6 through 11). EPA's *Guidelines for Exposure Assessment*[3] is the key reference document for the exposure assessment portion of the risk assessment, and air toxics risk assessors may want to obtain and become familiar with its contents.

Exposure assessment helps identify and evaluate a population receiving exposure to a toxic agent, and describe its composition and size, as well as the type, magnitude, frequency, route and duration of exposure. In other words, an exposure assessment is that part of the risk assessment that identifies:

- Who is potentially exposed to toxic chemicals;
- What toxics they may be exposed to; and
- How they may be exposed to those chemicals (amount, pattern, and route).

5.2.4.1 Exposure and Exposure Assessment: What's the Difference?

Exposure assessment is the overall process of evaluating who receives exposure to toxic chemicals, what those chemicals are, and how the exposure occurs. Exposure, on the other hand, (according to EPA definition[1]) represents contact with a chemical at the visible external boundary of a person, including skin and openings into the body such as mouth, punctures in the skin, and nostrils. This definition of exposure does not describe the contact of a chemical with the actual exchange boundaries in the body where absorption into the bloodstream can take place, such as the linings of the lung or digestive tract. (One exception to this is chemical contact with skin or punctures in the skin; in this case, the location of the exposure and the exchange boundary are one in the same.) Other than dermal exposure, chemicals must be physically taken into the body by ingestion or inhalation (a process called **intake**) before they can contact an exchange boundary and be taken into the bloodstream (a process called **uptake**).

The term **route of exposure** is used to describe the different ways a chemical enters the body. The three main routes of exposure are **inhalation**, **ingestion**, and absorbing a chemical through the skin (**dermal**). For inhalation risk assessments, we are only concerned with the inhalation route of exposure. The dermal and ingestion routes of exposure are generally only relevant to chemicals that persist and which also may bioaccumulate (e.g., the persistent, bioaccumulative HAP (PB-HAP) compounds). Discussion of these routes of exposure is reserved for Part III.

Some chemicals can cause harm in the part of the body where individuals take them in (e.g., in the respiratory system for inhaled chemicals or in the digestive tract for ingested chemicals). This is called a **portal of entry effect** because the adverse effect occurs at the place (i.e., the "portal") where the chemical enters the body. Other chemicals have to be taken into and distributed by the circulatory system to cause a harmful effect at a point distant from their portal of entry into the body. Such effects are called **systemic effects** because they have the potential to act at points throughout the system. As a chemical moves through the body, it may be metabolized (possibly to a more toxic entity); stored in the body; and/or eliminated in urine, feces, sweat, nails/hair, or exhaled breath.

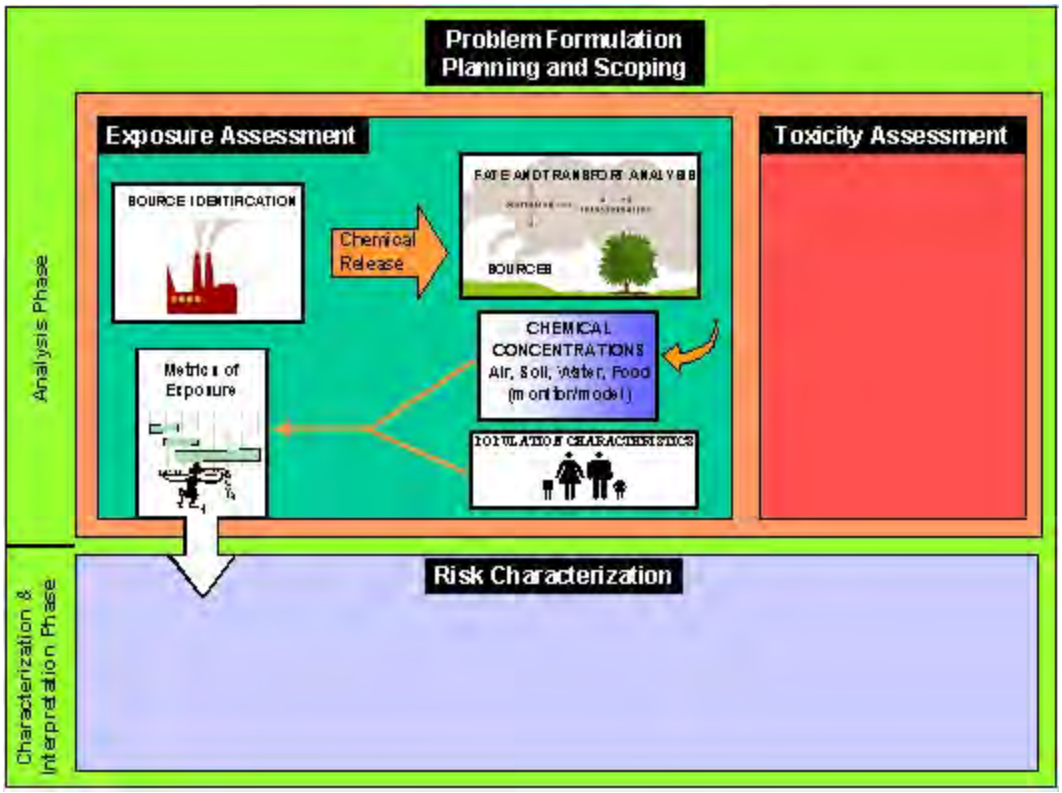
5.2.4.2 Components of an Exposure Assessment

The nature and complexity of the components within the exposure assessment are often functions of the particular risk management question (or other purpose) to be addressed. Simple screening analyses that rely on conservative default assumptions may be sufficient to rule out the need for further analyses or action. On the other hand, a more detailed exposure analysis may be needed to determine the necessity for emission controls, particularly when the application of those controls is associated with large economic consequences. Indeed, the exposure assessment raises and addresses many of the risk assessment's difficult and critical policy questions. As illustrated in Exhibit 5-4, the exposure assessment includes the following steps:

- **Characterization of the exposure setting**, including the physical environment, scale of the study area, important sources and chemicals, and potentially exposed populations and population characteristics (e.g., demographics). Most of this information is collected and organized during the problem formulation portion of the risk assessment (see Chapter 6).

- **Identification of exposure pathways**, including sources and mechanism of release, exposure points and routes of exposure, and transport media. Again, most of this information is collected and organized during problem formulation (see Chapter 6).

Exhibit 5-4. Exposure Assessment is the Most Time-Consuming Part of Risk Assessment



- **Quantification of exposure**, including an **evaluation of uncertainty** and **preparation of documentation**. Quantification of exposure includes three general steps which are discussed in several subsequent chapters.
  - Characterization of emissions is discussed in Chapter 7.
  - Evaluation of chemical fate and transport is discussed in three chapters. Chapter 8 discusses dispersal, transport, and fate of air toxics in the atmosphere. Chapter 9 discusses air quality modeling. Chapter 10 discusses air toxics monitoring.
  - Estimation of exposure concentrations (EC) is discussed in Chapter 11, along with exposure modeling, evaluation of uncertainty, and preparation of documentation.

5.3 Planning and Scoping

Planning and scoping is the first step in an air toxics risk assessment (good planning and scoping is important for any scientific study). It is both a *deliberate* and *deliberative* process that identifies the problems to be assessed; identifies stakeholders in the risk assessment process; establishes the bounds (i.e., the scope) of the analysis, including elements to be included or excluded from the analysis; develops a description of the potential interrelationship between air pollutants and receptors; and articulates the overall analysis plan for the assessment. This section provides an overview of how to plan for and scope an air toxics risk assessment. The discussion focuses on four key elements of planning and scoping:

- Why is planning and scoping important?
- What is the process?
- Who should be involved?
- What are the key products?

More detailed discussions of the planning and scoping process can be found in the EPA guidance documents *Guidance on Cumulative Risk Assessment*[4], *Framework for Cumulative Risk Assessment* [5], and *Risk Assessment Guidance for Superfund (RAGS): Volume I* [6] (Chapter 2 of this RAGS document discusses the role of the risk assessor in planning and scoping).

5.3.1 Why is Planning and Scoping Important?

Planning and scoping may be the most important step in the risk assessment process. Without adequate planning, most risk assessments will not succeed in providing the type of information that risk management needs to make a well-founded decision. Thorough planning and scoping is commonly conducted ***before*** any substantive work is done on the risk assessment. Planning and scoping is important for developing a common understanding of why the risk assessment is being conducted, the scope of the assessment, the quantity and quality of data needed to answer the assessment questions, and how risk managers will use the results. This step is also a focal point for stakeholder involvement in the risk assessment process. The specific goals of planning and scoping include:

- The approaches, including a review of the risk dimensions and technical elements that may be evaluated in the assessment;
- The relationships among potential assessment end points and risk management options;
- An analysis plan and a conceptual model (articulated in the problem formulation phase - see Chapter 6);
- The resources (for example, data or models) required or available;
- The identity of those involved and their roles (for example, technical, legal, or stakeholder advisors); and
- The schedule to be followed (including provision for timely and adequate internal, and independent, external peer review).

5.3.2 The Planning and Scoping Process

The five essential steps in the planning and scoping process include (1) identifying the concern; (2) identifying who needs to be involved; (3) determining the scope of the risk assessment; (4) describing why there may be a problem (i.e., describing the presumed interrelationship among sources of risk, humans receiving the exposure, and potential health effects); and (5) determining how risk managers will evaluate the concern. Each is described in a separate subsection below.

5.3.2.1 What is the Concern?

Most risk assessments are conducted because of a regulatory requirement, a community need or concern, or some other reason. The specific concerns and the resources available to address those concerns will largely shape the risk assessment scope and methods. For example, a simple, screening-level risk assessment may be adequate to support a typical pollution permitting process

while a detailed analysis may be necessary to respond to a particular community concern (e.g., are children in a nearby school exposed to harmful levels of air toxics from all sources in the community?).

At the end of this first step, risk assessors usually identify the full breadth of the concerns of the participating stakeholders and clearly articulate which of those concerns will be the focus of the risk assessment and why. For example, in a community-level multisource analysis, some community stakeholders may be concerned about nuisance odor while others are concerned about potential cancer health risks from airborne pollutants. At the end of this step, all stakeholders should be clear that the risk assessment cannot address the odor issue but, rather, will focus on the cancer concern. This is also the time to identify other resources or means for attempting to address the non-risk related odor issue.

Stakeholders often identify a wide range of concerns in the risk assessment process that risk assessment methods may be unable to address. It is always important to acknowledge the legitimacy of stakeholder concerns and to work to clarify the limitations of the risk assessment process – especially when assessors are working to respond to community concerns. At the same time, risk assessors often assist in identifying the proper path for responding to non-risk related issues. Proceeding in this manner will help create an attitude of trust, foster buy-in of the risk assessment process and results, and avoid creating false expectations.

5.3.2.2 Who Needs to be Involved?

The key participants in the planning and scoping process include, at minimum, the risk managers who will use the results of the risk assessment and the risk assessment technical team who will perform the analysis.

- **Risk managers** are the persons or groups with the authority to make the decisions about the acceptability of risk and how an unacceptable risk may be mitigated, avoided, or reduced. For regulatory requirements (e.g., permitting, compliance), the risk manager usually is a government agency such as EPA or a S/L/T authority. For voluntary efforts, the risk manager(s) generally will include members of the potentially affected or interested parties (e.g., industry representatives, community leaders, local government).

- The **risk assessment technical team** includes those experts who will perform the activities involved in the risk assessment, including environmental scientists, modelers, chemists, toxicologists, ecologists, and engineers.

These individuals need to understand the goals of the risk assessment, how the results will be used, the amount and quality of information necessary to make key decisions, and the uncertainties associated with the inputs, risk assessment methods, and resulting risk estimates.

The specific concerns from step one may generate the need for a diverse set of individuals or groups with an interest in having the assessment done ("interested or affected parties").[7] Each group may have a unique set of questions, concerns, and fears. It is important to design the risk assessment to address as many of these issues as possible within available time and resources. Planning and scoping begins with a dialogue among these individuals and groups; consequently, the initial planning and scoping team may need to expand over time to include additional

participants, including public officials, citizens, and industry representatives. In many cases, technical experts who live in the affected communities can be effective participants because they have both the trust of the local community and the technical skills to explain complex issues. A strong community involvement effort early in the process can help identify these concerns (see Part V of this Volume).

Examples of Possible Interested or Affected Parties

State governments	Affected industry
Tribal governments	Civic organizations
Local governments	Business owners
Community groups	Trade associations
Grassroots organizations	Labor unions
Environmental groups	Public health groups
Consumer rights groups	Academic institutions
Religious groups	Impacted citizens
Civil rights groups	Other federal agencies

One tool helpful in translating general goals into specific metrics is an objectives hierarchy, which is a hierarchic list starting with the overall goal of a project and moving down in levels to (component) purposes or outcomes, outputs and specific activities (see http://www.iac.wur.nl/ppme/content.php?ID=353&IDsub=338). A discussion of this is found in EPA's *Planning for Ecological Risk Assessment: Developing Management Objectives* (Section 3.4.2) at http://www.epa.gov/NCEA/raf/pdfs/eco_objectives-sab_6-01.pdf.

It is beneficial if planning and scoping participants understand the following six questions *before* the risk assessment begins:

- **What is the goal of the risk assessment and how will the results be used?** A risk assessment might be conducted to compare the costs of various emissions control options versus the benefits in terms of reduced risks. Some conduct risk assessments primarily for informational purposes – for example, how much do individual pollution sources contribute to total risks within a given community? Risk management goals may be risk-related (e.g., reducing risks from exposure to air toxics; reducing the incidence of a specific adverse effect such as cancer); economic (e.g., reducing risks without causing job loss or raising taxes); or related to public policy (e.g., protecting children and other sensitive populations). Generally, each risk assessment is designed to provide information that will support the identified goals.

- **What information will the risk assessors collect and what analyses will they perform on those data?** The risk assessors develop the scope of the risk assessment during planning and scoping. For example, participants may select a limited number of chemicals from all those released in an area to be analyzed throughout the risk assessment process (the chemicals of potential concern or COPC), or the assessment may focus on only a limited number of exposure pathways that may be most important. Stakeholders should understand exactly what the risk assessment is (and by extension, what it is not) going to evaluate.

- **What are the major concerns of the local community?** Significant concerns that the risk assessment does not address can result in "show stoppers" that complicate or delay the risk management decision. Clarifying what the risk assessment is not going to study, and why, before the assessment begins will help to reduce this possibility. As an example, many communities express concerns about perceived disease clusters. All stakeholders need to understand that the risk assessment process is not used to evaluate disease clusters or establish cause-effect relationships between air pollution and existing cases of disease. However, stakeholders often raise this concern, and it is imperative that the planning and

scoping team acknowledge these concerns and direct them to the appropriate resources. Given the prevalence of this concern in areas with air toxics concerns, this Volume includes a lengthy discussion in Part VI of this Volume on options for addressing such issues.

- **What are the roles and responsibilities of each participant?** Stakeholders often address many administrative issues during planning and scoping, including who will lead the risk assessment, who will perform each of the various tasks, who will pay for it, and when the participants need the results.

- **What are the available resources and schedules?** Time and money are always limited; therefore, the planning and scoping process will almost certainly involve trade-offs between the amount and quality of information participants desire and the time and monetary resources available to obtain and analyze the information. Participants often choose to determine critical milestones and institute a clear, yet reasonably flexible, schedule to keep the assessment on track.

- **What documentation and other products are required?** Regulatory requirements often include specific types of information in specific formats. In a community-level analysis, stakeholders may want specific information such as maps indicating estimated levels of air pollutants in different parts of the community. Thus, documentation requirements are meant to provide transparency throughout the risk assessment process, from the initiation of the planning and scoping step to the presentation of the final product. Participants are urged to document all important decisions, goals, discussions, schedules, resource allocations, roles and responsibilities, data quality objectives. Participants also may document the analytical approach such that anyone may follow the methodology of the risk assessment.

Finally, risk assessors, risk managers, and all other stakeholders generally recognize the sensitivity of their roles throughout the risk assessment process. Specifically, there must be no direct or indirect actions on the part of any stakeholder to influence the outcome of the science-based analysis. Even the appearance of such activity can severely undermine trust in the risk assessment as a valid analysis tool.

5.3.2.3 What is the Scope?

The risk assessment scope helps determine how comprehensive the analysis will be. The scope of a risk assessment may be narrow or broad, depending on the specific risk management goals. For example, a relatively broad goal such as “reducing risks from exposure to air toxics” may require a relatively broad risk assessment that examine many types of sources (e.g., stationary, mobile) and dozens of specific air toxics. In contrast, a more narrow goal such as “reducing the potential cancer risk in the community” may result in a risk assessment that focuses more narrowly on only those air toxics that contribute to cancer. Geography (e.g., political boundaries), demographics (e.g., focusing on a subset of exposed populations), legal requirements (e.g., statutes or regulations), or methodological or data limitations can all narrow the scope. Most importantly, time and money will almost always limit the scope of the risk assessment.

Participants can determine scope by listing and answering critical **assessment questions** such as:

- What specific sources are to be included?
- What specific air toxics are to be included?
- What are the physical boundaries of the study area?
- What are the temporal constraints of the study?
- What potential exposure pathways will be evaluated?
- What potentially exposed populations will be assessed?
- What types of health risks will be evaluated?

The details of scope (e.g., what sources are to be included, what potential pathways will be included) are developed during the problem formulation stage (see Chapter 6).

The goal of the scoping process is to produce a clear understanding of what the risk assessment should and should not include and why. For example, if available data or methods make it impossible to assess a potential exposure pathway, the planning and scoping team may need to re-evaluate the goals and expectations of the risk assessment process.

5.3.2.4 Why is There a Problem?

The **problem statement** often summarizes the end result of the scoping process, describing the specific concerns that the risk assessment will address. Problem statements often also include statements about how the risk assessors will evaluate these concerns. The problem statement is commonly as specific as possible and may also include explicit statements of what will not be assessed in the risk assessment.

Example Problem Statement

Air toxics emissions may be causing increased long-term inhalation health risk (both cancer and noncancer concerns) to people in the immediate vicinity of Acme Refining Company. A modeling risk assessment will be performed to evaluate potential long-term human health impacts of inhalation exposures to all air toxics emitted by the facility. Inhalation risks for populations within 50 km of the Acme property boundary will be assessed under residential exposure conditions. Non-inhalation pathways will not be assessed for either human or ecological receptors.

5.3.2.5 How will Risk Managers Evaluate the Concern?

The risk assessments are most often designed to provide input to risk managers to help inform the decisions they must make. Part of the planning and scoping process is developing an understanding of the types of information needed by the risk managers and the level of uncertainty in that information that can be tolerated. It does not make sense to conduct an expensive risk assessment if the eventual results will not be helpful to decision makers.

5.3.2.6 Lessons Learned on Planning and Scoping

EPA's Science Policy Council has evaluated the planning and scoping process, particularly as it relates to cumulative risk assessments (http://www.epa.gov/osp/spc/2cumrisk.htm). From an assessment of five case studies, a working group identified the following lessons learned:[8]

- Early and extensive involvement of the risk manager (decision maker) helped focus the process toward a tangible product.

- Purporting that planning and scoping will be quick and easy is likely to be counterproductive; it is a lot more work than people assume. However, it ultimately saves time by helping to organize everyone's thinking and usually results in a better quality assessment.

- Stakeholder engagement is essential at the beginning, because their patience is directly proportional to their sense of influence in the process. They have been helpful in identifying important public health endpoints that were not initially considered by EPA in the process of developing a conceptual model.

- Conceptual models are helpful in demonstrating how one program relates to other regulatory activities as well as the relationship between stressors and effects beyond traditional regulatory paradigms.

- Debate over terminology and brainstorming sessions are necessary to reach a consensus. A clear set of definitions aids this process.

- The planning and scoping process cannot be prescriptive, because the context of each situation is different. Planning and scoping is particularly valuable when the assessment will be complex, controversial, or precedential. At this time, planning and scoping usually precede cumulative risk assessments.

- Clear objectives, resource commitments, and estimated schedules from management will drive the approach and level of detail that can be considered.

- Explaining uncertainty to stakeholders is critical despite a hesitancy to reveal all that is known and not known about chemical risks. While revealing these uncertainties may lead to criticism and political ramifications, it can also develop a sense of trust, credibility, and support for the decision making process.

It should also be noted that the entire planning and scoping (and risk assessment process) is inherently iterative in nature. As the analysis proceeds and participants learn more about the study area, participants may find the initial assumptions in the conceptual model inadequate and they will need to modify the conceptual model (and, thus, the analysis plan). For example, suppose a conceptual model was developed that assumed a chemical was released from a facility that is generally thought to deposit quickly from the air, is highly persistent, and has a large bioaccumulation potential, thus requiring a multipathway analysis. Once the emissions inventory is verified, it is found that this chemical is actually not used or produced by facility, rendering the multipathway analysis moot for this chemical. (Multipathway analysis may still be needed for other chemicals in the emissions.)

When such changes are required in the conceptual model and analysis plan, all key stakeholders may be apprised of the change and ideally agree to any alterations in the goals of the overall assessment. The initial goal of "no surprises at the end of the assessment" is still maintained in light of evolving information.

References

1. U.S. Environmental Protection Agency. 2003. *Framework for Cumulative Risk Assessment*. Risk Assessment Forum, Washington, DC 20460, May 2003, EPA/630/P02/001F. Available at http://cfpub.epa.gov/ncea/raf/recordisplay.cfm?deid=54944.

2. U.S. Environmental Protection Agency. 1999. *Residual Risk Report to Congress*. Office of Air Quality Planning and Standards, Research Triangle Park, NC 27711, March 1999, EPA/453/R99/001. Available at http://www.epa.gov/ttncaaa1/t3/reports/risk_rep.pdf.

3. U.S. Environmental Protection Agency. 1992. *Guidelines for Exposure Assessment*. *Federal Register* 57:22888-22938, May 29, 1992. Available at http://cfpub.epa.gov/ncea/raf/recordisplay.cfm?deid=15263.

4. U.S. Environmental Protection Agency. 1997. *Guidance on Cumulative Risk Assessment. Part 1. Planning and Scoping* Science Policy Council, Washington, D.C. Available at: http://www.epa.gov/osp/spc/cumrisk2.htm.

5. U.S. Environmental Protection Agency. 2003. *Framework for Cumulative Risk Assessment*. Risk Assessment Forum, Washington, D.C. 2003. EPA/630/P02/001F. Available at: http://cfpub.epa.gov/ncea/raf/recordisplay.cfm?deid=54944.

6. U.S. Environmental Protection Agency. 2001. *Risk Assessment Guidance for Superfund (RAGS): Volume I - Human Health Evaluation Manual (Part D, Standardized Planning, Reporting and Review of Superfund Risk Assessments) Final*. Office of Emergency and Remedial Response, Washington, D.C. Available at: http://www.epa.gov/superfund/programs/risk/ragsd/index.htm.

7. U.S. Environmental Protection Agency. 1999. *Risk Assessment Guidance for Superfund: Volume 1 - Human Health Evaluation Manual. Supplement to Part A, Community Involvement in Superfund Risk Assessments.* Office of Solid Waste and Emergency Response, Washington, D.C. EPA/540/R98/042/PB99/963303.

8. U.S. Environmental Protection Agency. 2002. *Lessons Learned on Planning and Scoping for Environmental Risk Assessments*. Science Policy Council, Washington, D.C. Available at: http://www.epa.gov/osp/spc/handbook.pdf.

Chapter 6 Problem Formulation: Inhalation Risk Assessment

Table of Contents

6.1 Introduction 1

6.2 Developing the Conceptual Model 1

6.3 Developing the Analysis Plan 4
- 6.3.1 Identification of the Sources 5
- 6.3.2 Identification of the Chemicals of Potential Concern 6
  - 6.3.2.1 Toxicity-Weighted Screening Analysis 6
  - 6.3.2.2 Risk-Based Screening Analysis 7
- 6.3.3 Identification of the Exposure Pathways/Routes 8
  - 6.3.3.1 Characteristics of the Assessment Area 12
  - 6.3.3.2 Scale of the Assessment Area 14
  - 6.3.3.3 Use of Modeling versus Monitoring 14
  - 6.3.3.4 Estimation of Exposure 17
  - 6.3.3.5 Evaluation of Uncertainty 20
  - 6.3.3.6 Preparation of Documentation 20
- 6.3.4 Identification of the Exposed Population 20
- 6.3.5 Identification of the Endpoints and Metrics 21

6.4 Data Quality in the Risk Assessment Process 22

References 24

6.1 Introduction

This chapter discusses the problem formulation step, which takes the results of the planning and scoping process and translates them into two critical products:

- A **conceptual model** that explicitly identifies the sources, receptors, exposure pathways, and potential adverse human health effects that the risk assessment will evaluate (described in Section 6.2); and

- An **analysis plan** that outlines the analytical approaches that will be used in the risk assessment (described in Section 6.3).

An additional section on data quality (Section 6.4) is also included as a reference for those portions of the risk assessment that involve data collection (e.g., emissions inventories, monitoring). EPA's *Framework for Cumulative Risk Assessment*[1] provides a more detailed discussion of the problem formulation process.

6.2 Developing the Conceptual Model

The general concern and approach articulated in the problem statement usually receives more detail in a **study-specific conceptual model**. This model explicitly identifies the sources, receptors, exposure pathways, and potential adverse human health effects that the risk assessment is going to evaluate. The study-specific conceptual model comprises both a picture and written description that illustrate: the current understanding of what sources are releasing air toxics in a particular place; how the chemicals may be transported from the point of release to the point where people can breathe them; and the types of health effects that may result. Risk assessors commonly include both a pictorial illustration (such as a technical drawing) and a narrative description of each of the above elements in the conceptual model.

The conceptual model establishes the physical boundaries of the assessment area and focuses the risk assessment on several key elements, including sources, chemicals released, fate and transport mechanisms, potentially exposed populations, potential exposure pathways and routes of exposure (e.g., breathing, ingesting), and potential adverse effects. Although participants may revise or refine the conceptual model during the risk assessment, it is important to develop an initial conceptual model early on.

Critical elements to be included in the conceptual model include:

- **The sources of air toxics**. The identity, location (latitude/longitude), and physical nature of the sources being evaluated (which may include factories, small businesses, cars/trucks, forest fires, etc.), including general emissions characteristics (e.g., stack locations, heights, other stack parameters, control device efficiency, operating schedules).

- **Stressors**. The specific air toxics that will be evaluated. Information on air toxics may come from emissions inventories, previous monitoring or modeling studies, permits, or estimates based on the principal processes or activities occurring at the source or site. Many risk assessments begin with a relatively large number of stressors that are of potential concern

(**chemicals of potential concern**, or COPC) and narrow these to the subset that contributes most to exposure and risk.

- **The exposure pathways/media of concern**. The environmental compartments into which the air toxics move after they are released and through which human exposure can occur. Once released from the sources, air toxics begin to disperse by the wind away from the point of release and may remain airborne; convert into a different substance; and/or deposit out of the air onto soils, water, or plants. People may be exposed to air toxics by breathing contaminated outdoor and/or indoor air (inhalation); ingestion (for the small number of air toxics that can accumulate in soils, sediments, and foods – a process called bioaccumulation); and skin (dermal) contact with deposited air toxics. Air toxics risk assessments always evaluate the inhalation exposure pathway. However, when sources release chemicals that persist and which also may bioaccumulate, analysis of non-inhalation pathways may also be necessary (see Parts III and IV for information on inhalation pathways).

> **Chemicals of Potential Concern (COPC)**
>
> Chemicals of potential concern (COPC) are those air toxics that are evaluated in the risk assessment because they have the potential to affect the risk management decision. The corresponding term for ecological risk assessment are chemicals of potential ecological concern (COPEC). The risk assessment often finds that most of the risk is associated with a subset of the COPC. The subset, which drives the risk management decisions, is referred to as chemicals of concern (COC).

- **Routes of exposure**. Potential routes of exposure include inhalation, ingestion, and dermal absorption.

- **Subpopulations**. The human populations potentially receiving exposure to the air toxics, including information about demographics (race, ethnicity, economic status, etc.) and potentially sensitive subgroups (e.g., elderly, children). Depending on the goals of the risk assessment, the conceptual model may need to consider populations currently living in a given area as well as those that might move into the area in the future.

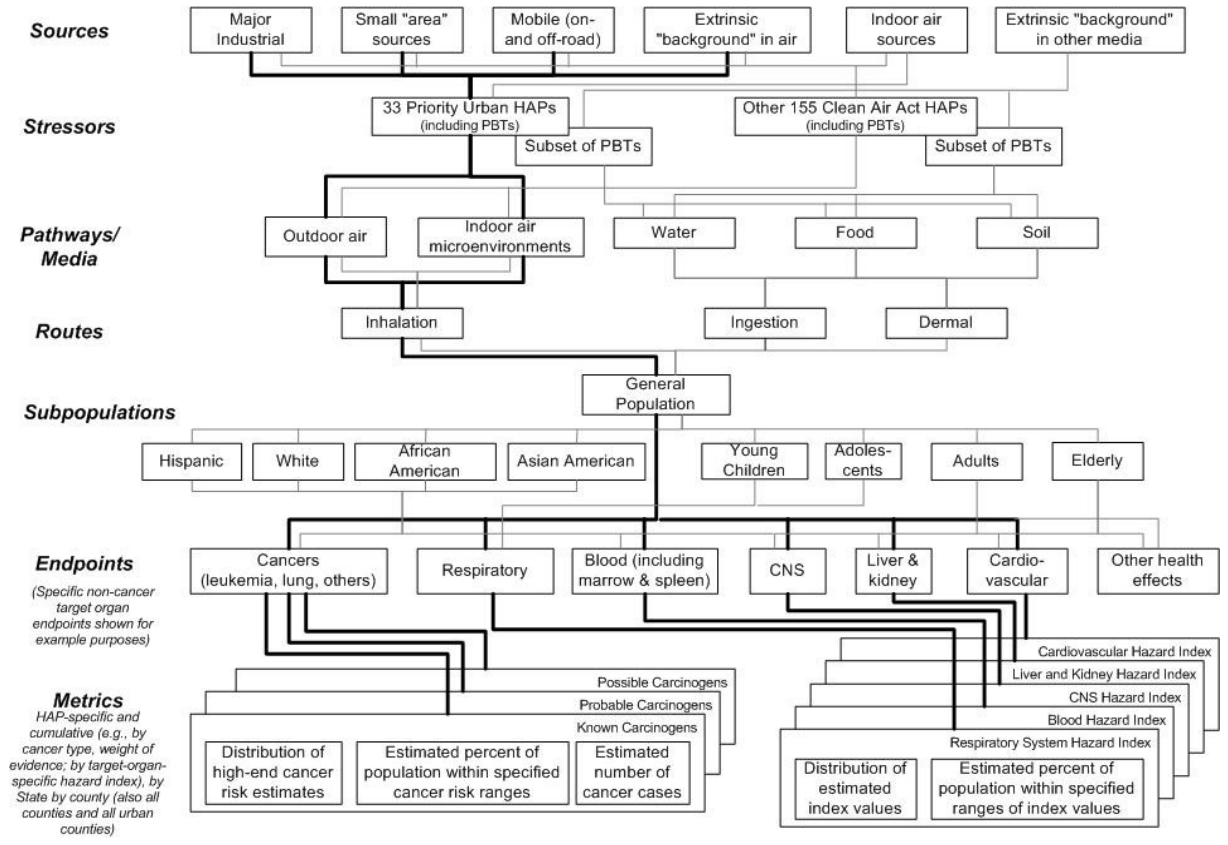
- **Endpoints**. The harmful effects that may result from exposure to air toxics, including cancer, respiratory effects, birth defects, and reproductive and neurological disorders. Air toxics can damage the organs at the initial point of contact or enter the body and move via the bloodstream to other target organs or tissues. Choice of endpoints generally depends on the toxic effects exhibited by the specific air toxics being assessed. Risk assessors generally represent potential adverse health effects to humans from exposure to air toxics through the inhalation pathway as cancer and noncancer outcomes (see Exhibit 5-3). Unless risk assessors study a specific chemical that is linked to a specific health outcome (which is not usually the case), a general statement that "risk of cancer and noncancer hazard will be evaluated" is usually sufficient.

- **Metrics**. It should be determined how cancer risk and noncancer hazard will be estimated and reported.

Exhibit 6-1 provides an example of a generalized conceptual model for air toxics risk assessments with examples of possible linkages. The example shown is a graphical illustration;

it would also be possible to develop a pictorial illustration. The conceptual model for a specific risk assessment will likely include only part of this general model. For example, pathways involving soil, water, and food will only be included if PB-HAP compounds are COPC. In the conceptual model, the sources, pathways, and expected health outcomes are drawn to illustrate what the assessors think may be happening in the study when sources are releasing air toxics to the environment. For a specific study, risk assessors would augment the illustration with the actual names/locations of sources, the COPC they release, the populations of concern and their location, and the specific health outcomes of concern (the generic endpoints of cancer and noncancer health outcomes, as drawn here, are usually sufficient for this stage of the assessment). The accompanying narrative will describe each of the elements of the illustration in detail and will provide sufficient information to clarify the critical elements of each piece of the picture.

Exhibit 6-1. Generalized Conceptual Model for Air Toxics Risk Assessments



This figure illustrates a general conceptual model for air toxics risk assessments with examples of potential linkages. The conceptual model for a specific risk assessment will likely include only part of this general model. In this figure, the heavy lines represent the conceptual model used for the initial National-scale Air Toxics Assessment (*Draft for EPA Science Advisory Board Review*, available online at http://www.epa.gov/ttn/atw/sab/sabrev.html). This assessment focused on 33 air toxics and was limited to inhalation exposures. Cancer and noncancer endpoints were assessed using distributions, estimated percentages of the population within specified risk or hazard index ranges, and estimated incidence (only for cancer cases).

If PB-HAP compounds identified in Exhibit 4-2 (or other air toxics that persist and may bioaccumulate and/or bioconcentrate) are present in emissions, both the conceptual model and the analysis plan may need to consider pathways other than inhalation (e.g., deposition to soil and surface waters, uptake by biota, and ingestion of these media and biota) for human and ecological receptors. For purposes of this Reference Manual, we discuss the elements/considerations for the conceptual model and analysis plan that are particular to multipathway human health risk assessment in Part III and ecological risk assessment in Part IV. However, the planning, scoping, and problem formulation process specific to multipathway analyses is generally integrated with the process for the inhalation analysis as early as feasible.

6.3 Developing the Analysis Plan

Risk assessors use the study-specific conceptual model as a guide to help determine what types, amount, and quality of data are needed for the study to answer the questions the risk assessment has set out to evaluate. Specifically, the analysis plan matches each element of the conceptual model with the analytical approach that the assessors will use to develop data about that element (Exhibit 6-2).

Most often, the analysis plan details the link between each element of the conceptual model and the specific analytical approach. The participants would then describe each of the analytical approaches in sufficient detail to provide the risk assessors with sufficient direction to allow them to produce the desired high quality data. For example, when determining exposure concentrations of COPC at the point of exposure to humans, the analysis plan will describe the exact sampling/analytical lab methods and/or models that risk assessors will use to generate this data, who will perform the analyses, when the analyses will be done, quality assurance/quality control requirements (including data validation procedures), roles/responsibilities of analysts, and documentation requirements. This section of the analysis plan would also provide a discussion of how data gaps should be identified and documented and how assessors will address uncertainties.

The analysis plan may also include a comparison between the level of confidence needed for the management decision and the actual level of confidence it expects from alternative analytical approaches; this will determine which alternative best meets the management goals, within the constraints of time and resources. In addition, the analytical approach may include a phased or tiered risk assessment approach to facilitate management decisions (see Section 6.4 below).

The analysis plan is most helpful when it contains explicit statements of how participants selected the various analytical approaches, what piece of the conceptual model they intended the approach to evaluate, how the approach integrates with other analytical elements, and specific milestones for completing the risk assessment. Assessors generally include uncertainties associated with analyses, and approaches for addressing these uncertainties, in the analysis plan when possible.

Exhibit 6-2. Important Elements to be Included in an Analysis Plan	
Sources	How will information on the sources in the analysis (e.g., source location, important release parameters) be obtained and analyzed?
Pollutants	How will chemicals of potential concern (COPC) be confirmed and their emissions values be estimated?
Exposure pathways	How will the identified exposure pathways be assessed? How will ambient concentrations be estimated?
Exposed population(s)	How will exposures to populations of interest be characterized? How will their exposure concentrations be estimated? What will be the temporal resolution? What sensitive subpopulations may be affected?
Endpoints	How will information on the toxicity of the COPC be obtained (what are the data sources)? What risk metrics will be derived for the risk characterization?
In addressing the above aspects of the analysis, the plan should also clearly describe the following: • How will *quality* be ensured in each step (e.g., what will be included in the quality assurance/quality control plans)? • How will *uncertainty and variability* in the results be assessed? • How will all stages of the assessment be *documented*? • Who are the *participants* and what are their *roles and responsibilities* in the various activities? • What is the *schedule* for each step (including milestones)? • What are the *resources* (e.g., time, money, personnel) being allocated for each step?	

The analysis plan may not result in just one document, but rather in a combination of multiple work plans that, taken together, constitute "the analysis plan." For example, for a study where assessors will perform both air dispersion modeling and air monitoring, participants may develop a separate work plan for both modeling and monitoring. However, assessors usually develop a master plan that describes all the different pieces and their relationship to one other.

The remainder of this subsection describes the important elements of the analysis plan, including:

- Identification of sources;
- Identification of chemicals of potential concern;
- Identification of exposure pathways/routes;
- Identification of exposed populations; and
- Identification of endpoints and metrics.

6.3.1 Identification of the Sources

As noted in Part I, EPA classifies sources of air toxics into a variety of categories for regulatory purposes, including stationary sources, mobile sources, and indoor sources (see Chapter 4). In addition, risk assessors also commonly group substances by their chemical and physical properties to both better estimate the fate and transport of chemicals in the environment and to

make inferences about the types of exposure pathways likely to be important in the exposure assessment.

This part of the analysis plan specifies the approach to be used to identify the specific sources that will form the initial focus of the analysis. Depending on the goals of the risk assessment, these sources may be limited to a single source or multiple sources at a facility (i.e., facility-specific risk assessments discussed in Volume II of this reference library) or may cover a wider variety of sources, including mobile sources, stationary sources, and possibly other sources such as indoor and natural sources (e.g., community-based risk assessments discussed in Volume III of this reference library). Identifying sources may be relatively straightforward (e.g., for facility-specific risk assessments) or may involve considerable research, particularly when dealing with a large number of smaller sources. In such an analysis, the initial tier of evaluation generally focuses on all identifiable sources within the assessment area. In subsequent tiers, it may be possible to remove some of these sources from the exposure assessment if one can determine that they contribute a very small fraction to the total risk estimate. Chapter 12 contains the techniques for conducting this type of screening.

6.3.2 Identification of the Chemicals of Potential Concern

This part of the analysis plan specifies the approach to be used to identify the most important air toxics that sources release (i.e., the chemicals of potential concern, or COPC). The COPCs will be the primary focus of the exposure and risk assessment. The initial tier of analysis often includes all of the air toxics released from the identified important sources. Depending on the specific air toxics of concern, the risk assessment also may need to consider secondary compounds that are formed from the reaction in the atmosphere.

Two techniques are available to focus the risk assessment on the most important air toxics:

- During problem formulation, a simple toxicity-emissions weighted screening approach can be conducted (discussed in Section 6.3.2.1).

- Once an initial risk characterization has been performed, subsequent tiers of analysis may remove specific chemicals from the COPC list if they are determined to contribute only a very small fraction to the total risk estimate (discussed in Section 6.3.2.2).

(Note that some assessors may wish to simply carry through the analysis all of the chemicals emitted to the assessment area. This is appropriate; however, it may require sufficient resources and result in little useful information.)

6.3.2.1 Toxicity-Weighted Screening Analysis

To determine which air toxics to include in the Tier 1 inhalation risk assessment, a relative risk evaluation called a **toxicity-weighted screening analysis (TWSA)** may be calculated based on the emissions data for all air toxics released from the facility/source being assessed. A TWSA is particularly useful if there are a large number of air toxics in the facility/source emissions and there is a desire to focus the risk analysis on a smaller subset of air toxics that contribute the most to risk. A TWSA can be performed as described below.

The TWSA is intended to be entirely emissions- and toxicity-based, without considering dispersion, fate, receptor locations, and other exposure parameters. It essentially compares the emissions rates of each air toxic to a hypothetical substance with an inhalation unit risk value of 1 per $\mu g/m^3$ (for carcinogenic effects) and/or a reference concentration (RfC) of 1 mg/m^3 (for noncancer effects). It requires emissions (release) information as well as the applicable dose-response values (see Chapter 12). However, is also can be used even with a single emission point and many air toxics. The steps for emissions-based toxicity-emissions weighted screening are presented below.

1. Identify all the inhalation unit risks (IURs) and RfCs for the air toxics in the facility/source emissions.
2. Determine the emission rate (e.g., tons/year) of each air toxic.
3. Multiply the emission rate of each air toxic by its IUR to obtain a toxicity-emissions product.
4. Rank-order the toxicity-emissions products and obtain the sum of all products.
5. Starting with the highest ranking product, proceed down the list until the cumulative sum of the products reaches a high proportion (e.g., 99 percent) of the total of the products for all the air toxics. Include in the assessment all the air toxics that contributed that proportion (e.g., 99 percent) of the total (see Exhibit 6-3 for an example calculation).
6. Repeat steps 3-5, but instead divide the emissions rate by the RfCs to obtain "noncancer equivalent tons"/year (see Exhibit 6-4 for an example calculation).

Chemicals with no toxicity data will necessarily not be included in the initial list of COPCs identified by the TWSA screening process. However, this does not necessarily mean that they are not potential risk drivers. Chemicals with no toxicity data are to be evaluated as part of the overall uncertainty analysis for the risk assessment. If there is sufficient evidence to support the hypothesis that an omitted chemical is a potential risk driver, the risk assessment team may opt to develop a toxicity value for the chemical (see Chapter 12 for more information on identifying toxicity values for chemicals). Also, if evidence suggests that a chemical that is screened out (e.g., is below the 99th percentile in the TWSA) would nevertheless have an individual HQ or cancer risk greater than the selected screening level, the assessor may consider keeping the chemical in the list of COPCs.

6.3.2.2 Risk-Based Screening Analysis

In subsequent tiers of analysis, a **risk-based screening analysis** can be used to further focus the assessment on the significant air toxics of concern. This approach would be similar to the TWSA except that estimated individual cancer risk and noncancer hazard estimates would be used instead of toxicity-weighted emissions (an example risk-based screening analysis is presented in Chapter 13). A risk-based screening analysis might include the following steps:

1. Using applicable input data, run a simple dispersion and/or exposure model and calculate cancer risk at a selected point (e.g., maximum exposed individual location).
2. Rank-order the individual risk estimates for each emitted air toxic and obtain the sum of the cancer risk.
3. Starting with the highest ranking cancer risk, proceed down the list until the individual air toxics contributing a large proportion (e.g., 99 percent) of the total risk are included. Include those air toxics in subsequent tiers of analysis.
4. Repeat steps 1-3 for noncancer hazard.

Exhibit 6-3. Example TWSA Calculation for Cancer Effects					
Air Toxic	**Emissions (tons/year)**	**IUR**	**Cancer Equivalent Tons/year**	**Percent of Total**	**Cumulative Percent**
1,3-butadiene	8.2×10^{1}	3.0×10^{-5}	2.5×10^{-3}	23.8%	23.8%
carbon tetrachloride	1.5×10^{2}	1.5×10^{-5}	2.2×10^{-3}	21.3%	45.1%
beryllium compounds	8.6×10^{-1}	2.4×10^{-3}	2.1×10^{-3}	19.8%	64.9%
arsenic compounds	4.2×10^{-1}	4.3×10^{-3}	1.8×10^{-3}	17.5%	82.4%
2,3,7,8-TCDD	2.0×10^{-5}	3.3×10^{1}	6.6×10^{-4}	6.4%	88.8%
chromium (VI) compounds	3.7×10^{-2}	1.2×10^{-2}	4.4×10^{-4}	4.3%	93.1%
polycyclic organic matter[(a)]	4.3	2.1×10^{-1}	3.7×10^{-4}	3.6%	96.7%
cadmium compounds	1.0×10^{-1}	1.8×10^{-3}	1.8×10^{-4}	1.8%	98.4%
formaldehyde	8.9	1.3×10^{-5}	1.2×10^{-4}	1.1%	99.5%
1,3-dichloropropene	5.2	4.0×10^{-6}	2.1×10^{-5}	0.2%	99.7%
allyl chloride	2.8	6.0×10^{-6}	1.7×10^{-5}	0.2%	99.9%
methylene chloride	1.9×10^{1}	4.7×10^{-7}	8.7×10^{-6}	0.1%	100.0%
benzene	9.3×10^{-2}	7.8×10^{-6}	7.3×10^{-7}	0.0%	100.0%
Total			1.0×10^{-2}	100.0%	

Heavy line denotes 99% cutoff. In this example, 1,3-dichloropropene, allyl chloride, methylene chloride, and benzene could be dropped from the cancer analysis.
[(a)] Cancer equivalent tons/year and IUR are based on the assumption that benzo(a)pyrene represents 5% of emissions.

6.3.3 Identification of the Exposure Pathways/Routes

This part of the analysis plan specifies the approach to be used to identify the specific exposure pathways/routes that will be assessed. An exposure pathway/route describes the movement of air toxics from the point of release to the point where exposure may occur and generally consists of four elements:

1. A source and mechanism of release (emissions);
2. A transport medium (for inhalation, air);
3. A point of potential human contact with the contaminated medium (the **exposure point**); and
4. An exposure route at the contact point (e.g., inhalation).

Exhibit 6-4. Example TWSA Calculation for Noncancer Effects					
Air Toxic	**Emissions (tons/year)**	**RfC**	**Noncancer Equivalent Tons/year**	**Percent of Total**	**Cumulative Percent**
beryllium compounds	8.6×10^{-1}	2.0×10^{-5}	4.3×10^{4}	38.3%	38.3%
1,3 butadiene	8.2×10^{1}	2.0×10^{-3}	4.1×10^{4}	36.7%	75.0%
arsenic compounds	4.2×10^{-1}	3.0×10^{-5}	1.4×10^{4}	12.6%	87.6%
cadmium compounds	1.0×10^{-1}	2.0×10^{-5}	5.1×10^{3}	4.6%	92.1%
carbon tetrachloride	1.5×10^{2}	4.0×10^{-2}	3.7×10^{3}	3.3%	95.4%
allyl chloride	2.8	1.0×10^{-3}	2.8×10^{3}	2.5%	97.9%
formaldehyde	8.9	9.8×10^{-3}	9.1×10^{2}	0.8%	98.7%
2,3,7,8-TCDD	2.0×10^{-5}	4.0×10^{-8}	5.0×10^{2}	0.4%	99.1%
chromium (VI) compounds	3.7×10^{-2}	1.0×10^{-4}	3.7×10^{2}	0.3%	99.5%
toluene	1.3×10^{2}	4.0×10^{-1}	3.2×10^{2}	0.3%	99.8%
1,3-dichloropropene	5.2	2.0×10^{-2}	2.6×10^{2}	0.2%	100.0%
methylene chloride	1.9×10^{1}	1.0	1.9×10^{1}	0.0%	100.0%
benzene	9.3×10^{-2}	6.0×10^{-2}	1.6	0.0%	100.0%
		Total	1.1×10^{5}	100.0%	
Heavy line denotes 99% cutoff. In this example, chromium (VI) compounds, toluene, 1,3-dichloropropene, methylene chloride, and benzene could be dropped from the noncancer analysis.					

A critical determination in the exposure assessment is whether the potential exposure pathways identified during scoping are **complete** (i.e., there is a plausible mechanism by which the air toxic emitted from the source can reach the exposure point and a plausible mechanism by which the human receptor can come into contact with the chemical at the exposure point). Exposure cannot occur without a complete exposure pathway; and therefore if assessors determine that a potential exposure pathway is incomplete, they will generally document and drop the exposure from the risk assessment.

The exposures to be assessed depend on the needs articulated in the planning and scoping and problem formulation steps, including the specific laws and regulations that mandate a potential decision. For example, air toxics risk assessments commonly rely primarily on current land uses when evaluating exposures, while risk assessments conducted in the Superfund program commonly assess current and future land uses (i.e., air toxics risk assessments usually presume that the current land use within the area of impact of a source(s) will remain unchanged into the foreseeable future). The need, reasons, and methodology to evaluate alternate (e.g., future) land use conditions may be carefully considered and fully articulated during the problem formulation and planning/scoping phase of the assessment. As will be discussed later, in screening-level air

toxics risk assessments, it is common to assess exposures at the point of maximum offsite ambient concentrations, whether or not someone actually lives there (the maximum exposed individual or MEI location).

In addition, advanced tools (such as the RAIMI approach; see Volume III of this reference library) allow exposure assessments to evaluate the contemporaneous impact of multiple sources on a assessment area, identify the main contributors to the impact, and evaluate "what if" scenarios (e.g., what if this source cut its emissions by half; what if a roadway doubled its traffic?). Ultimately, the needs of the risk manager will drive such decisions.

For inhalation risk assessments, assessors evaluate only one exposure pathway (inhalation); multipathway risk assessments, on the other hand, focus on all relevant pathways (i.e., inhalation *and* any other relevant pathway, such as ingestion or dermal; see Part III of this Reference Manual for a description of how multipathway analyses are done). Exhibit 6-5 illustrates the exposure pathways/routes that are commonly assessed for air toxics inhalation risk assessments. Note that depending on the types of sources and specific COPCs they release, some of these pathways may or may not be relevant for any particular study.

Exhibit 6-5. Most Commonly Assessed Exposure Pathways/Routes for Air Toxics Inhalation Risk Assessments
Outdoor emissions of vapor phase chemicals → outdoor air → indoor air (by penetration of outdoor air into indoor spaces) Outdoor emissions of particles → outdoor air → indoor air (by penetration of outdoor air into indoor spaces)
Note: • Other media/routes may be applicable for particular risk assessments; • When available, information on indoor source contributions may also be considered.

Whether the exposures to be assessed include workers depends on the needs articulated in the planning/scoping and problem formulation steps. For example, the Department of Labor's Occupational Safety and Health Administration (OSHA) generally regulates the exposures of workers to the chemicals they are exposed to in their workplace, and therefore these exposures generally are not considered in an air toxics risk assessment. When workers are exposed to chemicals not generated in their workplace (e.g., office workers exposed by a nearby factory), a decision may be made to consider the risks.

Exhibit 6-6 provides an example of an exposure pathway evaluation summary for a hypothetical study. The exposure pathways identified for further assessment will depend on the specific types of chemicals released (including their chemical and physical form), the physical relationship of the sources to the human receptors, meteorological conditions, and the relationship between indoor and outdoor air for the chemicals under study (for indoor exposure component).

Exhibit 6-6. Example Illustrating Possible Complete Exposure Pathways for a Hypothetical Inhalation Air Toxics Risk Assessment			
Potentially Exposed Population	**Exposure Route, Medium, and Exposure Point**	**Pathway Selected for Evaluation?**	**Reason for Selection or Exclusion**
Current Land Use Residents living in Smallville, USA	Inhalation of vapor phase chemicals during outdoor activities	Yes	Residents live year-round in Smallville
	Inhalation of particulate matter during outdoor activities	No	Preliminary analysis suggests that no significant particulate matter is released from sources in the assessment area and that the chemicals released remain in the vapor phase
	Inhalation of vapor phase chemicals during indoor activities	Yes	Residents live year-round in Smallville and released chemicals have the potential to penetrate indoors; the COPC are also released by indoor sources
	Inhalation of particle phase chemicals during indoor activities	No	Residents live year-round in Smallville and no significant particulate matter is released from sources in the assessment area and the chemicals released remain in the vapor phase. There are no known indoor sources.
Note: Assessment of completed non-inhalation exposure pathways are discussed in Part III of this reference manual.			

The approach for characterizing exposure pathways/routes in the analysis plan usually considers a variety of information about the assessment area (as articulated in the conceptual model), including how it will be bounded for the analysis. The analysis plan also specifies how exposure will be estimated and quantified, including whether modeling and/or monitoring will be used. The following subsections discuss:

- Characteristics of the assessment area;
- Scale of the assessment area;
- Use of modeling versus monitoring; and
- Quantification of exposure.

6.3.3.1 Characteristics of the Assessment Area

The physical characteristics of the assessment area provide a basis for identifying potential exposure pathways/routes and receptor populations of concern. They also are important considerations for selecting and providing input parameters for the air quality models to be used and/or for establishing monitoring sites. There is no universal classification system for describing the characteristics of the assessment area, but the following information is generally important for inhalation exposure assessments:

- **Urban versus rural setting**. This distinction provides general information about the way that air toxics will disperse in the environment once released and the expected number and types of receptors. For example, releases in rural areas may tend to move downwind with a relatively simple dispersion pattern, while releases in a large city are likely to disperse in very complex patterns depending the size and placement of buildings. Additionally, some of the newer dispersion models can adjust both for direction dependencies as well as time of year due to changes in foliage.

- **Simple versus complex terrain**. Terrain affects both the way that air toxics will disperse in the environment once released and the amount of dilution that will occur before they reach receptors. For example, a plume might pass over nearby receptors in simple terrain, but might intercept receptors located on elevated terrain (e.g., a plateau or hill) at the same distance from the source. Assessors can determine the terrain of any area in the United States from topographic maps available from the USGS (see below).

- **Climate and meteorology**. Climate features such as temperature and precipitation patterns, and meteorological features such as wind speed and direction will affect the fate and movement of air toxics in the atmosphere and after deposition. Seasonal and diurnal conditions may be major factors affecting rates of contaminant migration where precipitation rates or temperatures vary greatly according to the season or time of day. It also is important to note whether unusual weather conditions occur frequently within the assessment area, as these can have significant effects on contaminant fate and transport (see Appendix G).

- **Other important geographic features**. Nearby geographic features such as a lake or ocean can have significant effects on contaminant dispersion and may require the use of special dispersion models (see Chapter 9). For multipathway human health and/or ecological risk assessments, exposure setting also may include such elements as water bodies and associated watersheds, ecological receptors, and agricultural lands (see Parts III and IV).

Current land use (and in limited instances, potential future land use) is an important factor to consider in determining the exposure pathways and specific exposure points that are commonly evaluated in the risk assessment (particularly for higher-tier risk assessments). Land use can typically be identified by reviewing hard copy and/or electronic versions of land use land classification (LULC) maps, topographic maps, and aerial photographs. Sources and general information associated with each of these data types or maps are presented below. Also, assessors may want to verify the Universal Transverse Mercator (UTM) coordinate system format (North American Datum 27 (NAD27) or NAD83) to ensure consistency and prevent erroneous geo-referencing of locations and areas.

- **Land Use Land Cover (LULC) Maps**. LULC maps can be downloaded directly from the U.S. Geological Survey website (http://edc.usgs.gov/geodata/), at a scale of 1:250,000, in a file type Geographic Information Retrieval and Analysis System (GIRAS) format. LULC maps can also be downloaded from the website (http://www.epa.gov/ngispgm3/spdata/EPAGIRAS/egiras/), at a scale of 1:250,000, in an Arc/Info export format. It is recommended that the exact boundaries of polygon land use area coverages, in areas being considered for evaluation, be verified using available topographic maps and aerial photographic coverages.

- **Topographic Maps**. Topographic maps are readily available in both hard copy and electronic format directly from USGS (http://mapping.usgs.gov/index.html) or numerous other vendors. These maps are commonly at a scale of 1:24,000, and in a TIFF file format with TIFF World File included for georeferencing.

- **Aerial Photographs**. Hard copy aerial photographs can be purchased directly from USGS (http://mapping.usgs.gov/index.html) in a variety of scales and coverages. Electronic format aerial photographs or Digital Ortho Quarter Quads (DOQQs) can also be purchased directly from USGS, or from an increasing number of commercial sources, such as Microsoft's® areal photo map server called "terraserver" (http://www.terraserver.com).

While these data sources do not represent the full universe of information available on human activities or land use, they are readily available from a number of government sources (typically accessible via the Internet), usually can be obtained at no or low cost, and when used together provide a good starting point to identify and define, in a defensible manner, land use areas to be considered for evaluation in the risk assessment. However, while the use of these or other data can be very accurate, verifying identified land use areas "on the ground" may be important for higher-tier risk assessments. Discussions with representatives of private and government organizations which routinely collect and evaluate land use data (e.g., agricultural extension agencies, U.S. Department of Agriculture, natural resource and park agencies, and local governments) can also be helpful in updating current land use information or providing information regarding future land use. Information on reasonable potential future land use can also be obtained from local planning and zoning authorities, which may help determine what level of development is now allowed under current regulations and what development is expected in the future. EPA's Superfund program has developed a specific directive on the process of how to go about determining future land use in a particular place.[2] This directive may be consulted for information on how to formulate realistic assumptions regarding future land use.

6.3.3.2 Scale of the Assessment Area

The scale of the assessment area is determined to a large part by the specific question(s) or problem(s) being addressed in the risk assessment. In determining the scale of the assessment area, both the capabilities of the tools to be used and the physical characteristics of the assessment area are considered by assessors. For example, some commonly used air dispersion models are only considered by EPA to be valid out to about 50 km because of limitations in their conceptual basis (e.g., Gaussian plume modeling has this limitation). A 50-km limit may be sufficient for assessments that focus on highly impacted areas occurring within a few kilometers of the emissions sources. However, other situations may involve a more distant area of significant impact. For example, if there are unusual source characteristics such as very tall stacks or unusual physical characteristics such as a nearby plateau where people live, modeling may need to be extended to these more distant areas.

A separate, but related issue, is how to consider scale for assessments that incorporate monitoring to characterize exposure. Since a monitor only assesses exposure at the point where the monitor is located, the "scale" that this one point represents becomes much more difficult to determine. Thus, the term "scale" can represent two different things for exposure assessment. When using modeling, the "scale" of the assessment area is simply the geographical land area around the sources within which modeling nodes will be placed and modeling will be done (for example, the model may predict ambient concentrations at every point on a 100 × 100 m grid out to 50 km in all directions from the sources). When assessors use monitoring to evaluate exposure, the "scale" refers to the area around the monitoring location (and the types of exposures) the analysts consider the monitoring data to represent (for example, a monitor located in an urban area that does not directly receive the impacted of an identifiable point source is usually designated as an "urban scale" monitor because it reflects general urban ambient air concentrations for populations not directly impacted by point sources). A full discussion of this distinction is provided in Chapter 9.

Scale can also refer more generally to the coverage of the analysis (see Exhibit 6-7). For example, the 1996 NATA risk characterization provided risk estimates, at the county level, for every county in the US. The "scale" of this analysis was nationwide. A real person, on the other hand, who was outfitted with a personal monitoring device, might be described as "personal" or "individual" scale.

6.3.3.3 Use of Modeling versus Monitoring

As this document has previously noted, risk assessors can base estimates of exposure concentrations on either actual measurements (i.e., monitoring data) or air quality modeling. Exhibit 6-8 provides a brief comparison of modeling and monitoring. Many studies may benefit by using some combination of modeling and monitoring, because the two approaches can complement one another.

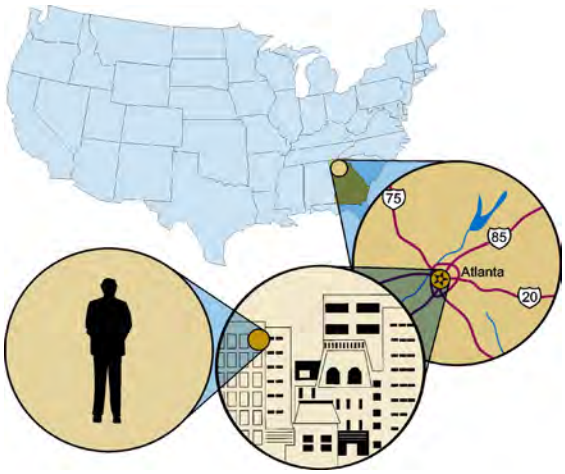
Benefits of modeling include the ability to:

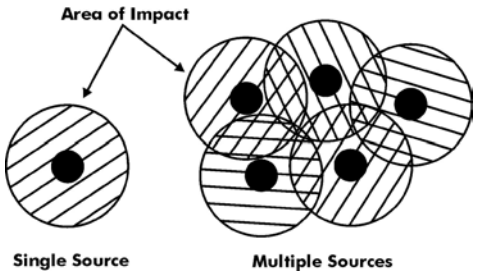
- Obtain a relatively quick, screening-level estimate of the potential for risk;
- Identify the subset of air toxics that contribute most significantly to the risk estimate;
- Identify the areas where the highest exposure concentrations are likely to occur;

- Estimate concentrations over a broad assessment area; and
- Examine individual variability in exposure.

One of the limits in the usefulness of modeling may be the accuracy of the air toxics emissions inventory (discussed in Chapter 7). Also, models can only provide estimates of exposure concentrations; often monitoring is performed to confirm model predictions.

Exhibit 6-7. Air Toxics Risk Assessments May be Conducted at Different Scales





Air toxics risk assessments may be performed on a variety of geographical levels ranging from the national level (e.g., the National-Scale Assessment), to the state, local, neighborhood, or even individual levels. Within a given scale, the risk assessment could look at the impact from a single source or multiple sources. The specific tools, approaches, and metrics used are likely to differ depending on the geographic scale of interest.

Benefits of monitoring include the ability to:

- Provide actual concentrations, which often provide a stronger basis for leveraging emissions reductions;
- Provide site-specific information to verify or calibrate model predictions;
- Provide time- and space-integrated measures of the actual concentrations at which individuals are exposed when they move from place to place within the assessment area; and
- Measure episodic releases, which are otherwise difficult to measure and quantify and are not well addressed in emissions inventories.

One of the limits in the usefulness of monitoring may be the representativeness of the location(s) in which monitors are placed (i.e., if placed in the wrong locations, monitors can provide incorrect and misleading information about exposures). Also, monitoring may not always be an effective tool to link ambient concentrations to specific sources (if, for example, one is monitoring benzene in an urban environment).

Exhibit 6-8. Comparison of Modeling and Monitoring Approaches for Estimating Ambient Air Concentrations	
Modeling	**Monitoring**
Modeling is relatively fast and inexpensive. Many screening-level models can be run in spreadsheet formats and require relatively simple input parameters. Many dispersion models, along with technical reference manuals and other support documents, are available for free download from EPA's Support Center for Regulatory Air Models (SCRAM) website (http://www.epa.gov/ttn/scram/). Resources normally need to be expended to enhance the local air toxics emission inventories to make air toxics modeling more precise.	Monitoring takes time to build data, and there are methodological limits and logistical issues. How expensive monitoring is depends on what you are trying to do and how much you have to buy or pay for. Monitoring does not always require equipment purchase and some states and local areas already have equipment. Some less expensive monitoring techniques are now available (i.e., passive samplers).
Modeling results can estimate concentration over a large spatial area (e.g., a 50-km radius from a source) and can provide a "big picture" view of the assessment area. Modeling also allows for analysis of exposure concentration at multiple points throughout the assessment area. The downside of modeling, however, is that these are predicted concentrations.	Monitoring results provide actual measured concentrations. Multiple locations may be required to characterize concentration over an area, although GIS methods facilitate interpolation between locations. The downside is that the monitoring may not be very representative of a large geographic area.
Screening-level models can provide a predicted estimate of whether significant concentrations are likely. A simple screening analysis may be sufficient to make a risk management decision that no action is required.	Monitoring can be used to identify and measure exposures for specific individuals at a specific location of concern (e.g., a school). This data can provide a quick screen to determine whether more extensive monitoring is needed.
Models can be used to identify areas where maximum concentrations are likely to occur, and thus to focus efforts for additional tiers of the assessment. Uncertainties in model parameters, and the discrete division of the wind field used in models (often with only eight wind directions) can result in incorrect identification of the locations of maximal concentration.	Monitoring can identify areas and actual levels of exposures occurring at the monitoring sites. Monitoring can also be used to indicate the point of maximal exposure if the monitoring is designed for that purpose. The selection of the monitoring locations is critical; if placed in the wrong locations, monitors can provide incorrect and misleading information about maximal exposures.
Models can be used to identify the subset of COPC and exposure pathways/routes that have the greatest contribution to risk. This can be helpful in focusing efforts for additional tiers of the assessment as well as determining appropriate risk management actions.	Monitoring can be used to confirm significant exposure pathways and routes. (Measured concentrations can be compared to risk-based screening levels.) It also can be used to identify compounds that may not have been suspected and, hence, were not included in models (i.e., monitoring allows identification of gaps in the emissions inventory).
Models allow "what if" scenarios to be evaluated (e.g., what if a permitted emission were doubled?).	Monitoring can only evaluate current conditions.
More complex modeling may allow explicit prediction and estimate of variability in exposure.	A large number of samples generally is needed to characterize variability; this may be prohibitively expensive. Monitoring, however, provides a direct and reliable means to characterize variability.
Models often use simplifying assumptions and data inputs that may or may not be representative of the specific assessment area. This introduces uncertainty into model predictions.	Monitoring can be used to confirm actual exposure levels as well as investigate assumptions or calibrate models to site-specific conditions, and to close gaps in data, reducing uncertainties.

6.3.3.4 Estimation of Exposure

An important element of the analysis plan is the specific approaches for developing numerical estimates of exposure concentrations for each of the COPC for each of the populations the assessment is studying (i.e., how exposure will be estimated and quantified). As noted in the previous subsection, this may involve the use of air quality models and/or monitoring data. Quantitation of exposure includes three general steps:

- **Characterization of releases to the air**. Characterizing the location, nature, and magnitude of emissions released from the sources being evaluated, including release parameters such as stack height and temperature of release (when modeling is being performed). This is discussed Chapter 7.

- **Estimation of chemical fate and transport**. Modeling and/or measuring the ambient concentrations of air toxics in the environment, as a result of transport, and including any physical or chemical transformations that may occur during this movement, from the emission point to the exposure points. This is discussed in Chapters 8, 9, and 10.

- **Estimation of exposure concentrations**. Developing a numerical estimate of exposure concentrations of air toxics to the selected exposure points. This is discussed in Chapter 11.

The Metrics of Exposure for Inhalation

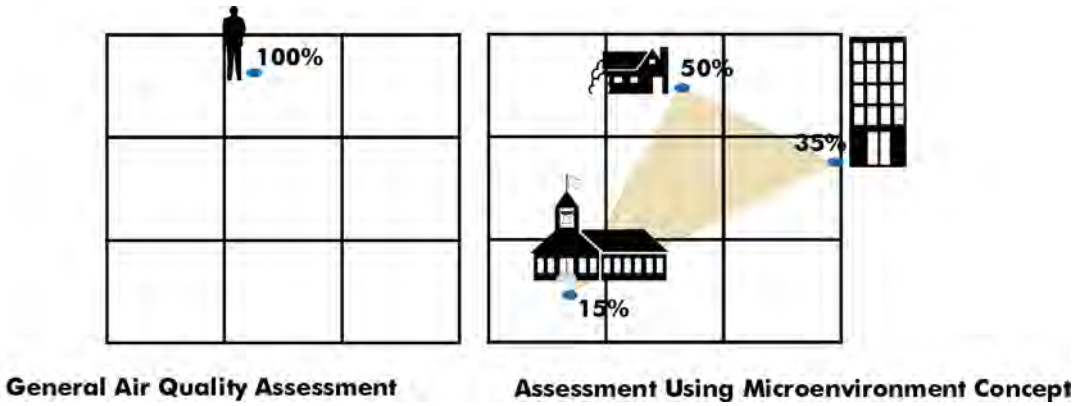
The metric of exposure for inhalation is simply the **exposure concentration (EC)** – the concentration of a chemical in the air at the point where a person breathes the air.

For the inhalation route of exposure, the metric of exposure is the concentration of the chemical in the air the population of interest is breathing over the period of interest. This concentration is called the **exposure concentration (EC)** and is the primary quantitative output of the inhalation exposure assessment. As we will see in Chapter 11, this metric is intended to represent the time weighted average exposure(s) to the population(s) of interest during the exposure period. (Note that exposure models are often also applied to better reflect how different people interact with contaminated air. In other words, the air quality model evaluates how chemicals move and change in the environment. The exposure model evaluates how different types of people interact with the resulting contaminated air - with the result that the EC is refined to provide more realistic estimates of exposure. A discussion of exposure modeling is provided below.)

There are two general ways to estimate the EC (Exhibit 6-9); these are discussed in greater detail in Chapter 11.

- **Ambient Air Concentrations**. For screening-level evaluations, assessors use the concentration of air toxics generated at each modeling node (or interpolated nodes) or the concentration determined by a monitor. The default assumption in such a screening assessment is that the population of interest is breathing air continuously around-the-clock at the modeled or monitor location. Proceeding in this manner, in the initial stages, is often done because of the additional cost, time, and specialized expertise needed to run the exposure model. Such results, depending on the purpose of the analysis, may be sufficient for some risk management decisions (Chapter 3 provides a discussion on how to phase or "tier" a risk assessment from simple but conservative to more complex yet realistic.)

Exhibit 6-9. Two General Ways to Estimate Inhalation Exposure Concentration

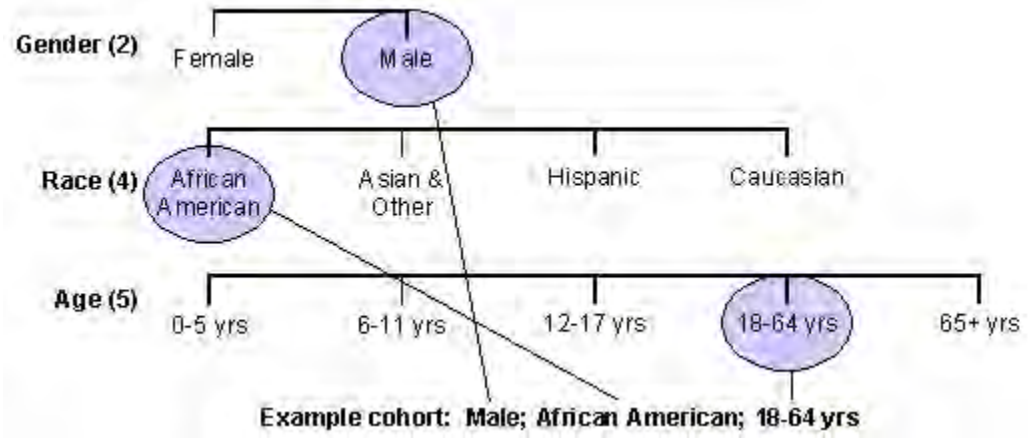


The left-hand side illustrates the use of ambient air concentrations as a surrogate for the EC. In this example, the analysis assumes that individuals spend 100 percent of their time at a given location, so the estimate of ambient concentration thus represents the EC. The right-hand side illustrates the use of exposure modeling. In this example, the analysis assumes that an individual spends 50 percent of his/her time at home; 15 percent at a school; and 35 percent at an office. The EC is the weighted sum of the product of the ambient concentrations at each location and the amount of time spent there. Both indoor and outdoor concentrations usually are considered at each location.

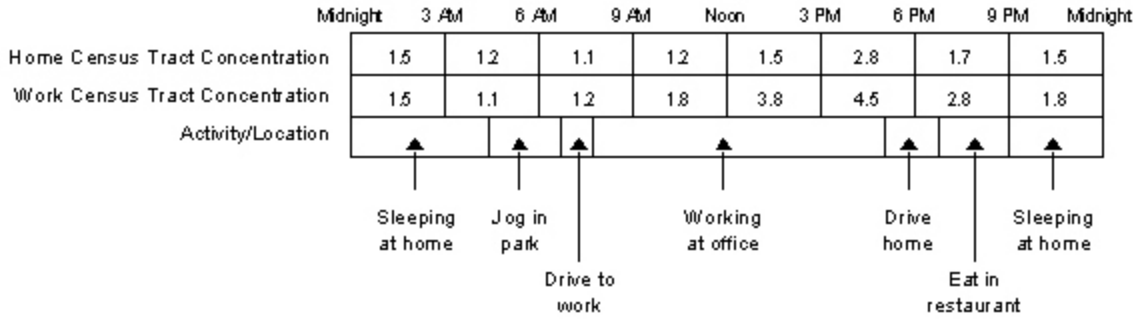
- **Exposure modeling**. More comprehensive inhalation exposure assessments combine estimates of ambient outdoor pollutant concentration (e.g., from air quality models) with information about the population of interest, including the types of people present (e.g., ethnicity, age, sex), time spent in different microenvironments, and microenvironment concentrations. The assessment objective is to obtain a representative estimate of the pollutant concentration in the inhaled air in each microenvironment. For risk assessments focusing on chronic effects resulting from chronic exposures, a long-term estimate of exposure is the EC of interest. As discussed in Chapter 9, the resulting estimate is a refined metric of personal exposure concentration (EC). This EC reflects the time spent in different microenvironments (and the activities within these microenvironments) throughout the daily routine of either representative individuals (selected statistically to be representative of the potentially exposed population) or different groups of people with similar attributes (called **cohorts**). The EC is essentially a time-weighted average exposure concentration for all of the cohorts combined (see Exhibit 6-10).

People living in the vicinity of one or multiple air toxics sources have the potential to receive exposure to emitted chemicals many different ways. For example, they might be exposed occasionally, but to very high concentrations (e.g., when an accident occurs that releases large amounts of chemical to the air in a very short amount of time). On the other hand, they might receive exposure quite often (or even continuously) to low levels that would likely go unnoticed. Air toxics inhalation exposure assessments usually focus on two of these different types of possible exposure scenarios:

Exhibit 6-10. Example Cohort Group



In this hypothetical example, cohort groups are defined based on gender (two categories); race (four categories), and age (five categories). This example illustrates an African American male aged 18-64 years.



In this hypothetical example, daily exposure scenarios are developed based on ambient air concentrations at work, and indoor and outdoor concentrations are assumed (for this example) to be equal at a given location, and home and the specific activity patterns modeled for each cohort. In this example, the African American male aged 18-64 years divides his activities among sleeping at home, jogging in the park, driving to work, working at the office, driving home, and eating at a restaurant. The daily exposure concentration is obtained by multiplying the time in each activity by the appropriate ambient air concentration(s) for the time period(s) of interest, then summing the products. For example, the product for jogging would be 1.2 (home concentration 3-6 AM) × 1.5 hours jogging (during the 3-6 AM time period) + 1.1 (home concentration 6-9 AM) × 0.5 hours jogging (during the time period 6-9 AM).

- **Chronic exposure** refers to situations in which the exposure occurs repeatedly over a long period of time (usually years to lifetime). If there is substantial variation in exposure concentration during segments of the chronic period, it may be appropriate to evaluate the segments separately using the appropriate dose-response values.

- **Sub-chronic exposure** refers to situations in which the exposure occurs repeatedly over a period of time that ranges between acute and chronic exposures (As toxicity values are less widely available for this duration, it is less routinely assessed than the others. For air toxics assessments, this exposure period is not commonly assessed.)

- **Acute exposure** refers to situations in which the exposure occurs over a short period of time (usually minutes, hours, or a day) and usually at relatively high concentrations. The averaging times commonly used to represent acute exposures concentrations (i.e., acute ECs) are a 24-hour average, a one-hour average, or a 15-minute average.

The EC values the assessor develops to represent acute and chronic exposures should match the assumptions built into the dose-response values that the assessor uses to characterize risk (see Chapter 12). For example, it would be inappropriate to compare a one-week average exposure concentration to a one-hour acute dose-response value. For chronic exposures, the scale of time-weighted averaging performed to develop the exposure estimate should be generally similar to that used in developing the dose-response value. For example, inhalation chronic RfCs are derived from studies involving regularly repeated exposures (e.g., six hours a day, five days a week in animal studies) over a chronic period. Thus, exposures occurring on a much lesser frequency (e.g., a several days a week on a handful of occasions during a couple of years), should not be averaged over the exposure period and compared to a chronic RfC. Such very infrequent exposures may be more appropriately assessed as separate shorter-term or sub-chronic exposures.

6.3.3.5 Evaluation of Uncertainty

This part of the analysis plan specifies the approach to be used to evaluate uncertainty in the exposure and risk estimates. Decision-makers will weigh the importance of the exposure (and resulting risk) estimates in the eventual decision in the context of the uncertainties inherent in these estimates. Assessment and presentation of uncertainty is discussed in Chapter 3.

6.3.3.6 Preparation of Documentation

This part of the analysis plan specifies the approach to be used to document all aspects of the risk assessment. For most individual air toxics risk assessments, the exposure assessment represents the majority of effort (and the majority of the documentation) and therefore may require the greatest amount of work. A comprehensive documentation of the methods, assumptions, and uncertainties associated with the exposure assessment is encouraged. Chapter 13 discusses documentation in greater detail.

6.3.4 Identification of the Exposed Population

This part of the analysis plan specifies the approach to be used to characterize the location and size of the populations of interest to the assessment. Additional information on population characteristics may assist in characterizing exposure, and in identifying sensitive sub-populations.

- **Population data**. In identifying and also characterizing a potentially exposed population, the U.S. Census Bureau (www.census.gov) is the primary source of population information (e.g., the most recent data on the US population is contained in the 2000 Census).

- **Sensitive sub-populations**. Human exposure and susceptibility and sensitivity to pollutant effects may vary with factors such as age, gender, intensity and amount of activity, time spent in microenvironments, diet, overall health, lifestyle, genetic factors, and the concentration of pollutant. The extent to which these factors are considered in the risk assessment depends on the purpose of the assessment as defined in the planning/scoping and problem formulation steps, available resources, uncertainties in the assessment, and data quality and quantity.

6.3.5 Identification of the Endpoints and Metrics

This part of the analysis plan specifies which human health endpoints will be evaluated in the risk assessment and the metrics by which they will be evaluated. For inhalation exposures, EPA generally evaluates individual cancer risk and noncancer hazard (see Chapter 12 for a more detailed discussion).

- Estimated individual cancer risk is generally expressed as a numerical probability that a person will develop cancer over the course of their lifetime as a result of the exposures under study.

- Noncancer effects are generally evaluated by comparing exposure concentrations to reference concentrations (RfCs), which are estimates (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious non-cancer effects during a lifetime. Noncancer effects generally are assessed for both acute and chronic exposure times.

Risk is usually described as either the risk experienced by different individuals within a population or the risk experienced by groups of people. The former is called risk to an individual (or simply individual risk), and the latter is called risk to a population (or simply population risk). The difference between the two is that individual risk describes risk to one person at a time, while population risk generally describes the number of people in a population experiencing the same risk. Thus, in a city block containing 400 people with an estimated risk (calculated at the block internal point) of two in 10,000 (2×10^{-4}), one could describe the risk to each of the individual 400 people as "individual risk = 2×10^{-4}." Alternatively the population risk could be described as "400 people living at a risk of 2×10^{-4}." While this distinction may seem arbitrary, risk often varies substantially over the exposed population. The use of both types of risk estimates assists risk managers in balancing concerns of small numbers of highly exposed people and larger numbers of people with lower exposures.

It generally is preferable to present a range of risk estimates, particularly in higher-tier assessments. Distributions are often more useful than point estimates. However, since developing fully distributional estimates of risk is usually out of the scope of most risk assessments, a sense of the range of risks is usually provided by developing both central tendency and high end point estimates.

- **Central tendency** estimates are intended to give a characterization of risk for the typical individual in the population. This is usually either based on the arithmetic mean risk (average estimate) or the median risk (median estimate).

- **High end** estimates are intended to estimate the risk that is expected to occur in the upper range of the distribution (e.g., risk above about the 90th percentile of the population distribution).

Risk characterization is discussed in more detail in Chapter 13.

6.4 Data Quality in the Risk Assessment Process

All air toxics risk assessments involve some data collection (e.g., emissions inventories will be developed to support air quality modeling, and/or monitoring data will be collected). For data collection efforts, a central component to the analysis plan is data quality assurance. The credibility of the risk assessment depends in part on the quality of the data that it uses. EPA uses its Quality System to manage the quality of its environmental data collection, generation, and use. The EPA quality website (http://www.epa.gov/quality) is an excellent resource for quality-related information that assessors will want to become familiar with as they develop an analysis plan for a risk assessment project.

As part of its effort to develop an Agency-wide data quality program, EPA has developed a number of specific tools that have direct applicability in performing risk assessment projects, including:

- Data quality assessment;
- Systematic planning (and the Data Quality Objectives Process);
- Quality assurance project plans;
- Standard Operating Procedures;
- Technical Audits; and
- Verification and Validation.

The use of these tools will help in the development of enough high quality data to allow assessors to answer the assessment questions in a robust way. A brief discussion of each of these tools follows. More in-depth discussion of each of these tools can be found on EPA's Quality website.

- **Data Quality Assessment** helps assess the type, quantity, and quality of data. This assessment, in turn, helps to verify that assessors satisfy the planning objectives. A Quality Assurance Project Plan components and sample collection procedures help ensure that the data are suitable for its intended purpose. Data Quality Assessment is a five-step procedure for determining statistically whether or not a data set is suitable for its intended purpose. This assessment is a scientific and statistical evaluation of data to determine if it is of the type, quantity, and quality needed and may be performed either during a project to check the process of data collection or at the end of a project to check if objectives were met.

- **Systematic Planning** is necessary to define the type, quantity, and quality of data a decision maker needs before collecting or generating environmental data. The Data Quality Objectives Process is an example of a systematic planning process that assessors would use to translate a decision maker's aversion to decision error into a quantitative statement of data quality needed to support that decision. Data Quality Objectives are not required under EPA's quality system; however, EPA does require that a systematic planning process such as

the Data Quality Objectives Process be used for all EPA environmental data collection activities. EPA recommends using the Data Quality Objectives Process when decision-makers are using data to select between two opposing conditions, such as determining compliance with a standard.

- **Quality Assurance Project Plan (QAPP)** documents the planning, implementation, and assessment procedures for a particular project, as well as any specific quality assurance and quality control activities. It integrates all the technical and quality aspects of the project in order to provide a "blueprint" for obtaining the type and quality of environmental data and information needed for a specific decision or use. *Note*: **All work performed or funded by EPA that involves the acquisition of environmental data must have an approved QAPP.**

- **Standard Operating Procedures** are written documents that describe, in great detail, the routine procedures to be followed for a specific operation, analysis, or action. Consistent use of an approved Standard Operating Procedure ensures conformance with organizational practices, reduced work effort, reduction in error occurrences, and improved data comparability, credibility, and defensibility. Standard operating procedures also serve as resources for training and for ready reference and documentation of proper procedures.

- **Technical audits** are systematic and objective examinations of a program or project to determine whether environmental data collection activities and related results comply with the project's QAPP and other planning documents, are implemented effectively, and are suitable to achieve its data quality goals. Technical audits are not management assessments nor are they data verification/validation processes, which occur during the assessment phase of the project. Technical audits include readiness reviews, technical systems audits, surveillance, and performance evaluations.

- **Data verification and validation** is used to evaluate whether data has been generated according to specifications, satisfy acceptance criteria, and are appropriate and consistent with their intended use. Data verification is a systematic process for evaluating performance and compliance of a set of data when compared to a set of standards to ascertain its completeness, correctness, and consistency using the methods and criteria defined in the project documentation. Data validation follows the data verification process and uses information from the project documentation to ascertain the usability of the data in light of its measurement quality objectives and to ensure that results obtained are scientifically defensible.

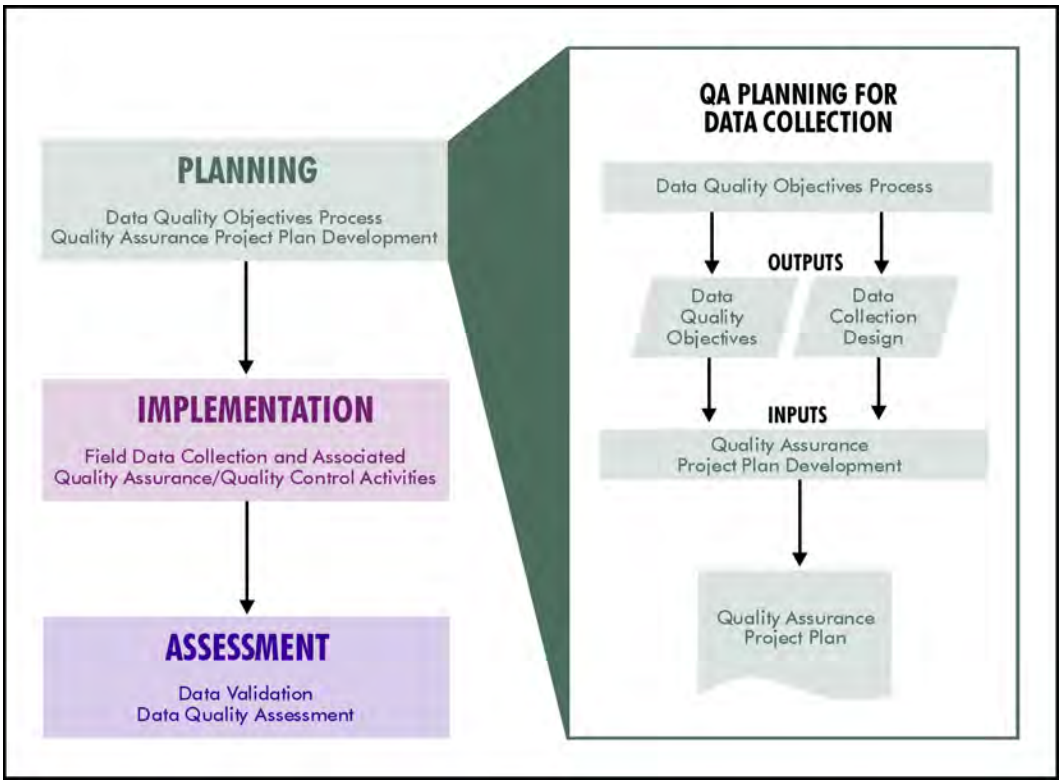
Quality Assurance is an integral part of data collection and analysis throughout the risk assessment project and the various activities addressed and documented in the QAPP cover the entire project life cycle, integrating elements of the planning, implementation, and assessment phases (Exhibit 6-11).

- **Planning**. The Data Quality Objectives (DQOs) are together a structured, systematic planning process that provides statements about the expectations and requirements of the data user (such as the decision maker).

- **Implementation**. The QAPP translates these requirements into measurement performance specifications and QA/QC procedures for the data suppliers to provide the information needed to satisfy the data user's needs.

- **Assessment**. The QAPP includes plans for data validation and data quality assessment.

Exhibit 6-11. QA Planning and the Data Life Cycle



References

1. U.S. Environmental Protection Agency. 2003. *Framework for Cumulative Risk Assessment.* Risk Assessment Forum, Washington, D.C., May 2003. EPA/630/P02/001F. Available at: http://cfpub.epa.gov/ncea/raf/recordisplay.cfm?deid=54944.

2. U.S. Environmental Protection Agency. 1995. *Land Use in the CERCLA Remedy Selection Process.* Office of Emergency and Remedial Response, Washington D.C., May 25, 1995. OSWER Directive No. 9355.7-04. Available at: http://www.epa.gov/superfund/resources/landuse.htm.

Chapter 7 Quantification of Exposure: Development of the Emissions Inventory for the Inhalation Risk Assessment

Table of Contents

7.1 Introduction 1

7.2 Process for Developing an Emissions Inventory 1
- 7.2.1 Planning 2
- 7.2.2 Gathering Information 3
- 7.2.3 Estimating Emissions 3
  - 7.2.3.1 Direct Measurement 5
  - 7.2.3.2 Emission Estimation Models 5
  - 7.2.3.3 Emission Factors 8
  - 7.2.3.4 Mass Balance 9
  - 7.2.3.5 Engineering Judgment 9
- 7.2.4 Compiling Data Into a Database 9
  - 7.2.4.1 Selection of Production Rates 9
  - 7.2.4.2 Unusual Conditions: Process Upsets, Accidental Releases, and Maintenance 11
- 7.2.5 Data Augmentation 12
- 7.2.6 Quality Assurance/Quality Control 13
- 7.2.7 Documentation 14
- 7.2.8 Access to Data 14

7.3 Data Sources 14
- 7.3.1 Permit Files 15
- 7.3.2 Regional Inventories 15
- 7.3.3 Industry Profiles 16
- 7.3.4 AP-42 Emissions Factors 17
- 7.3.5 Factor Information Retrieval System 17
- 7.3.6 Locating and Estimating Documents 18
- 7.3.7 RCRAInfo 18
- 7.3.8 Emissions and Dispersion Modeling System (EDMS) 19
- 7.3.9 Summary 19

References 21

7.1 Introduction

An emission inventory is a comprehensive listing, by source, of the air pollutant emissions within a specific geographic area in a specific time period. EPA prepares a **National Emissions Inventory (NEI)** with input from numerous state, local, and tribal (S/L/T) air agencies (see Chapter 4 and http://www.epa.gov/ttn/chief/net/index.html for more information on the NEI). NEI data are used for air quality modeling, regional strategy development, regulation setting, air toxics risk assessment, and tracking trends in emissions over time. The NEI Input Format (NIF) is the format most widely used by S/L/T agencies to transfer data to the NEI. The current versions of the NIF and all user documentation are available on the website noted above. The advantages, disadvantages, and uncertainties associated with NEI data are discussed in Chapter 4.

Emission inventories generally serve as the first step in quantifying exposure for an air toxics risk assessment. In addition to source information (e.g., location, chemicals released), they provide most of the critical input data for air quality models used to predict air toxics fate and transport in the atmosphere. The Emission Inventory Improvement Program (EIIP)[1] has published a ten-volume set of technical reports on the development of emissions inventories for the NEI.[2] Related technical documents, updates, reports, and information regarding the organization and progress of EIIP in developing new methods can be found linked to the main EIIP webpage. These include training manuals that provide in-depth descriptions of each step of the process, for various types of sources and air pollutants. Emissions inventories that are prepared in a manner consistent with these methods and guidance will provide data of known quality for a risk assessment.

For risk assessments, local enhancements of existing air toxics emissions inventories may be advantageous to a particular air toxics assessment effort as a very critical initial step. Air toxics inventories are not always at the quality that would provide the results desired in a modeling assessment, and improving the entire statewide toxics inventory may be unrealistic. An enhancement of the local air toxics inventory in the assessment area of interest may be beneficial for providing more accurate and precise risk assessment results and, consequently, a better basis for any air toxics risk- or airshed-program management decisions. Also, local emissions inventory work in specific areas of concern or study makes these air toxics efforts smaller and easier for agencies and participating facilities to manage and conduct, particularly in the shorter time frames commonly sought in local air toxics assessment projects.

The remainder of this chapter describes a process that can be used to develop an emissions inventory, including the general steps for developing an emissions inventory (Section 7.2), and data sources (Section 7.3)

7.2 Process for Developing an Emissions Inventory

There are eight steps for developing an emissions inventory:[3] (1) planning; (2) gathering information; (3) estimating emissions; (4) compiling data into a database; (5) data augmentation; (6) quality control/quality assurance; (7) documentation; and (8) access to data. Each is described in a separate subsection below.

The Emissions Inventory Improvement Program (EIIP)

To develop a systematic method for preparing an emission inventory, EPA's Emission Factor and Inventory Group (EFIG) has worked as a key member of the EIIP. The EIIP is a jointly sponsored effort of the State and Territorial Air Pollution Program Administrators/Association of Local Air Pollution Control Officials (STAPPA/ALAPCO) and EPA. Both of these organizations are represented in the Standing Air Emissions Work Group (SAEWG), which endorsed the original EIIP plan. Funding is provided by S/L/T agencies through the Federal 105 grant programs. While EPA coordinates the EIIP efforts, all of the tasks are performed by working committees. The EIIP Steering Committee and technical committees are composed of S/L/T, industry, and EPA representatives. Membership on technical committees is open to any S/L/T agency representative, industry group, and the public; interested individuals can contact the appropriate committee co-chair for information.

7.2.1 Planning

Planning is the first stage in preparing an emissions inventory. Perhaps the most important activity in compiling an inventory, planning ensures a focused and streamlined process and avoids later costly and embarrassing mistakes. The Inventory Preparation Plan (IPP) is developed during the planning stage and is the overarching guidance document for the entire emission inventory development process.[4] First, the IPP identifies the end-use(s) of the inventory (e.g., to support a risk assessment) and subsequently, an acceptable data quality level for those uses. Once the end-use(s) are determined, the risk manager defines the inventory to be created, identifying the necessary components:

- The air toxics to be carried through the risk assessment (i.e., the COPCs);
- The specific sources or source categories to be assessed;
- The geographic area (scale) of the assessment area; and
- The time interval over which emissions are to be inventoried.

Generally, the IPP reflects the complexity of the risk assessment being conducted. That is, an assessment of a single stationary source with known pollutants and well-documented emissions would not require as elaborate a plan as would a risk assessment addressing multiple sources and source types affecting a broad community. Exhibit 7-1 lists the steps in developing an IPP.

Exhibit 7-1. Steps in Developing an Inventory Preparation Plan
• Identify the end-uses of the inventory • Determine Data Quality Objectives • Define the inventory to be created • Select an inventory data management and reporting system • Summarize data reporting and documentation • Establish QA/QC procedures • Determine staffing and resource requirements • Develop a schedule • Identify partners and develop a communication plan
Source: Pope, A. *Inventory Preparation for Toxics*.[3]

The level of precision for emissions data required may differ among different tiers of analysis. For example, screening-level risk assessments often incorporate conservative assumptions (e.g., all sources are co-located, all emissions are of the most toxic species of a particular chemical) in order to minimize the time and effort required to develop the emissions inventory. If the screening-level analysis indicates that there is a potential for a risk, then additional effort is made to characterize sources and emissions with greater precision. The IPP for a given risk assessment will identify the requisite level of precision for each potential tier of analysis.

7.2.2 Gathering Information

The next step in the development of an emission inventory is to gather the relevant information from existing sources. The information gathered should, at a minimum, include applicable pollutants, their sources, and emissions data (e.g., chemicals, emissions rates over time). If air quality modeling will be a part of the exposure assessment, the emissions inventory will need to include all of the source term data required by the model(s) to be used (e.g., latitude and longitude coordinates for each source, building size and shape for assessing downwash, chemical speciation).

A comprehensive information search may include guidance documents, existing emissions data, preliminary screening studies, emission factors, models, source characterization documents, and activity data references. A good starting point in this search is EPA's *Handbook for Air Toxics Emission Inventory Development*.[5]

7.2.3 Estimating Emissions

After gathering data from existing information sources, the analyst estimates the emissions to be reported in the inventory. There are two main approaches for estimating emissions: the top-down approach and the bottom-up approach.

- In the **top-down approach**, national- or regional data are allocated to a state or county based on a surrogate parameter such as population or employment in a specific sector. This approach typically is used for nonpoint sources when: (1) local data are not available, (2) the cost to gather local information is prohibitive, or (3) the end-use of the data does not justify the required cost. The top-down approach requires minimum resources, but at the expense of emissions accuracy.

- In the **bottom-up approach**, the inventory is developed from site-specific information on emissions sources, activity levels, and emission factors. This approach, typically used for point sources, requires more resources, but results in more accurate estimates than the top-down approach.

Exhibit 7-2 compares several methods for estimating point source emissions. Available methods for nonpoint sources include material balance, emissions factors, emissions estimation models (all listed in Exhibit 7-2), and surveys and questionnaires. Mobile source emissions estimates come from models, such as EPA's NONROAD[6] model for nonroad mobile sources (construction equipment, lawn mowers, airplanes, trains, and others) and MOBILE[7] for on-road mobile sources (automobiles, trucks). Section 7.3 below provides additional information on potential sources of emissions data.

Exhibit 7-2. Point Source Emission Estimation Methods		
Method	**Advantages**	**Disadvantages**
Continuous Emission Monitors (CEM)	• Measures actual emissions • Can be used to estimate emissions for different operating periods • Considered high quality data	• Often cost prohibitive
Source (Manual Stack) Testing	• Yields more accurate estimates than Emission Factors or Material Balance • Data can be used to develop emission factors • Data can be extrapolated to other representative (nonpoint) emission sources	• Cost prohibitive (especially if large number of pollutants to be tested) • Uncertainty issues due to representativeness of estimates over time • There may be no standardized testing reference methods
Material Balance	• Useful when other developed methods are not available or practical • Useful for sources resulting in evaporative losses	• Must have specific knowledge of all process parameters (amount of material entering and leaving the process, amount of material packaged as product itself)
Fuel Analysis	• Useful when other developed methods are not available or practical	• Estimates not as accurate due to inherent uncertainties in input parameters
Emission Estimation Models	• Useful for complex calculations	• Estimates not as accurate due to inherent uncertainties in input parameters
Emissions Factors	• Ease of availability	• Uncertain accuracy
Engineering Judgment	• Useful as a last resort when no other methods generate accurate emission estimates	• Estimates based on individual judgment and therefore not as defensible as more developed methods

Determining the best method for estimating emissions requires a trade-off between cost and the accuracy of results obtained. When estimating emissions, it is important to consider:

- Intended end-use of the inventory (as described in the IPP);
- Availability of data of the specified quality (preliminary screening can be helpful here);
- Practicality of the method for the specific source category;
- Source category priority; and
- Resources (time, staffing, funding) available to prepare the inventory.

The Emissions Inventory Improvement Program (EIIP) series of documents provides further guidance in choosing the most appropriate method for the specific inventory's needs.[1]

7.2.3.1 Direct Measurement

Direct measurement of source-specific emission rates is relatively infrequent except for certain permitted facilities with specific monitoring requirements written into their permits. For example, source monitoring is typically available for large point source releases at facilities covered under the Title IV emissions tracking system associated with the acid rain control program. Various state and local permitting programs that may also require intermittent or continuous monitoring, depending on the nature of the process.

In some instances, source testing is required as part of the process of obtaining a permit. For example, a hazardous waste incinerator must do stack testing during trial burns to ensure that the incineration units and air pollution control equipment meet the limits established in the permit before full operation is allowed to begin. Subsequent to full operation, the facility will usually be required to perform continuous monitoring of stack emissions to ensure continued compliance.

EPA's **Emission Measurement Center (EMC)** provides linkages to available source monitoring methodologies in five general categories (Exhibit 7-3).

7.2.3.2 Emission Estimation Models

Specific emission measurements are generally the best and most accurate method to quantify emissions; however, source data are not always available and/or practical to obtain. As an alternative, emission estimation software and accompanying models may be used to generate emissions data. Emission estimation models are used when a large number of complex calculations must be undertaken in order to estimate a given emission or when a combination of parameters has been identified that affect emissions but individually do not provide a direct correlation. EPA provides a variety of approved models that can be used to determine point, nonpoint, and mobile source emissions based on a variety of known input parameters. Some of these emission estimation models are discussed below.

CHEMDAT8

CHEMDAT8 is a Lotus®1-2-3 spreadsheet prepared by EPA's Emissions Standards Division that includes analytical models for estimating VOCs from treatment, storage, and disposal facility processes. The models cover releases from disposal impoundments, closed landfills, land treatment facilities, and aeration and nonaeration impoundment processes. Additional information is available for download from the CHIEF software index website at: http://www.epa.gov/ttn/chief/software/index.html.

Exhibit 7-3. Categories of Source Monitoring Methodologies
EPA has established the EMC (http://www.epa.gov/ttn/emc/tmethods.html), as part of its Technology Transfer Network, which is a collection of technical internet sites containing information about many areas of air pollution science, technology, regulation, measurement, and prevention. The EMC identifies five general categories of source monitoring methods: • **Category A: Methods Proposed or Promulgated in the Federal Register**. These methods have been proposed or promulgated in the Federal Register and codified in the Code of Federal Regulations (CFR). • **Category B: Source Category Approved Alternative Methods**. These methods are approved alternatives to the methods required by 40 CFR Parts 60, 61, and 63 as described by the General Provisions of the corresponding Parts. • **Category C: Conditional Methods**. EPA has evaluated these methods, and they may be applicable to one or more categories of stationary sources. EPA confidence for these methods is based upon review of various technical information including, but not limited to, field and laboratory validation studies, EPA understanding of the most significant quality assurance (QA) and quality control (QC) issues, and EPA confirmation that the method addresses these QA/QC issues sufficiently to identify when the method may not be acquiring representative data. The method's QA/QC procedures are required as a condition of applicability. • **Category D: Preliminary Methods**. The performance of these methods is not as well defined as that of the conditional methods of Category C. EPA is providing these as they may be useful in limited applications until more supporting information is available (i.e., can be '"gap filling'" methods). EPA expects the methods to work under the conditions of the applicability statement but is uncertain of the methods' applicability without additional data on broader application. EPA encourages submission of data to support broader applicability. • **Category E: "Idea box."** The idea box includes method concepts intended to promote information exchange only, and the concepts may not be used by sources to fulfill Federal requirements. These technical ideas have been provided to EPA for posting on the EMC web site. Concepts in the idea box generally have had little or no EPA review or analysis and are not technically supported by EPA. However, information that resides here may be considered for further assessment by EPA and non-EPA entities for the purposes of method development for placement into higher categories.

WATER9

WATER9 is a Windows-based computer program available for estimating air emissions of individual waste constituents in wastewater collection, storage, treatment, and disposal facilities. It also contains a database listing many of the organic compounds and describes procedures for obtaining reports of constituent fates, including air emissions and treatment effectiveness. WATER9 is a significant upgrade of features previously contained in WATER8, CHEM9 (a compound properties processor that can estimate compound properties that are not found in EPA's database of over 1000 compounds), and CHEMDAT8, and contains a set of models that can provide a holistic picture of emissions from a facility. The models produce emission estimates for each individual compound that is identified as a constituent of the wastes leaving

the facility based on the physical/chemical properties of the compound and its concentration in the wastes. Therefore, the analyst should be able to identify the constituent compounds and provide their respective concentrations. WATER9 has the ability to use site-specific compound property information and the ability to estimate missing compound property values. Estimates of the total air emissions from the wastes are obtained by summing the estimates for individual compounds. Program software may be downloaded from http://www.epa.gov/ttn/chief/software/water/index.html.

Landfill Gas Emissions Model v2.01

The Landfill Gas Emissions Model is a program specifically designed for use by state and local regulatory agencies to monitor the air emissions from landfills. The system allows the user to enter specific information regarding the characteristics and capacity of a landfill and to project the emissions of methane, carbon monoxide, nonmethane organic compounds, and individual HAPs over time using the Scholl Canyon decay model for landfill gas production estimation. The Scholl Canyon Model is a first-order decay equation that uses site-specific characteristics for estimating the gas generation rate. In the absence of site-specific data, the program provides default values for regulatory uses of the model and provides default values drawn from EPA's *Compilation of Air Pollutant Emission Factors* (AP-42) for inventory uses. For additional information, contact EPA's Air Pollution Prevention and Control Division, Office of Research and Development at (919) 541-2709. Program software may be downloaded from http://www.epa.gov/ttn/chief/software/index.html.

TANKS

TANKS is a Windows-based computer software program that estimates emissions of volatile organic compounds (VOCs) and hazardous air pollutants (HAPs) from fixed- and floating-roof storage tanks and is designed for use by S/L/T and Federal agencies, environmental consultants, and others who need to calculate air pollutant emissions from organic liquid storage tanks. The calculations are performed according to estimation procedures outlined in EPA's *Compilation of Air Pollutant Emission Factors* (AP-42). The user provides specific information concerning the storage tank and its contents, and the program then estimates annual or seasonal emissions and produces a report. The tank contents can consist of single or multiple liquid components. The program may be downloaded from http://www.epa.gov/ttn/chief/software/tanks/index.html.

MOBILE6 Vehicle Emission Modeling Software

MOBILE6 is an emission factor model for predicting gram per mile emissions of hydrocarbons (HC), carbon monoxide (CO), nitrogen oxides (NO_x), carbon dioxide (CO_2), particulate matter (PM_{10} and $PM_{2.5}$), and other toxics from cars, trucks, and motorcycles under various onroad conditions. The program is available for download from http://www.epa.gov/otaq/m6.htm.

NONROAD Model

The Draft NONROAD Model is a Windows-based software program intended for use by professional mobile source modelers for their use in estimating emissions specifically for emissions inventory development. The model is still in draft form, so EPA warns that some

emission rates and activity levels predicted from NONROAD may substantially change in future versions. The program is available for download from http://www.epa.gov/otaq/nonrdmdl.htm.

Please note that EPA's Office of Transportation and Air Quality is currently developing a new modeling system, Multi-scale mOtor Vehicles and equipment Emission System (MOVES) that will replace the existing MOBILE6 and NONROAD models. This new system will estimate emissions for onroad and nonroad sources, cover a broad range of pollutants, and allow multiple scale analysis, from fine-scale analysis to national inventory estimation. For further information on MOVES, visit http://www.epa.gov/otaq/ngm.htm.

7.2.3.3 Emission Factors

Emission factors are constants that assessors can use to relate release rates to the amount of specific activities that occur at a source. An emission factor is typically represented as a mass of chemical released per unit of activity. For example, releases from a coal burning combustion device are represented as pounds of pollutant emitted per BTU coal burned. Depending on the emission source, there may be a lot of emissions testing data, just one or two measurements (the usual case), or none. For a screening-level assessment it may be possible to obtain an estimate of maximum emissions in one of several ways.

- If sufficient data are available, the assessment could use the highest available value.
- If only one or two measurements are available, the assessment could assume that all the emissions occur in a short period of time (such as only for 8 hours a day) and/or assume that all sources of emissions are co-located.
- If no data are available, the assessor may need to rely on professional judgment based on similar types of sources.

Certain types of sources (e.g., incinerators) typically undergo various test or trial "burns" to establish emissions factors pursuant to RCRA permitting requirements. Data to support the development of emissions factors also may be collected to support compliance with maximum achievable control technology (MACT) standards or Toxics Substances Control Act (TSCA) permitting. For stable and well established processes, the emission factors are usually reliable estimates. However, for sources that are subject to different operational conditions, with limited testing, the emission factors may represent an estimate of a higher or lower release rate.

Frequently, emission factors contain an associated confidence level by species, which assists in determining the appropriate emission factor. Thus, the use of the emission factor for any specific source may over- or under-predict actual release rates. In some cases, accurate measurements of the activity rates are not available and estimates of activity rates can also contribute uncertainty to the release rate estimate for any particular source type. An example is for individual motor vehicles; this source model estimates an average emission factor for a fleet of vehicles in a particular location. Modeling approaches for traffic activity estimate the total amount of miles driven by vehicle class. Finally, multiplying the emissions factor by the number of vehicle-miles driven produces the total emissions. Thus, any individual motor vehicle may have a release rate significantly far removed from the average, but when averaged across the fleet, the release rate provides a more reliable estimate.

EPA suggests emission factors for criteria pollutants and HAPs in its national database, Factor Information Retrieval System (FIRE), which includes emission factors from EPA documents (such as *Compilation of Air Pollutant Emission Factors* (AP-42) and the *Locating and Estimating Air Emission* series) factors derived from state-reported test data, and factors taken from literature searches. FIRE is available for download at http://www.epa.gov/ttn/chief/software/fire/.

7.2.3.4 Mass Balance

Assessors can use the mass balance approach in complex processes in which a known amount of air toxics material is introduced to a process, and at the end of the process, a known amount of air toxics material is still retained in the final product. The difference between the two represents the production release. Engineering estimates can then suggest into what medium the process released the air toxic (e.g., to air or water, or as solid waste).

As an example, consider the use of a VOC as a carrier medium for a solid (e.g., paint particles). In this surface coating situation, the organic solvent that suspends the solids makes application of the coating possible. Once the mixture is exposed to the air, the solvent evaporates, leaving the solid coating film on the object. Mass balance techniques in this type of application may assume that 100 percent of the solvent is released to the air through evaporation. Other mass balance estimates may assume that some stable amount of the solvent is retained in the product that is shipped to customers. Mass balance estimates also may need to consider how much of the solvent is recycled at various stages in its life-cycle.

7.2.3.5 Engineering Judgment

With engineering judgment, users can estimate emission releases through engineering and operational observations about a process. For example, if a certain process must be operated at a set temperature and pressure to achieve the ideal result, engineers who understand the history of the process can often estimate how the release rate actually varies under changing operational conditions. Engineering judgment is a less desirable approach for estimating releases than actual measurements; however, it is often used because of a lack of any better information or options (e.g., it may not be possible to measure all fugitive leaks at a large facility with thousands of joints and valves).

7.2.4 Compiling Data Into a Database

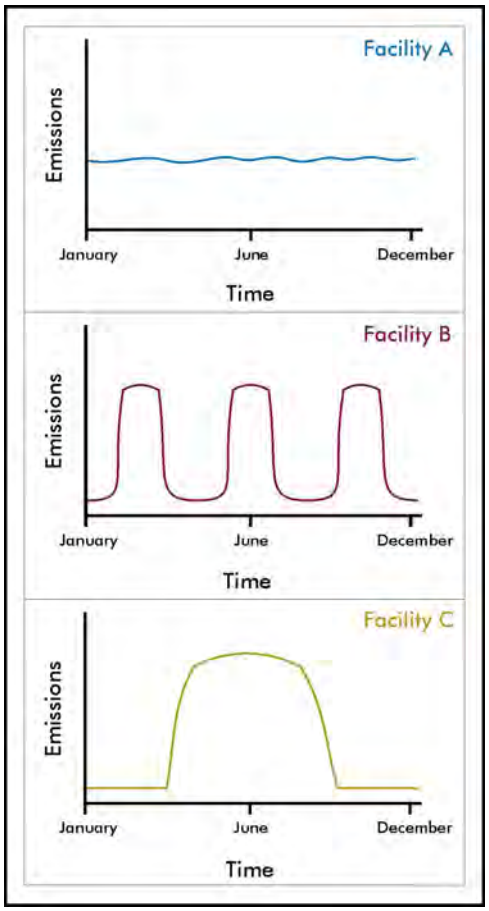
After estimating the applicable emissions from each source, the analyst compiles the data into the inventory database, based on the data management system delineated in the Inventory Preparation Plan. Three elements of data compilation are of note for a risk assessment: selection of production rates; unusual conditions; and how emissions are quantified for risk assessment purposes. Each is discussed in a separate subsection below.

7.2.4.1 Selection of Production Rates

The variability in a source's emissions rate can make it difficult to arrive at a single source-specific emissions level. Prior to collecting or reviewing data in support of a risk assessment, assessors will need to decide whether to use release data that reflects either annual average

emissions or "worst-case" operating conditions (or both). In some limited cases, it may be possible to obtain data on daily and seasonal variable emissions, although this is not common. Likewise, information on both the actual release rates and maximum permitted or allowable release or potential emission rate may be available. In addition to information on annual releases, a description of the release pattern over the year (see examples in Exhibit 7-4) and during the weeks of operation will be useful in characterizing the resultant ambient air concentrations over the exposure duration (e.g., is release occurring around-the-clock or only during the work week?).

Exhibit 7-4. Examples of Variable Patterns of Emissions



Emissions patterns may vary over time. In this hypothetical example, Facility A's emissions occur at a fairly constant rate. In contrast, Facility B's emissions levels fluctuate with spikes in their emissions evident at different times of the year. Facility C's emissions occur only during a specific part of the year.

Information on variability in operating conditions and the factors or conditions influencing that variability will be useful. This will assist in the selection of the release data for the scenario of interest in the assessment. For example, if the assessment is evaluating what may be released under current permit conditions, it may be appropriate to use release rate data corresponding to the maximum permitted release rate, regardless of reported actual rates. This method is best-suited for screening-level analyses, where the objective is to conduct a risk assessment for the purposes of screening out sources that pose negligible risk while efficiently conserving available resources (i.e., time and money), which may be needed for a more refined analysis of the remaining sources. However, use of reported release data (e.g., annual estimates) may be more appropriate for refined analyses of facilities with well-defined production capabilities and limited

operational variability. Note that more detailed information on variability may be needed for analysis of air concentrations (and resultant exposures) over shorter periods (e.g., acute analysis).

7.2.4.2 Unusual Conditions: Process Upsets, Accidental Releases, and Maintenance

Release characteristics frequently differ during atypical operations. Process upsets often result in the venting of large amounts of raw materials, intermediate products, finished products, or wastes to the air. Sources often flare organic releases during process upsets (i.e., burned as the chemicals are being released to the air) to reduce the mass of potentially toxic compounds, which can result in releases of particulate matter or other chemicals as a result of incomplete combustion. Shutting down and starting up processing equipment results in a period of time when the process is operating at less than ideal conditions, and release rates can change significantly during these shut down and start up procedures. Accidental releases associated with truck accidents, train derailments, or chemical spills from shipping operations can also cause significant releases that affect a local area for a short period of time.

Many operating permits require sources to report periods of process upset and maintenance activities, although assessors may not estimate release rates during these periods because measurement data are often unavailable. Local emergency authorities often possess information on accidental releases from trucking accidents and train derailments. These reports may be useful to provide modeling input data in subsequent acute risk assessment activities. Risk assessors can contact local HazMat Teams, and state or federal emergency response personnel to gather available information on accidental and upset releases. This type of information could be quite useful in local episodic acute risk analysis,[(a)] but may not be included in long-term risk evaluations.

Another source of startup/shutdown/malfunction data may be available in state/local/tribal permit files for the facilities. Specifically, many permitted facilities must file routine reports in which the provide information on spills, excursions, and other unusual circumstances where non-routine releases occur.

7.2.4.3 Quantifying Emissions for the Risk Assessment

Once the assessor has compiled the above information about the source in question, he or she would quantify the emission rate as the amount of pollutant released per unit of time. Most air toxics releases are expressed as tons of pollutants released per year in emissions inventories. However, as noted above, a yearly value may not provide the level of information required to evaluate the risk assessment questions, and more detailed information may be necessary. For example, are there seasonal fluctuations in emissions? Are the releases continuous around-the-clock, seven days a week, or more intermittent with a different schedule? The particular air dispersion model may require that emission rates be expressed in different units (e.g., pounds per

[a]EPA's Chemical Emergency Preparedness and Prevention Program web page describes the development of emergency management plans relying on such acute risk analysis to assist in the response to accidental releases: http://yosemite.epa.gov/oswer/ceppoweb.nsf/content/RMPoverview.htm

Process Upset and Accidental Release Information Sources

The **National Response Center (NRC)** has an on-line query system that provides access to all non-Privacy Act data collected by the NRC since 1990. This information may be accessed at http://www.nrc.uscg.mil/foia.html.

The U.S. Chemical Safety and Hazard Investigation Board maintains a database of **Incident News Reports**, which may contain information on process upsets and accidental releases. The Incident News Reports database is available at http://www.chemsafety.gov/circ/.

RMP*Info is a national database that provides information on risk management plans (RMPs). Each RMP contains a hazard assessment that includes an accident history covering the facility's previous five years of operation. This information can be obtained by submitting a written request for the RMP database (without the Offsite Consequence Analysis data) to the RMP Reporting Center, P.O. Box 1515, Lanham-Seabrook, Maryland 20703-1515.

hour, grams per second). The NEI contains emission estimates of HAPs for periods of a year or less. Risk assessors generally consult the emission period table in the NEI and use the emission type field to determine the period for which emissions are reported. The EPA summarizes NEI data in summary files to annual emissions, but more detail on the reporting period is available in the NEI. The NEI also contains emissions estimates for actual, allowable, potential, and maximum emissions for the same emission release points.

7.2.5 Data Augmentation

If previous efforts at estimating emissions fail to obtain data to assemble an emissions inventory of sufficient quality or to provide the necessary inputs for an emissions model, the next step would be data augmentation. The analyst first identifies any missing information, most notably emission data, vent parameters, and location coordinates.

- **Emissions data**. When developing emission inventories for nonpoint sources, analysts sometimes find that no direct measure of activity exists at the local level. In cases where this occurs, national, regional, or state-level emission estimates already in existence may be allocated to the local level (i.e., a top-down approach). This practice is know as **spatial allocation** and is a common form of data augmentation. Similarly, emissions can be temporally allocated to the time period required by an emissions model. Other suggestions for filling emission data gaps include:
  - Additional searches of databases to identify appropriate surrogate data;
  - Extrapolation of emissions from other geographic areas; and
  - Estimation of emissions data from past inventories within the same geographic area.

- **Vent parameters**. Common vent parameters required for air quality modeling include height, diameter, temperature, exit velocity, and flow rate. If measures for any of these parameters are missing or incomplete, the NEI provides default lookup tables generated from Source Category Classification (SCC) and Standard Industrial Classification (SIC) codes.

  SCC codes serve as a primary identifying data element in the NEI (as well as other EPA databases) and many S/L/T agency emissions data systems. These codes are assigned to

specific release points within a facility based on the process to which the release point is linked and on various characteristics of the release point. A complete listing of SCC codes along with additional background information is available from EPA.[8]

SIC codes are numerical codes developed by the U.S. government as a means of consistently classifying the primary business of business establishments. A list of the industry groups that are required to report to the Toxics Release Inventory (TRI) is provided in Chapter 4 and also can be found at http://www.epa.gov/tri/report/siccode.htm.

For facilities that are regulated pursuant to a National Emissions Standard for Hazardous Air Pollutants (NESHAP), the MACT codes, based on the MACT source category into which a specific process falls, may provide additional information about the nature of the business, primary production processes, or activities related to the release of air pollutants.

If no SCC or SIC code is available for the emission source in question, the analyst may use the national default values for each parameter (see Exhibit 7-5).

Exhibit 7-5. National Vent Parameter Default Values

Parameter	Default Value
Height	10 ft.
Diameter	1 ft.
Temperature	72° F
Velocity	15 ft./sec.
Flow Rate	12 $ft.^3$/sec.

Source: Pope, A. *Inventory Preparation for Toxics*.[3]

- **Location coordinates** may be identified from the NEI (stationary sources) or topographic maps (discussed in Chapter 6).

7.2.6 Quality Assurance/Quality Control

Quality assurance and quality control (QA/QC) procedures are vital to the validity of the emissions inventory and ensure that the modeling input parameters derived from the inventory are of specified quality. The quality assurance plan (QAP) for the emissions inventory is usually a part of the quality assurance project plan (QAPP) for the overall risk assessment that is developed during problem formulation (see Chapter 6). The QAP documents the procedures of the QA/QC elements of the emissions inventory. Quality control measures include:

- Technical reviews;
- Use of approved standardized procedures for emissions calculations;
- Data verification procedures;
- Completeness checks;
- Consistency checks;
- Accuracy checks; and
- Reasonableness tests.

To the extent practicable, risk assessors have emissions data verified by external review and audit procedures conducted by a third party. Exhibit 7-6 identifies the typical errors that occur in developing an emissions inventory.

Exhibit 7-6. Typical Errors in Developing an Emission Inventory		
Facility Errors • Missing facilities • Duplicate facilities • Closed facilities • Improper facility locations	**Data Errors** • Missing operating or technical data • Erroneous technical data • Errors in calculations • Data entry and transposition errors • Data coding errors • Failure to identify all HAPs	**Double-counting Errors** • Overlap between point and nonpoint sources • Overlap between nonpoint source categories

7.2.7 Documentation

Documentation is the next step in developing an emission inventory. The key documents to be compiled into a final written report include:

- Inventory Preparation Plan (IPP);
- Quality Assurance Plan (QAP);
- Methods;
- Assumptions;
- Raw data (database); and
- Calculations.

7.2.8 Access to Data

The risk manager generally ensures appropriate access to the data compiled in the emission inventory. A key part of the planning and scoping process for the risk assessment is determining who needs access to the emissions data and how they will access the data. If it is necessary to report the results of the emission inventory to the EPA as part of a S/L/T agency's responsibilities under the Consolidated Emission Reporting Rule, data preparation and submission procedures prepared by EPA for HAP data should be followed.

7.3 Data Sources

The two data sources for emissions inventory information that are most applicable to air toxics risk assessments are S/L/T agencies and the NEI. The TRI can provide some helpful information about the types of emissions from sources, but TRI data have not been collected to support air toxics risk assessments and therefore may be of limited value. The following subsections describe several other sources of information that may provide information to assist in developing an emissions inventory for the risk assessment.

7.3.1 Permit Files

Most stationary sources (especially large sources) are subject to one or more emissions limitation standards to control criteria emissions and/or HAP emissions. These sources are usually subject to a Title V operating permit that will include all of the operating and emissions limit requirements subject to that facility. In addition, they may be subject to additional S/L/T regulations. Operating permits require routine reporting to confirm that the operating conditions and emission limits are being met. Frequently, these reports are based on some kind of monitoring information that is directly related to the release process(es). In most cases, actual release rates are reported. Therefore, permit compliance reports represent an excellent source of information that will provide the actual release rate directly through continuous emissions monitoring, or they will provide sufficient information to estimate the release rates with a fairly high level of reliability.

Unfortunately, using the permit compliance system is not always an attractive source for data on release rates that are suitable for risk assessment activities. EPA does not maintain a central database of Title V permit or compliance information. Therefore, gathering data from the permit program can be a time-consuming task if many sources are needed for the risk analysis. The reports will also be represented in terms that match the requirements of the permit (e.g., if the permit conditions specify an annual release limit, release rates may be presented as an annual average; if the permit conditions specifies a maximum release rate for any hour, then the compliance report will document the maximum hourly rate observed at the facility). Therefore, some adjustments and assumptions may be necessary. In addition, the permit compliance report will only include the specific pollutants named in the regulations to which the permit applies. For example, for NESHAPs that are applicable to the source, only HAPs specifically listed in the rule may be included on the permit; other HAPs may also be emitted which are not required to be reported. Additionally, not all important HAP sources are required to have a Title V permit, and even small annual release rates of certain highly toxic HAPs may post significant risk.

In general, EPA has made an effort to include permit data in the NEI database (via data submissions from S/L/T offices) where appropriate and has taken steps to review the data. In many cases, it may be more reasonable to consult the NEI prior to attempting to gather release rates directly from permit files.

7.3.2 Regional Inventories

Several regional organizations provide emission data specific to their geographic area of concern. For example, the Great Lakes Commission (a partnership among EPA, the eight Great Lakes states, and the province of Ontario, Canada), with funding from EPA and the Great Lakes Protection Fund, have developed the Regional Air Pollutant Inventory Development System (RAPIDS). This ongoing initiative seeks to provide researchers and policy makers with detailed, basin-wide data on the source and emission levels of toxic contaminants. Originally focused on 49 toxic air pollutants, the inventory database has been expanded to include 82 toxic air pollutants which have been identified as significant contributors to the contamination of the Great Lakes. RAPIDS uses the FIRE database to estimate emissions for both point and nonpoint sources. The software may be downloaded from http://www.glc.org/air/rapids/.

Additionally, EPA provides funding to five regional planning organizations throughout the U.S. to address regional haze and visibility impairment issues. These organizations exist to evaluate technical information to better understand how their states and tribes impact national park and wilderness areas (Class I areas under the CAA) across the country and to then pursue the development of regional strategies to reduce emissions of particulate matter and other pollutants contributing to regional haze. To this end, each regional planning organization assesses its member states' emission inventories, and some provide funding through EPA for the development of regional emission inventories. Information regarding regional emission inventorying activities may be found at the organizations' respective websites as listed below:

- Central Regional Air Planning Assocation (CENRAP) – http://www.cenrap.org/
- Western Regional Air Partnership (WRAP) – http://www.wrapair.org/
- Midwest Regional Planning Organization (Midwest RPO) – http://64.27.125.175/
- Mid-Atlantic/Northeast Visibility Union (MANE - VU) – http://www.manevu.org/index.htm
- Visibility Improvement State and Tribal Association of the Southeast (VISTAS) – http://www.vistas-sesarm.org/

7.3.3 Industry Profiles

To help assessors understand the nature of releases from sources, EPA has compiled a variety of guidance documents and information resources that explain how various industries operate and the types and locations of emissions that commonly are associated with their processes. Two key groups of these documents are the "Sector Notebooks" and the TRI Facility Specific profile.

The sector notebooks are a series of profiles or notebooks containing information on selected major industries. These notebooks, which focus on key indicators that holistically present air, water, and land pollutant release data, have been thoroughly reviewed by experts from both inside and outside EPA. Each notebook provides:

- A comprehensive environmental profile;
- Industrial process information;
- Pollution prevention techniques;
- Pollutant release data;
- Regulatory requirements;
- Compliance/enforcement data;
- History government and industry partnerships;
- Innovative programs contact names;
- Bibliographic references; and
- Description of research methodology.

The notebooks cover a wide variety of activities, including:

- Agricultural chemical, pesticide and fertilizer industry;
- Dry cleaning industry;
- Ground transportation industry;
- Inorganic chemicals industry;
- Fossil fuel electric power generation industry;
- Metal fabrication industry; and

- Organic chemical industry.

Regarding TRI resources, the TRI website provides a number of very industry-specific and chemical-specific guidance documents that were developed to help stakeholders understand the nature of major industrial process and how emissions may occur from those processes (see http://www.epa.gov/tri/guide_docs/index.htm#industry_sp). Example titles include:

- Presswood and Laminated Products Industry;
- Coal Mining Facilities;
- Electricity Generating Facilities;
- Petroleum Terminals and Bulk Storage Facilities;
- Rubber and Plastics Manufacturing;
- Printing, Publishing, and Packaging Industry;
- Textile Processing Industry;
- Leather Tanning and Finishing Industry; and
- Semiconductor Industry.

Assessors can take advantage of these materials to help them better understand the nature of potential risk posed by facilities on local populations.

7.3.4 AP-42 Emissions Factors

Emission factors and emission inventories have long been fundamental tools for air quality modeling. The Emission Factor and Inventory Group (EFIG) in EPA's Office of Air Quality Planning and Standards (OAQPS) develops and maintains emission estimating tools. The AP-42 series is the principal means by which EFIG can document its emission factors. It is available from EPA online.[9] These factors are cited in numerous other EPA publications and electronic databases, but without the process details and supporting reference material provided in AP-42. Information about emission factors for mobile sources can be found on EPA's Office of Transportation and Air Quality website (http://www.epa.gov/otaq/).

So just what is an AP-42 Emission Factor? It is a representative value that attempts to relate the quantity of a pollutant released to the atmosphere with an activity associated with the release of that pollutant. These factors are usually expressed as the weight of pollutant divided by a unit weight, volume, distance, or duration of the activity emitting the pollutant (e.g., kilograms of particulate emitted per megagram of coal burned). Such factors facilitate estimation of emissions from various sources of air pollution. In most cases, these factors are simply averages of all available data of acceptable quality and are generally assumed to be representative of long-term averages for all facilities in the source category (i.e., a population average).

7.3.5 Factor Information Retrieval System

The Factor Information Retrieval (FIRE) Data System is a database containing EPA's recommended release rate estimation factors for criteria and hazardous air pollutants. FIRE 6.24 (released March 2004) is a Windows-based program. Users can browse through records in the database or select specific emission factors by source category, source classification code (SCC), pollutant name, chemical CAS number, or control device. FIRE 6.24 contains emission factors from the Compilation of Air Pollutant Emission Factors (AP-42 Fifth Edition) through March

2004, the Locating and Estimating (L&E) series of documents, and the retired AIRS/Facility Subsystem Emission Factors (AFSEF) and Air Toxic Emission Factor Database Management System (XATEF) documents. FIRE can be accessed at: http://www.epa.gov/ttn/chief/software/fire/index.html.

7.3.6 Locating and Estimating Documents

This report series characterizes some of the source categories for which releases of a toxic substance have been identified. These volumes include general descriptions of the emitting processes, identifying potential release points and emission factors. Some of the locating and estimating documents were prepared as early as 1984 and the information may be dated. Others have been developed since 1994 and will provide more up-to-date information (see http://www.epa.gov/ttn/chief/le/index.html). EPA does not maintain L&Es and has not published new L&Es since the mid 1990s.

7.3.7 RCRAInfo

RCRAInfo is EPA's comprehensive information system providing access to data supporting the Resource Conservation and Recovery Act (RCRA) of 1976 and the Hazardous and Solid Waste Amendments (HSWA) of 1984. The RCRA law is the primary statute under which EPA monitors and regulates the management of nonhazardous and hazardous solid waste by entities that produce, store, treat, transport, or otherwise manage such wastes (all of which are potential sources of air toxics emissions in a community). This RCRAInfo replaces the data recording and reporting abilities of the Resource Conservation and Recovery Information System (RCRIS) and the Biennial Reporting System (BRS).[10]

RCRIS was the national program management and inventory system of facilities that handle RCRA hazardous waste.[11] Facilities fit one or more of the following categories: treatment, storage, and disposal facilities (TSDFs); large quantity generators (LQGs); small quantity generator (SQGs); and transporters. RCRIS contains the following information:

- General information on all handlers (e.g., name, address, activity type);
- Permitting and corrective action program status, and Standard Industrial Classification (SIC) code information for TSDFs only; and
- Enforcement and compliance actions for specific facilities, regardless of type, which have been subject to inspections or other enforcement activity.

States and regions populated RCRIS with data necessary for their program implementation. Those portions of the data that were relevant for national program oversight and management were contained in a RCRIS national database.

The **BRS** was the national system that collected data on the generation, management, and minimization of hazardous waste. BRS captured detailed data on the generation of hazardous waste from large quantity generators and data on waste management practices from treatment, storage, and disposal facilities. These data were collected every other year, providing the ability to perform trend analyses. The data were reported by the facilities to EPA on even years regarding the hazardous waste activities of the previous year. EPA produced a report on hazardous waste generation and management activity that included the data files. The BRS can

be queried to identify facilities treating hazardous wastes with technologies that may generate air toxics emissions. BRS reports are available from EPA through 2003.[12]

RCRAInfo data is made available to the public through EPA's Envirofacts Data Warehouse[13] through monthly extracts or through the Right to Know Network.[14] The same files that are provided to Envirofacts and the Right to Know Network are also available for downloading from EPA's publically accessible FTP server.[b]

The RCRAInfo system that is replacing the RCRIS and the BRS allows tracking of many types of information about the regulated universe of RCRA hazardous waste handlers. RCRAInfo characterizes facility status, regulated activities, and compliance histories and captures detailed data on the generation of hazardous waste from large quantity generators and on waste management practices from treatment, storage, and disposal facilities. Although the BRS does not contain emissions monitoring data, it does identify hazardous waste constituents, quantities managed, and other facility information. For example, the RCRA files include trial burn data for hazardous waste incinerators, certification of compliance test data, and facility plot plans, all of which could be useful in risk assessments of air toxics.

7.3.8 Emissions and Dispersion Modeling System (EDMS)

For aircraft emissions, the Federal Aviation Administration has developed an emission estimation method model, Emissions and Dispersion Modeling System (EDMS), Version 4.0.[15] This model can be applied to specific airports and used to develop air toxics emissions for both commercial and general aviation emissions. The primary basis for estimating emissions is based on landing and take off data available from FAA's airport activity statistics database. EDMS includes emissions and dispersion calculations, the latest aircraft engine emission factors from the International Civil Aviation Organization (ICAO) Engine Exhaust Emissions Data Bank, vehicle emission factors from EPA's MOBILE5a, and EPA-validated dispersion algorithms.

7.3.9 Summary

Exhibit 7-7 lists the different data sources that provide information on air toxics emissions that are being used or can be adapted for air toxics risk assessments.

---

[b]The server is available at: ftp://ftp.epa.gov/rcrainfodata/brfiles/. A comprehensive web-enabled help module (RCRAInfo_Flat_File_WebHelp.zip) is also available to explain the flat file specifications and data element values (see ftp://ftp.epa.gov/rcrainfodata/rcra_flatfiles/).

Exhibit 7-7 Summary of Emissions Inventory and Related Information				
Data Source	**Maintained By**	**Sectors Covered**	**Comments**	**Address**
S/L/T Inventories	Individual S/L/T agencies	Large point sources; nonpoint sources; mobile sources from selected S/L/T agencies; coverage is variable	Many S/L/T agencies have specific information collected for special studies; attempts have been made to include most of the S/L/T-level data into the NEI, but higher resolution data may be available	Various S/L/T-specific and other web pages http://www.cleanairworld.org/
National Emissions Inventory (NEI)	U.S. EPA	Point sources; nonpoint sources; on-road and nonroad mobile sources	Point sources are reported for individual release points (includes other modeling data); nonpoint and mobile sources are reported at the county level	http://www.epa.gov/ttn/chief/net/index.html
Title V Permit Conditions	States/EPA	Large stationary point sources and limited coverage of nonpoint sources; only HAPs greater or equal to 10 tons/25 tons per year covered	Source-specific operating conditions to achieve permitted emissions levels; actual emissions reported for compliance in many cases; includes MACT requirements	Generally available in paper format only; some regional offices maintained databases
EIIP	States/EPA	Large point sources; nonpoint sources; mobile sources	Series of reports with recommended and alternative emissions estimation methods, and recommended emission factors	http://www.epa.gov/ttn/chief/eiip/index.html
Clearinghouse for Inventories and Emission Factors (CHIEF)	U.S. EPA	Collection of information, tools, and guidance on emissions from all sectors	EPA's main web page for emissions inventories and related data	http://www.epa.gov/ttn/chief/
Emissions Tracking System (ETS)	U.S. EPA	Large electric generating units	Annual reports of actual monitored emissions of SO2, NOx and CO2 from Title IV affected facilities, no toxics reported	http://www.epa.gov/airmarkets/emissions/index.html
RCRAInfo	U.S. EPA	Generally point sources; does include information on waste transporters	Reporting of releases from Hazardous Waste Treatment Storage and Disposal Facilities (TSDF); includes data from Biennial Reporting System (BRS) and Resource Conservation and Recovery Information System (RCRIS)	http://www.epa.gov/epaoswer/hazwaste/data/index.htm#rcra-info
Toxic Release Inventory (TRI)	U.S. EPA	Generally only point sources; includes only those sources that are subject to reporting thresholds	Self reported information at facility level; no other data necessary for modeling are reported (e.g., vent characteristics); updated annually; source and pollutant coverage can be limited by reporting thresholds; generally not recommended for modeling	http://www.epa.gov/tri/

References

1. U.S. Environmental Protection Agency. 2004. Technology Transfer Network Clearinghouse for Inventories & Emission Factors. *Emission Inventory Improvement Program.* Updated March 2, 2004. Available at: http://www.epa.gov/ttn/chief/eiip/index.html. (Last accessed March 2004).

2. U.S. Environmental Protection Agency. Technology Transfer Network Clearinghouse for Inventories & Emission Factors. *Emission Inventory Improvement Program*: *Technical Reports.* Updated November 5, 2002. Available at: http://www.epa.gov/ttn/chief/eiip/techreport/index.html. (Last accessed March 2004).

3. Pope, A. and Finn, S. *2003. Inventory Preparation for Toxics.* U.S. Environmental Protection Agency. 12th Annual International Emission Inventory Conference, San Diego, CA. April 29-May 1, 2003.

4. U.S. Environmental Protection Agency. 2001. 1999 National Emissions Inventory Preparation Plan - Revised (February 2001). Office of Air Quality Planning and Standards, February 2001. Available at: http://www.epa.gov/ttn/chief/net/nei_plan_feb2001.pdf.

5. U.S. Environmental Protection Agency. 1998. *Handbook for Air Toxics Emission Inventory Development.* Office of Air Quality Planning and Standards, Research Triangle Park, NC. EPA-454/B-98-002. Available at: http://www.epa.gov/ttn/chief/eidocs/airtoxic.pdf.

6. U.S. Environmental Protection Agency. 2004. Modeling and Inventories. *NONROAD Model (nonroad engines, equipment, and vehicles).* Updated March 11, 2004. Available at: http://www.epa.gov/otaq/nonrdmdl.htm. (Last accessed March 2004).

7. U.S. Environmental Protection Agency. 2004. Modeling and Inventories. *MOBILE Model (on-road vehicles).* Updated March 12, 2004. Available at: http://www.epa.gov/otaq/mobile.htm. (Last accessed March 2004).

8. U.S. Environmental Protection Agency. 2004. Technology Transfer Network Clearinghouse for Inventories & Emission Factors. *Source Codes.* Updated February 24, 2004. Available at: http://www.epa.gov/ttnchie1/codes/index.html. (Last accessed March 2004).

9. U.S. Environmental Protection Agency. 2003. Technology Transfer Network Clearinghouse for Inventories & Emission Factors. *Compilation of Air Pollutant Emission Factors, AP-42, Fifth Edition, Volume I: Stationary Point and Area Sources.* Updated December 10, 2003. Available at: http://www.epa.gov/ttn/chief/ap42/. (Last accessed March 2004).

10. U.S. Environmental Protection Agency. 2003. *Hazardous Waste Data.* Updated December 17, 2003. Available at: http://www.epa.gov/epaoswer/hazwaste/data/index.htm#rcra-info. (Last accessed March 2004).

11. U.S. Environmental Protection Agency. 2004. Planning and Results: Data Sources. *Resource Conservation and Recovery Act (RCRA) Information (RCRAInfo).* Updated February 10, 2004. Available at: http://www.epa.gov/Compliance/planning/data/multimedia/idea/datasources.html#RCR (Last accessed March 2004).

12. U.S. Environmental Protection Agency. 2003. Wastes. *Hazardous Waste Data, RCRAInfo.* Updated December 17, 2003. Available at: http://www.epa.gov/epaoswer/hazwaste/data/index.htm#br (Last accessed March 2004).

13. U.S. Environmental Protection Agency. 2002. Envirofacts Data Warehouse. *Resource Conservation and Recovery Act (RCRAInfo); Overview*. Updated September 16, 2002. Available at: http://www.epa.gov/enviro/html/rcris/. (Last accessed March 2004).

14. OMB Watch. 2004. The Right-to-Know Network. *Welcome.* Available at: http://www.rtk.net/. (Last accessed March 2004).

15. Federal Aviation Administration, Office of Environment and Energy. 2003. *Emissions and Dispersion Modeling System.* Updated August 8, 2003. Available at: http://www.aee.faa.gov/emissions/EDMS/EDMShome.htm (Last accessed March 2004).

Chapter 8 Quantification of Exposure: Dispersion, Transport, and Fate of Air Toxics in the Atmosphere

Table of Contents

8.1 Introduction 1

8.2 Dispersion and Atmospheric Transport of Air Pollutants 1
- 8.2.1 General Types of Releases 2
- 8.2.2 Characteristics of Releases that Affect Dispersion and Transport 3
- 8.2.3 Physical and Meteorological Factors Affecting Air Toxics Dispersion and Transport . 7

8.3 Fate of Air Toxics in the Atmosphere 13
- 8.3.1 Physical Processes Removing Air Toxics 13
- 8.3.2 Chemical Reactions that Remove Air Toxics 15
- 8.3.3 Chemical Reactions that Result in the Secondary Formation of Pollutants 16
- 8.3.4 Overall Persistence of Air Toxics in the Atmosphere 17

References 19

8.1 Introduction

This chapter describes the major physical processes that affect the movement of air toxics through the atmosphere. Section 8.1 describes the mechanisms through which sources release air toxics into the air and how specific release characteristics and meteorological factors affect air toxics dispersion and transport. Section 8.2 discusses the major physical and chemical processes that affect the fate of air toxics, including deposition and chemical reaction. The discussion of air toxics fate in this chapter is focused on describing the presence of air toxics in the atmosphere and processes that influence this presence. The fate of deposited air toxics in other media and ecosystems is included in Chapter 17.

The atmosphere and atmospheric processes is a complex and expansive subject. An understanding of at least the rudiments of this subject is central to an understanding of how air toxics disperse and persist or are removed once released to the air. Appendix G provides an overview of atmospheric and meteorological concepts and terms relevant to this chapter. Appendix G also provides information on sources of meteorological data for modeling air toxics dispersion and transport. Whenever possible, it recommended that a meteorologist be part of the risk assessment technical team.

8.2 Dispersion and Atmospheric Transport of Air Pollutants

Several characteristics of the source can affect the movement of air toxics while they are still close to the source (e.g., source height, gas exit temperature). Once air toxics are transported beyond the immediate vicinity of the source, atmospheric and meteorological factors (particularly wind speed and direction) govern air toxics dispersion and transport. This section describes how the movement of air toxics is affected by source characteristics, chemical properties, and atmospheric processes.

Dispersion, Transport, and Fate: What's the Difference?

Dispersion is a term applied to air toxics releases that means to spread or distribute from a source, with (generally) a decrease in concentration with distance from the source. Dispersion is affected by a number of factors including characteristics of the source, the pollutants, and ambient atmospheric conditions.

Transport is a term that refers to the processes (e.g., winds) that carry or cause pollutants to move from one location to another, especially over some distance.

Fate of air pollution refers to three things:

- Where a pollutant ultimately ends up (e.g., air distant from the source, soil, water, fish tissue);
- How long it persists in the environment; and
- The chemical reactions which it undergoes.

The fate of an air pollutant is governed both by transport processes and by the characteristics of the pollutant (e.g., its persistence, its ability to undergo reaction, and tendency to accumulate in water or soil, or to concentrate in the food chain).

8.2.1 General Types of Releases

The discussion of air toxics sources in Chapter 4 described air toxics emissions from a regulatory perspective (e.g., "stationary" versus "mobile" sources). This chapter focuses instead on emissions from the perspective of the primary types of industrial or physical processes through which sources release most pollutants to the air. The distinction between the major types of releases is not always clear, and the same air toxic can often be released in more than one way from a single source or process. Section 8.2.2 below discusses how the characteristics of these different types of release affect dispersion and transport of air pollutants.

The following terms are routinely used to generally describe or categorize emissions at a facility:

- **Stack or Vent Emissions**. These emissions are how most people envision air pollution. Stacks and vents include "smokestacks" that emit combustion products from fuel or waste combustion, as well as vents that carry air toxics away from people or industrial processes. The major characteristics that stack and vent emissions share are that the release is intentional, they remove airborne materials from specific locations or processes, and they channel the releases through dedicated structures designed specifically for that purpose. Often, stack and vent releases involve the active "pumping" of pollutant-laden air to the external atmosphere by using fans or the "draft" associated with the tendency of hot gases to rise rapidly through cooler, denser air. For many industrial operations, stack and vent emissions account for the bulk of releases (according to the most recent TRI reports from large industrial sources, about 86 percent).[1] For this reason, firms often install pollution control equipment in stacks or vents to reduce the concentration of potentially toxic pollutants released to the environment.

- **Fugitive Emissions**. "Fugitive" emissions are uncontrolled air pollutant releases that "escape" from physical, chemical, or industrial processes and activities, and which do not travel through stacks or vents. Examples include dust or vapors that are generated by the transfer of bulk cargo (e.g., coal, gravel, occasionally organic liquids) from one container to another (e.g., from a tank or hopper car to a storage silo, tank, or bin). Another example includes leaks from joints and valves at industrial facilities and evaporative emissions of fuel from mobile sources.

  "Fugitive dust" emissions often occur when quarrying, earth moving, construction, or excavation activities produce particulates. Such emissions are often called "fugitive" because they are uncontrolled (though this is not always the case). Historically, EPA has regarded fugitive emissions as being less important than stack and vent emissions; this is because either the amount of material released was relatively small or the characteristics of the release (large particle size, for example) precluded transport over large distances. However, the combined fugitive emissions from intensive or widespread industrial activities can be as important a contributor to risk as stack emissions.

The following terms are routinely used to describe processes that generate emissions.

- **Particle Suspension and Entrainment**. Particle suspension refers to a set of physical release mechanisms, without reference to specific types of sources and can overlap with the

previous definition of fugitive emissions. Suspension and entrainment refers to any process that results in the release of particles into the air from soils or other surfaces. Suspension and entrainment can occur as a result of artificial soil disturbance; or the action of wind on loose soil, sand or dust. Depending on the nature of the material and atmospheric conditions, suspended particles transport only a few feet, or may transport very long distances before redepositing (for example, dust storms that originate in the Sahara Desert may blow across the Atlantic and impact Central and North America). In some sections of the western United States, the majority of particulate matter detected in air is "crustal material" (soils and fine rock particles) suspended and transported by the wind, rather than human-made pollutants.

- **Volatilization/Vapor Release**. Many organic compounds and some inorganic compounds may "volatilize" to some extent; this means that these compounds tend to evaporate at normal atmospheric temperatures and pressures when not contained. Volatilization can occur for chemicals contained in mixtures as well as from concentrated or pure forms. Common examples include the lighter components of gasoline such as benzene, which volatilize to a sufficient degree that they can be smelled (and sometimes seen) when cars are refueling at filling stations without vapor control systems. Sources can release vapors when handling highly-concentrated or pure organic compounds, as well as when solids and liquids with low levels of organic contamination are exposed to the atmosphere. Volatilization releases are common from chemical manufacturing and processing operations, from metal cleaning and dry cleaning operations that use organic solvents, and from waste management facilities. Volatilization also is important for many natural sources of organic emissions. For example, in heavily forested areas, terpenes (volatile chemicals emitted from pines and other tree species) can account for a large proportions of total organic air pollution.

Common Measures of Chemical Volatility

Henry's Law Constant. The ratio at equilibrium of the gas phase concentration to the liquid phase concentration of a gas. Note that the Henry's Law constant can be defined in several ways and expressed using different units.

Vapor pressure. The pressure exerted by a vapor, either by itself or in a mixture of gases; often taken to mean saturated vapor pressure, which is the pressure of a vapor in contact with its liquid form.

Volatile organic compounds (VOCs) are not the only types of substances that vaporize. Some metals (e.g., elemental mercury), organometallic compounds, and other inorganic substances (e.g., ammonia and chlorine) have a high vapor pressure. Many semivolatile organic compounds (SVOCs) also volatilize relatively quickly, albeit at a slower rate than VOCs. Some solids also have a high vapor pressure (mothballs are a common example).

8.2.2 Characteristics of Releases that Affect Dispersion and Transport

EPA and others have developed several air "dispersion" models to predict the often-complex behavior of air pollution releases. Most air dispersion models take into account a number of characteristics of the source and pollutants released. The most common of these characteristics are described below.

- **Release Rate and Volume**. The rate of release (exit velocity) strongly influences the behavior of the pollutant **plume** as it moves through the atmosphere. In the case of stack and

vent releases, sources can release pollutants as pure vapors, as dilute solutions of vapor in air or other gases, or as suspended particles. Large volumes are often released at a relatively high velocity from stacks or vents, which can also serve to drive pollutants higher in the atmosphere. Air quality models often calculate volume from data on exit area and exit velocity. In the case of fugitive releases or volatilization, the "volume" of release has less meaning, and often does not receive explicit consideration in fate and transport modeling.

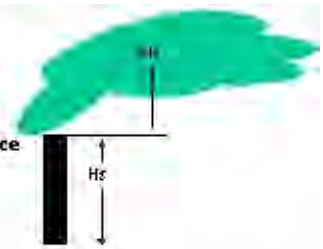
- **Concentration**. Concentration (the mass of pollutant per unit volume of released gases) is the other half of the equation that determines the amount (mass) of pollutant released. Pollutants at higher concentrations may also be more likely to condense onto particles or liquid droplets.

- **Temperature**. The temperature of a plume emitted from a stack or vent influences the dispersion and transport of pollutants. A plume that is warmer than the surrounding air will generally rise, which tends to increase the distance over which pollutants will be transported. The combination of temperature and vertical velocity of stack emissions combine to affect the height to which the plume will rise and the layer of the atmosphere in which it will initially be transported. As with concentration, the temperature of the plume also affects the physical form of pollutants, with less volatile pollutants condensing faster from cooler plumes.

- **Height**. Pollutants may also be released into the atmosphere at different heights, and the height of release can strongly affect dispersion and transport. Greater release heights generally result in increased pollutant dilution in the atmosphere, lower ground-level concentrations, and a greater distance to peak ground-level concentrations. Release height also is important in evaluating local effects on air transport, such as building downwash. While power generation or industrial activities may release combustion products from stacks that are hundreds of feet tall, volatilization releases or suspension of particulate pollutants often occur at or near the ground surface.

Source Characteristics Affecting Dispersion

- Release rate
- Plume height = Hs + ΔH
  - *Physical release height (Hs)
    - From a stack
    - From an area/volume source
    - From the ground
  - *Plume rise (ΔH)
    - Exit velocity
    - Stack temperature
    - Wind speed



- **Timing and Duration**. Multiplying the release duration by the release rate produces the total mass of pollutant released. The timing of release relative to specific meteorological conditions determines the particular dispersion and transport of pollutants. Unfortunately, only total annual or average daily release data are available for most sources, making it difficult to fully characterize time varying releases. Fortunately, chronic exposure assessments usually focus on the average long-term (annual) concentrations. Acute exposure assessments, however, usually focus on the maximum short-term (24-hours or less) concentration. Acute exposures are derived from conservative meteorological factors that

lead to the highest short term peak values for a screening exercise; for a more detailed exercise, the actual meteorology should determine the short term peaks.

- **Physical Form**. The physical form of pollutant releases greatly affects the dispersion, transport and chemical reactions that pollutants undergo. Generally, pollutants are characterized as being **vapors** (not bound to particles, but existing as single molecules or very small aggregates "dissolved" in air – also called **gaseous**), **particle-bound** (reversibly absorbed or condensed onto the surface of particles), or **particulate** (irreversibly incorporated into airborne particles). The distribution of pollutants in these three "phases" is known as **partitioning**. Partitioning is a function of the chemical and physical properties of the pollutants and the temperature and pressure of the atmosphere into which the chemicals have been released (e.g., the partitioning behavior of pollutants can vary greatly with temperature). As noted above, sources can emit chemical pollutants in the vapor phase at relatively high temperatures, and these pollutants can condense into or onto particulates as the emitted gases cool in the atmosphere. Sources generally emit most metals (with the important exception of mercury) as particles in the atmosphere.

- **Particle Size**. When sources release pollutants as particles (or if released as gases, if these pollutants condense into particles or absorb onto the surface of existing particles), the rate of pollutant removal from the atmosphere to surfaces (e.g., plants, soils, surface water) depends upon particle size. The typical size of particles that different activities and processes emit into the air can vary by many orders of magnitude (powers of ten). As the size of particles increases, the rate at which particles fall due to gravity (the settling velocity) increases. Thus, fine particles (approximate diameter less than a few microns)[(a)] may remain suspended in air indefinitely, but particles larger than about 20 microns in diameter settle rapidly and may not transport far from sources of release.

  For purposes of air toxics risk assessment, particles less than 10 microns in diameter are of primary concern because they are small enough to be taken into and deposited in the lung after inhalation. These particles are divided into two size ranges: "fine" particles less than 2.5 micrometers in size ($PM_{2.5}$), and "coarse" particles covering the range from 2.5 to 10 micrometers in diameter (PM_{10}). (Thus, monitoring analyses for metals in particulates, for example, would commonly collect particulate samples of $PM_{2.5}$ and PM_{10} for analysis, not total suspended particulate (TSP), because such samples contain particles that are too large to be effectively respired – thereby leading assessors to overestimate inhalation risk).

  Particles emitted from combustion and high-temperature chemical processes can be very fine, on the order of 0.01 to 1.0 microns in diameter. Such fine particles tend to condense to form larger aggregates up to the limit of perhaps a few microns, and participate in a wide range of chemical reactions.

[a]One micron is one one-millionth of a meter, or about 0.00004 inches.

Aerosols and Particulate Matter

Aerosols are mixtures of fine solid or liquid particles in a gas. They can be emitted directly as particles or formed in the atmosphere by gas-to-particle conversion processes. The terms dust, smoke, fume, haze, and mist all describe different types of aerosols. Dust refers to solid particles produced by disintegration process; smoke and fume are particles formed from the gas phase. Mists are composed of liquid droplets. Some aerosols occur naturally, originating from volcanoes, dust storms, forest and grassland fires, living vegetation, and sea spray. Human activities, such as the burning of fossil fuels and the alteration of natural surface cover, also generate aerosols.

Particulate matter is the term given to the tiny particles of solid or semi-solid material found in the atmosphere. Particulates less than about 50 micrometers in size are called total suspended particulates (TSP). Particles larger than that range tend to quickly settle out of the air. Particulate matter 10 micrometers in diameter and smaller (PM_{10}) is considered inhalable. These particles are divided into two size ranges: fine and coarse.

"Fine" particles less than 2.5 micrometers in size ($PM_{2.5}$) are responsible for causing the greatest harm to human health. 1/20th the width of a human hair, these fine particles can be inhaled deep into the lungs, reaching areas where the cells replenish the blood with oxygen. They can cause breathing and respiratory symptoms, irritation, inflammation, damage to the lungs, and premature deaths. Some $PM_{2.5}$ are released directly to the atmosphere from industrial smokestacks and automobile exhaust, but a large percentage is actually formed in the atmosphere from other pollutants such as sulphur dioxide, nitrogen oxides, and volatile organic compounds.

"Coarse" particles covering the range from 2.5 to 10 micrometers in diameter are also known to cause adverse health effects, such as aggravation of respiratory disease. When inhaled, particles larger than 10 micrometers tend to be deposited in the upper parts of the respiratory system, from which they can be eventually expelled back into the throat. Coarse particles generally remain in the form in which they are released into the atmosphere without chemical transformation, eventually settling out under the influence of gravity. While some of these coarse particles are generated naturally by sea salt spray, wind and wave erosion, volcanic dust, windblown soil, and pollen, they are also produced by human activities, such as construction, demolition, mining, road dust, tire wear, and grinding processes of soil, rocks, or metals.

- **Chemical Form**. Chemical form is generally more of a concern for inorganic pollutants, because organic chemicals tend to have well-defined chemical compositions and properties. The most important chemical properties of inorganic metal compounds, for example, include the **oxidation** or **valence state** of the **cationic metal**, the identity of the **anionic counterion**, and the chemical and physical properties of the compound that the cation and anion comprise. As an example of the importance of valence state, consider the metal chromium. When emitted in the *hexavalent* form (with six positive charges – Cr^{6+}), chromium is highly reactive chemically and is readily reduced under certain conditions to the *trivalent* form, which is Cr^{3+}. Cr^{6+} can cause respiratory irritation and cancer in humans. Cr^{3+}, on the other hand, is much more stable, is much less toxic to humans and animals (and is actually an essential mineral), and is not thought to cause cancer.

Available air pollution dispersion models differ in the ways in which they use these characteristics. While Chapter 9 presents a general discussion of these, users can find specific details in the documentation for the various models at EPA's (Support Center for Regulatory Air Models) SCRAM website.[2]

8.2.3 Physical and Meteorological Factors Affecting Air Toxics Dispersion and Transport

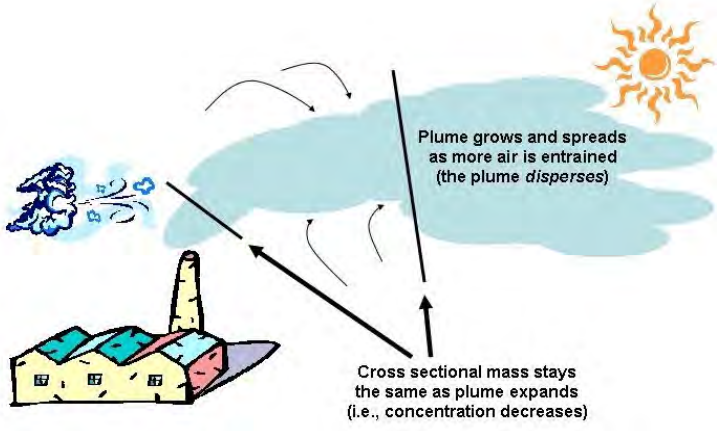
This section describes some of the most important physical and meteorological factors that affect the movement of air pollutants after their release. For definitions of and further details about the atmospheric and meteorological terms this section uses, refer to Appendix G.

Wind Speed and Direction Affect Plume Dispersion

The wind will determine which direction the plume goes and how fast it gets there. To look at long-term impacts (chronic exposure) a wind-rose (a distribution of winds around a compass) can be used to determine the areas of persistent wind; downwind of the largest persistent winds will generally be the areas to expect the maximum long-term impacts.

Although this section focuses on releases from stationary point sources (i.e., stacks), most of the factors may apply to releases from other source types as well. Stacks come in all sizes, from a small vent on a roof to stacks hundreds of feet in height. The function of a stack is to remove pollution of high concentration and to discharge it to the atmosphere for dispersion and transport. Stacks release pollutants high enough above the earth's surface that pollutants can sufficiently disperse in the atmosphere before reaching ground level. All else being equal, taller stacks disperse pollutants more effectively than shorter stacks because the pollutants release into higher wind speeds and travel through a greater depth of the atmosphere before reaching ground level.

Concentrations of Air Toxics in a Plume Decrease With Distance



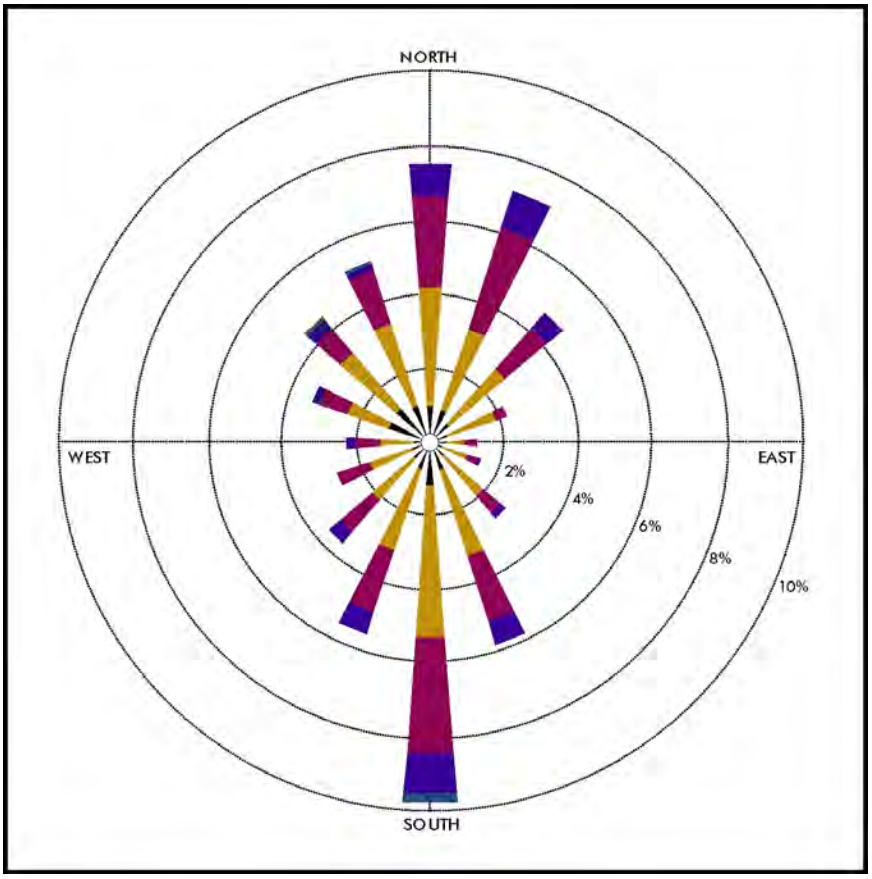
As a plume grows, it entrains clean ambient air and disperses. In other words, the amount of pollutant mass at any given cross section in the plume is generally the same; thus, as the plume spreads, its concentration goes down.

The air space the stack pollutant occupies can be described as a **plume**. As the plume travels, it spreads and disperses, reducing ambient pollutant concentrations even though the cross sectional mass of the plume remains the same. Eventually, the plume may intercept the ground. The combination of emission velocity, emission temperature (see below), vertical air movement and horizontal airflow all influences how high a plume will rise and how fast and far it travels. Another factor is wind meander (i.e., changes in wind direction during light wind speed

conditions), which can cause the plume to deviate in the horizontal direction due to turbulence and wind fluctuation.

As the gases exit the stack, they mix with ambient air. This mixing of ambient air into the plume is called **entrainment**. The plume grows in volume as it entrains ambient air and travels downwind. Because stack gases are often warmer than the ambient outdoor air, the gases may be less dense and than the outdoor air and are therefore buoyant (like a helium filled balloon). Gases that stacks emit are often pushed out by fans giving the gas momentum as it enters the atmosphere. The combination of this momentum and the buoyancy of the stack gases that are warmer than the ambient air cause the gas to rise.

Example of a Wind Rose



A wind rose illustrates, for a given locality or area, the frequency and strength of the wind from various directions for a selected time period, typically a year or longer. In this example, the length of the bars indicates the percentage of the year the wind blows in each of 16 directions and the colors indicate the percent of time at that wind speed for each of these directions.

This plume rise allows air toxics emitted in this stack stream to be lofted higher in the atmosphere. Since the plume is higher in the atmosphere where the winds are generally stronger, the plume will generally disperse more before it reaches ground level. Plume rise depends on the stack's physical characteristics and on the effluent's exit temperature and velocity.

Vertical Air Motions

When air is displaced vertically, atmospheric behavior is a function of atmospheric stability. A stable atmosphere resists vertical motion, and air that is displaced vertically in a stable atmosphere tends to return to its original position. This atmospheric characteristic determines the ability of the atmosphere to disperse pollutants. To understand atmospheric stability and the role it plays in pollution dispersion, it is important to understand the dynamics of the atmosphere as they relate to vertical atmospheric motion.

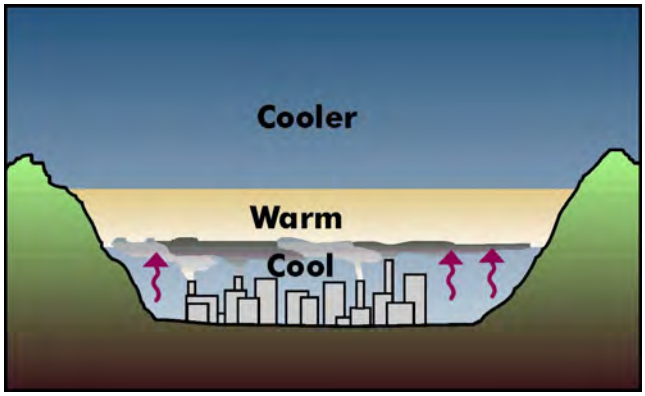
The degree of stability of the atmosphere is determined by the temperature difference between an air parcel and the surrounding air. This difference can cause the parcel to rise or fall. There are three general categories of atmospheric stability.

- In **stable** conditions, vertical movement tends not to occur. Stable conditions occur at night when there is little or no wind. Air that lifts vertically will remain cooler, and therefore denser than the surrounding air. Once the lifting force ceases, the air that has lifted will return to its original position.

- **Neutral** conditions ("well mixed") neither encourage nor discourage air movement. Neutral stability occurs on windy days or when there is cloud cover such that there is neither strong heating nor cooling of the earth's surface. Air lifted vertically will tend to remain at the higher level once the lifting force ceases.

- In **unstable** conditions, the air parcel tends to move upward or downward and to continue that movement. Unstable conditions most commonly develop on sunny days with low wind speeds where strong solar radiation is present. The earth rapidly absorbs heat and transfers some of it to the surface air layer. As warm air rises, cooler air moves underneath. The cooler air, in turn, may be heated by the earth's surface and begin to rise. Such conditions enhance vertical motion in both directions and considerable vertical mixing occurs.

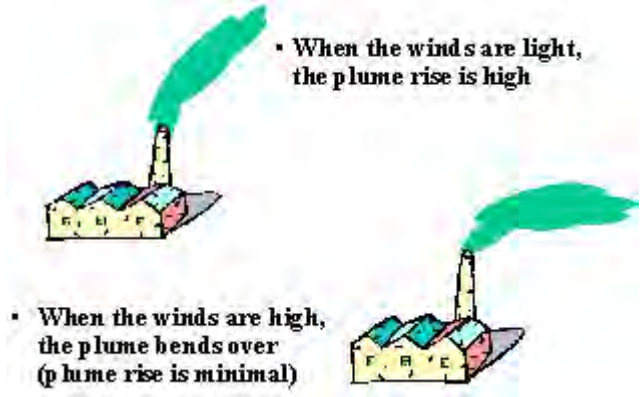
- **Inversions** occur whenever warm air over-runs cold air and "traps" the cold air beneath. Within these inversions there is little air motion, and the air becomes stagnant. High air toxic concentrations can occur within inversions due to the limited amount of mixing between the "trapped" air and the surrounding atmosphere. Inversions can limit the volume of air into which emissions are dispersed, even from tall stacks.



The condition of the atmosphere (i.e., the vertical profile of the winds and temperature) along the path of the plume also determines how far the plume rises in the atmosphere. As described in Appendix G an **inversion layer** (formed when a layer of warm air "traps" a layer of cold air beneath) may act as a barrier to vertical mixing. The height of a stack in relation to the height of the inversion layer may often influence ground-level pollutant concentrations (Exhibit 8-1).

The initial velocity of the plume (stack exit velocity) reduces quickly as the plume entrains ambient air and acquires horizontal momentum from the wind. This momentum causes the plume to bend over. The greater the wind speed, the more horizontal momentum the plume acquires. Wind speed usually increases with height above the earth's surface. Therefore, as the plume continues upward the stronger winds tilt the plume even further. This process continues until the plume may appear to be horizontal to the ground. The point where the plume looks level may be a considerable distance downwind from the stack (Exhibit 8-2).

Wind Speed Affects Plume Rise

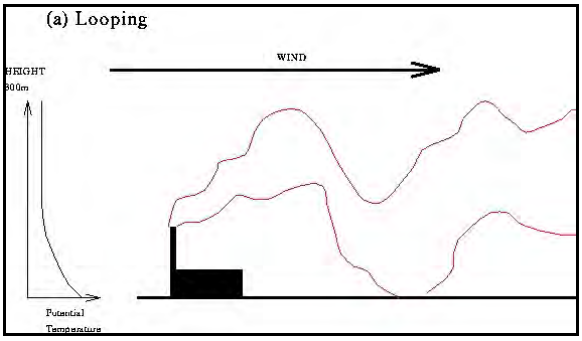


The strength of the wind will help determine how high the plume rises; a strong wind will "knock-over" the plume right away, while a light wind will allow a plume to rise from its own buoyancy and initial inertia.

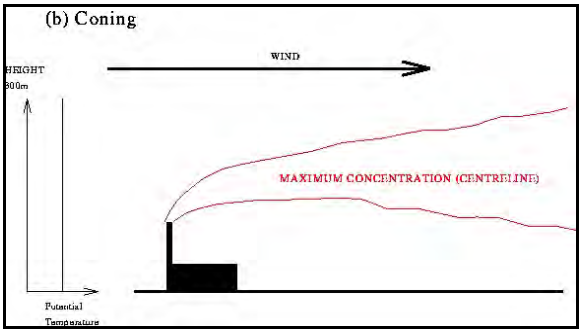
Due to configuration of the stack or adjacent buildings, the plume may not rise freely into the atmosphere. The way in which the wind moves around adjacent buildings and the stack can force the plume toward the ground instead of allowing it to rise in the atmosphere. Stack-tip downwash can occur where the ratio of the stack exit velocity to horizontal wind speed is small. In this case, low pressure in the wake of the stack may cause the plume to draw downward behind the stack. Pollutant plume rise reduces when this occurs and elevates pollutant concentrations immediately downwind of the source. As air moves over and around buildings and other structures, it forms turbulent wakes. Depending upon the stack height, it may be possible for the plume to be pulled down into this wake area. The reduction in plume height is known as **aerodynamic or building downwash** (Exhibit 8-3).

Once air toxics have equilibrated with ambient conditions (e.g., temperature, velocity), atmospheric and meteorologic factors primarily influence dispersion and transport of air toxics. In particular, the rate of dispersion is influenced by both the thermal structure of the atmosphere and mechanical agitation of the air as it moves over the different surface features of the earth (see Appendix G). As the next section describes, exposure to solar radiation and moisture, as well as other properties in the atmosphere, complement the factors above and contribute to the eventual fate of the air toxics.

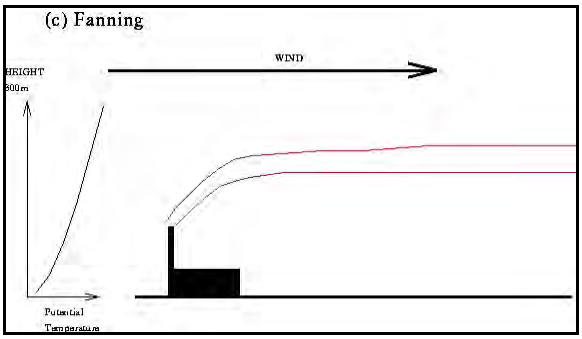
Exhibit 8-1. Effects of Boundary Layer Conditions on Plume Dispersion



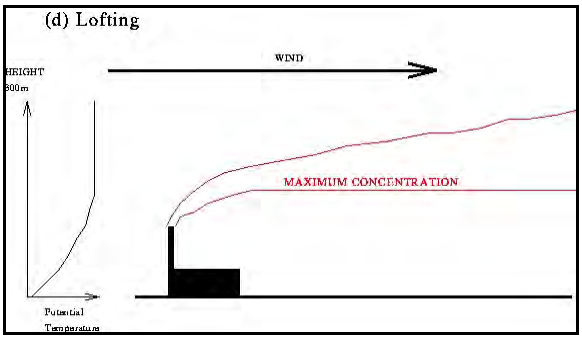
(a) Looping. When the atmosphere is very unstable through a deep layer, convective currents carry the plume up and down, forming a looping pattern and rapidly diluting the plume through intense vertical mixing.



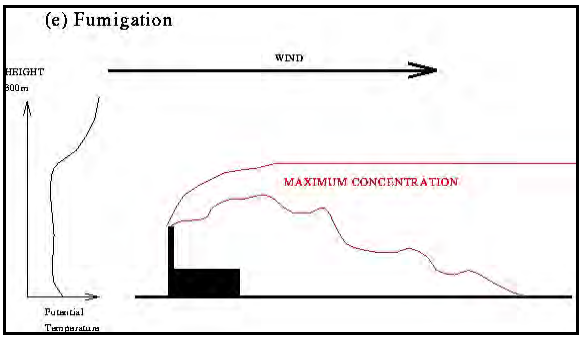
(b) Coning. Under neutral conditions, the vertical mixing also transports freely, but the turbulent motions that irregularities of the ground and shearing of the wind introduce are about equal, and the plume resembles a cone.



(c) Fanning. When the plume rises into an inversion layer, the stability limits diffusion up or down, so that the only spreading of the plume is sideways and when viewed from above has a fanning appearance.



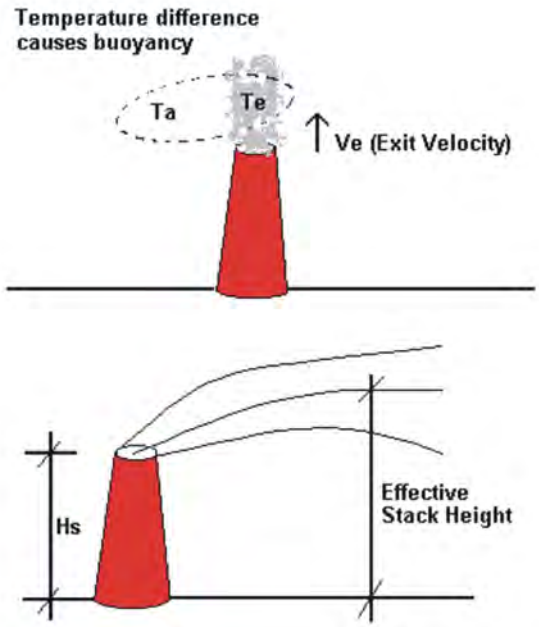
(d) Lofting. When conditions are unstable or neutral above an inversion, the release of a plume above the inversion is more likely to result in effective dispersion and lower ground-level concentrations around the source



(e) Fumigation. If the plume is released overnight just under an inversion layer, it can become trapped. As solar radiation warms the ground in the morning, the air below an inversion layer becomes unstable. If the unstable conditions then extend upwards and reach the plume that is still trapped below the inversion layer, the pollutants can be rapidly transported down toward the ground. Ground-level pollutant concentrations can be very high when fumigation occurs.

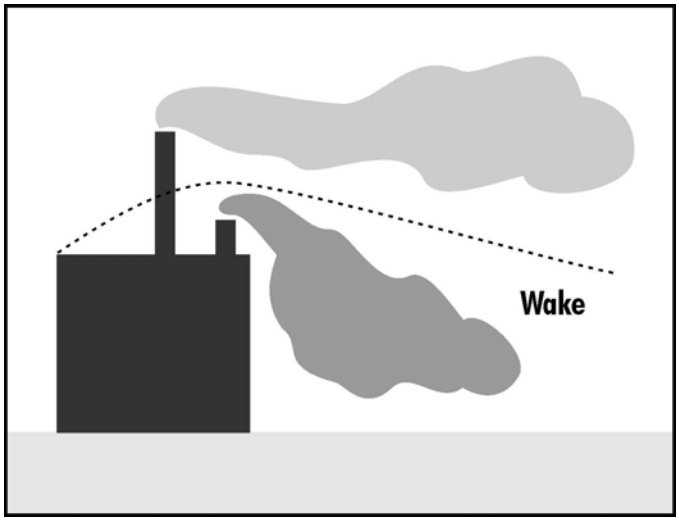
Graphics courtesy of Doug Parker, University of Leeds, and available at: http://www.env.leeds.ac.uk/envi1250/lectures/lect11.html.

Exhibit 8-2. Plume Rise Schematic



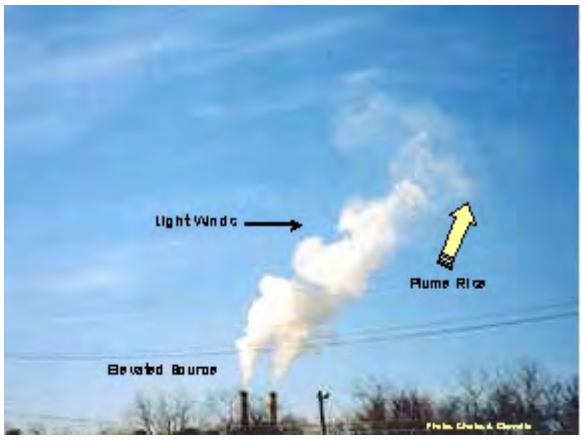
As emissions exit a stack, they can be lofted higher into the atmosphere as a result of plume rise. Plume rise is caused by (1) buoyancy resulting from emissions with higher temperatures than ambient air (i.e., Te > Ta) and (2) the initial momentum (i.e., exit velocity) of the emissions leaving the stack. The effective stack height of a stack is equal to the actual physical height of the top of the stack (Hs) plus the plume rise, minus any downwash associated with wake turbulence behind objects on the ground (see Exhibit 8-3).

Exhibit 8-3. Aerodynamic or Building Downwash



Nearby structures can disturb the horizontal flow of the wind (indicated by the dashed line) and cause a plume to get "downwashed" to the ground quickly. This is how a snow fence works.

Examples of Plume Rise



The picture on the left is an example of an elevated point source that probably has a hot plume or high exit velocity, as the plume rise indicates. Because of the light wind and neutral or unstable atmospheric conditions, there are essentially no impacts at ground level near the source. The picture on the right is another example of elevated point sources, but this time with higher winds. There is little plume rise, and the plumes fan straighter out from the stacks, resulting in narrower plumes.

8.3 Fate of Air Toxics in the Atmosphere

This section discusses the major physical and chemical processes that affect the fate of air toxics in the atmosphere. The scope of this section is limited to processes that remove air toxics from the atmosphere. Part III of this reference manual discusses the fate and transport of air toxics in other environmental media and the ecosystem once a chemical has been removed from the air.

8.3.1 Physical Processes Removing Air Toxics

A number of important physical processes (processes that do not alter the chemical nature of pollutants) affect how air toxics move in and out of the atmosphere. In particular, this section discusses how gravity and precipitation remove air toxics from the atmosphere. The process through which particulates fall (or settle) to the surface in the absence of precipitation is known as **dry deposition**, and the removal of pollutants from the air through precipitation events is called **wet deposition**.

- **Dry Deposition**. As the previous section noted, dry deposition is the settling of particles due to gravity. The maximum speed at which a particle will fall in still air is known as the **settling velocity (settling rate)**. A particle's settling velocity is a function of its size, density, and shape. Larger, denser particles settle more rapidly, and particles with more irregular shape settle more slowly (Exhibit 8-4). For particles smaller than a few microns in diameter (fine and ultrafine particles), the gravitational settling rate is so slow that other forces, such as local air currents and collisions with gas molecules, tend to offset it. Thus, in the absence of other removal mechanisms (e.g., condensation and/or aggregation to form larger particles, wet deposition), particles in this size range tend to remain suspended in the

air for long periods of time. Depending on the conditions, fine particles may persist in the atmosphere for days or weeks and travel hundreds or thousands of miles from their source. At the other extreme, coarse dust particles (> 50 microns in diameter), such as those generated while handling materials, have large settling velocities. Under normal conditions, such particles generated near the ground will deposit on the surface within a few seconds or minutes, generally within less than a kilometer of the source. Particles in between these two extremes in the size distribution will settle at intermediate velocities, and will distribute at intermediate distances from their sources.

Exhibit 8-4. Approximate Settling Rates for Typical Particles in Air

Equivalent Diameter* (microns)	Settling Rate (cm/sec)
0.01	0.00001
0.1	0.0002
1.0	0.01
10	0.6
100	40

* Diameter of a sphere that is approximately equivalent to a particle's diameter

In typical air dispersion models, the modeler must specify a particle size distribution, classifying what proportion of the emitted particles are within particular size ranges. In initial screening-level analyses of pollutant levels in air, users assume that particulate settling does not occur. This is conservative relative to the air concentration - if the amount deposited to the ground is the key issue of concern, then a high removal rate from the atmosphere would be "conservative." Users can assess dry deposition for low-volatility pollutants that partition out of the air primarily onto airborne particles in the same fashion as non-volatile particulates. Volatile chemicals that exist primarily in the vapor phase have negligible settling velocities, and modelers generally need not consider dry deposition for these pollutants. In the case of pollutants whose vapor-particle partitioning is unclear, it is common to run air dispersion models assuming a range of partitioning behavior from fully particle-bound to fully vapor phase.

- **Wet Deposition**. Wet deposition involves the "washing out" of pollutants from the atmosphere through precipitation events (including rain, snow, and in some cases hail). Wet deposition affects both particulate and vapor-phase pollutants. For larger particles and vapor phase pollutants that are soluble in water, precipitation is very efficient at removing pollutants from the air and depositing them on the earth's surface. Wet deposition may be less efficient at removing fine particulates, and has limited effect on the levels of gaseous pollutants with high Henry's Law constants (indicating low solubility in water compared to vapor pressure). Because wet deposition depends on the occurrence of precipitation events, it is best characterized over long periods (e.g., seasons or years). The relative importance of precipitation in removing pollutants from the air depends on the climatic conditions in the areas affected by pollution.

Transport and Deposition of Mercury

Mercury is a natural trace element in nearly all coal, and the large quantities burned in major electric power plants can release considerable mercury. In both municipal and medical incinerators the variety of waste materials burned can include mercury. Some mercury remains in the ash-materials, some may be captured by pollution controls on exhaust, and the rest is emitted to the air in three forms or chemical "species:" gaseous elemental mercury (Hg(0)), gaseous ionic or "inorganic" mercury (Hg(II)), and particle-bound mercury (Hg(p)). Elemental mercury gas is nearly insoluble in water and rather inert chemically, so it can be transported up to thousands of miles while gradually being converted to other forms and deposited. The ionic form, Hg(II) or Hg (2+), is soluble in water and thus incorporated into rain, fog, and snow. Also, Hg(II) is both physically and chemically active and is known as "reactive gaseous mercury" or RGM. Most of the Hg(II) emitted is deposited via both precipitation and dry gases within about 30 to 60 miles from a stack. In many cases, this "local" deposition can be the most important impact of mercury from combustion sources. The fate of particle-bound mercury depends on the size of the particles, though generally they deposit to earth within a few hundred miles of the emitting stack.

8.3.2 Chemical Reactions that Remove Air Toxics

In addition to deposition, chemical reactions may occur that reduce air toxics concentrations. Air toxics may be destroyed through the action of sunlight, through reactions with atmospheric chemical pollutants, or through a combination of these pathways. In estimating the ambient air concentration associated with air toxics releases, it is therefore necessary to consider chemical reactions as well as deposition. As will be discussed in the next section, not all chemical reactions result in the destruction of air toxics, or their conversion to less harmful products. Potentially harmful pollutants may also be formed as a result of atmospheric chemical reactions (a process that is called **secondary production** or **secondary formation)**. This section, however, focuses on atmospheric chemical reactions which are known or believed to destroy air toxics (i.e., resulting in less toxic forms).

Chemical Transformations Can Occur in the Atmosphere

$$X + Y \xrightarrow{\text{light}} Z$$

Numerous complex chemical transformations may occur in the atmosphere, some of which are photochemical in nature (i.e., reactions in the presence of light to form new chemicals).

- **Major Chemical Reactions of Air Toxics**. Generally, organic compounds are much more susceptible to chemical reactions in the atmosphere than metals or other inorganic contaminants. The major chemical reactions undergone by organic chemicals in the atmosphere include:

  – Photolysis (destruction by sunlight alone);
  – Reactions with the hydroxyl radical (OH•);

– Reactions with the nitrate radical (NO_3•); and
– Reaction with ozone (O_3).

Often these reactions occur in combination with reactions that are strongly affected by sunlight. While reaction rates vary widely for pollutants, under typical atmospheric conditions, reactions with the hydroxyl radical are the most rapid, and account for a large portion of pollutant degradation during daylight hours. Reactions with nitrate radical occur primarily during the night, and reactions with ozone occur both day and night. Except in the case of a few pollutants, "pure" photolysis is a relatively minor reaction process. Other reactive species such as the hydroperoxide radical (OOH•) may also participate in pollutant degrading reactions under some conditions. The relative importance of these reactions is dependent not only on climatic factors (e.g., duration and intensity of sunlight), but also on the overall concentrations of pollution present. For example, high levels of nitrogen oxide (NO_x) emissions and emissions of VOCs increase the levels of nitrate radicals and ozone in the atmosphere, thereby increasing reaction rates for subsequent reactions where these species are involved.

8.3.3 Chemical Reactions that Result in the Secondary Formation of Pollutants

As noted previously, not all chemical reactions result in the destruction of pollutants or in reaction products that are of less concern than the pollutants from which they derive. In some cases, the immediate reaction products result in products that are more toxic and/or more persistent than the chemicals that were originally released into the atmosphere.

Examples of large-scale chemical reactions that result in products that can be hazardous to health include the generation of acid particulate through photo-oxidation after the release of sulfur dioxide (SO_2) and NO_x from combustion sources (i.e., to make sulfuric acid and nitric acid), and the formation of ozone and photochemical oxidant in areas with high levels of NO_x and volatile organic emissions. In addition, there are many reactions of specific organic pollutants that generate air toxics of concern, as Exhibit 8-5 shows. The extent to which these reactions are important at any given location depend, of course, on the emissions and resulting concentrations of the precursor materials. In addition, many of these reactions are catalyzed directly and indirectly by sunlight, so weather and climatic factors are important in judging the importance of secondary formation. While it is difficult to generalize, the secondary formation of formaldehyde and acrolein are thought to be important in many regions of the country with significant industrial and mobile source emissions.[3]

Exhibit 8-5. Examples of Secondary Pollutants

Pollutant	Formed From
acetaldehyde	propene, 2-butene
acrolein	1,3-butadiene
carbonyl sulfide	carbon disulfide
o-cresol	toluene
formaldehyde	ethene, propene
hydrogen chloride	nitric acid, chlorinated organics
methylethyl ketone	butane, branched alkenes
N–nitroso-N–methylurea	N–methylurea
N–nitrosodiethylamine	dimethylamine
N–nitrosomorpholine	morpholine
phosgene	chlorinated solvents
propionaldehyde	1-butene

Source: Rosenbaum et al., 1998[3]

8.3.4 Overall Persistence of Air Toxics in the Atmosphere

In analyzing the potential impacts of air toxics releases, it is necessary to combine considerations of all the above processes to characterize the overall pattern of air toxics concentrations and estimate the time periods and distance scales over which air toxics impacts need to be evaluated.

Detailed quantitative comparisons of removal pathways may be too complex and expensive to include in most risk assessments. However, air pollution scientists have developed a number of simple models and gathered data on a large number of pollutants that enable them to assess the relative impacts of different physical processes and chemical reactions on single chemicals under typical conditions. The most common model uses the simplifying assumption that pollutant removal through each chemical and physical processes can be approximated using processes that have characteristic **half-lives** and **atmospheric lifetimes** (Exhibit 8-6).

Under the most commonly used approach (many variations exist), the overall lifetime of a pollutant in the environment is:

$$\frac{1}{r_{overall}} = \frac{1}{r_{physical}} + \frac{1}{r_{chemical}} \qquad \text{(Equation 8-1)}$$

That is, the overall lifetime ($1/r_{overall}$) of a chemical in the environment is equal to the sum of the atmospheric lifetime when considering only physical processes ($1/r_{physical}$) plus the lifetime when considering only chemical processes ($1/r_{chemical}$). This equation is the same as saying that the overall rate constant for pollutant removal/destruction ($r_{overall}$) is equal to the sum of the rate constant for physical removal ($r_{physical}$) plus the rate constant for chemical reaction ($1/r_{chemical}$). This relationship follows from the nature of first-order reaction kinetics, and is known to be only an approximate description of actual physical processes (see Exhibit 8-5). It is a useful approximation, however, that can be used to evaluate the importance of atmospheric processes for many pollutants.

As noted in Section 8.2.2, organic chemical pollutants can undergo a number of chemical reactions that may be important under different sets of conditions. Thus, the atmospheric lifetime for chemical reactions in the above equation is often broken down to consider contributions from each important reaction:

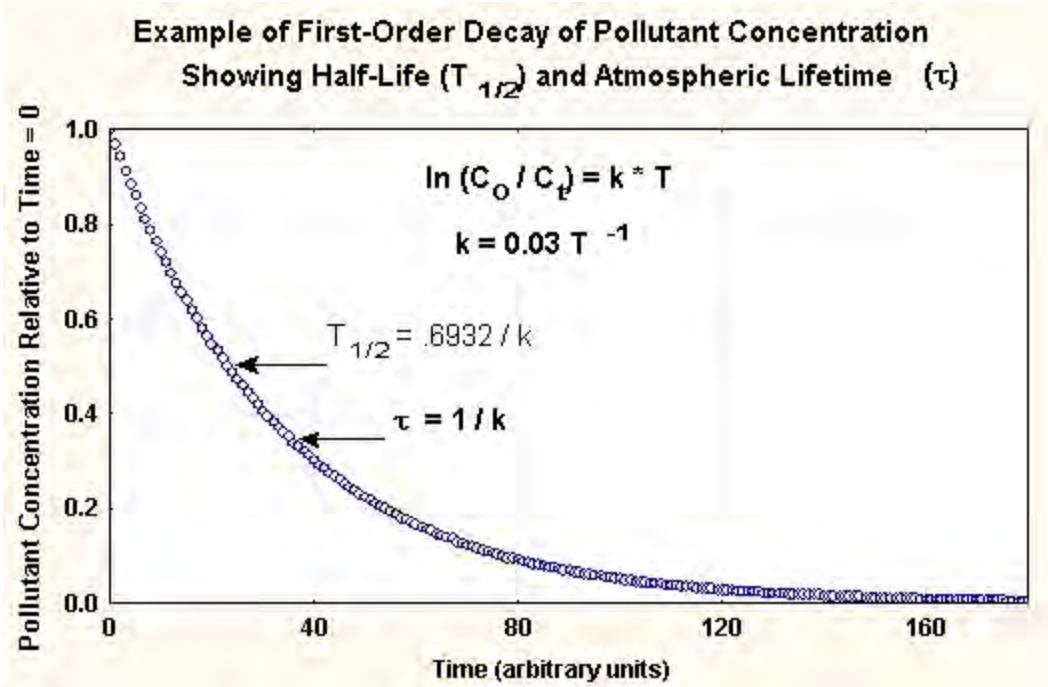
$$\frac{1}{r_{chemical}} = \frac{1}{r_{OH}} + \frac{1}{r_{NO3}} + \frac{1}{r_{O3}} + \frac{1}{r_{photolysis}} \qquad \text{(Equation 8-2)}$$

where the terms on the right side of the equations represent the rates of pollutant removal through the reactions with hydroxide radical, nitrate radical, ozone, and photolysis, respectively.

For any given air toxic, overall persistence in the atmosphere depends on particle-vapor partitioning behavior, particle size distribution (if the material is non-volatile), and susceptibility

to various types of chemical reactions. Atmospheric half-lives due to deposition (wet and dry) tend to be highly variable depending on particle size, ranging from a few minutes for coarse particles to many days for very fine particles. Most fine particles (less than a few microns) are removed from the troposphere (the lower level of the atmosphere where most weather takes place) with an average lifetime of between 5 and 15 days.

Exhibit 8-6. Example First-Order Decay of Pollutant Concentration



Simple physical and chemical reactions often proceed according to what are called "first-order kinetics." In a first-order reaction, the rate of the reaction at any given time is proportional to the concentration of one reactant (in this case, the air toxic that is being destroyed). The overall rate of the reaction is governed by the first-order (or "pseudo first-order") rate constant, "k." A higher rate constant implies a higher reaction rate for a given concentration of reactant. First-order reactions have the properties that the "half-life" of the reaction (the time in which one-half of the original concentration of reactant is destroyed) is the same no matter what the initial concentration. Air pollution scientists also measure the "atmospheric lifetime" of pollutants, which is abbreviated as the Greek letter τ (tau) or the letter "r," which is equal to 1/k. In this example, the rate constant (k) represents the sum of both physical and chemical removal.

Exhibit 8-7 presents estimated *chemical* half-lives for a few example chemicals based on measured reaction rates. Estimated atmospheric lifetime for chemical reactions ranges from many thousands of hours for the least reactive chemicals to only a few hours for chemicals that are more reactive. As noted above, the lifetimes for reactions with the hydroxide radical (r_{OH}) are the shortest, indicating that this pathway is the most important for most of the chemicals in the table, at least during daylight hours. Reactions with ozone and nitrate radicals are much slower

for most of the chemicals. "Pure" photolysis is not important for most chemicals, with the notable exceptions of the two aldehydes, where it is a major degradation pathway.[4]

Exhibit 8-7. Typical Atmospheric Half-lives for Chemical Reactions (hours) for Selected Pollutants for Reactions with Hydroxide, Nitrate, Ozone, and for Photolysis

Compound	r_{OH}	r_{O3}	r_{NO3}	$r_{photolysis}$
methane	28,000	$2x10^8$	$2x10^6$	–
ethane	8,40	$3x10^7$	$1x10^5$	–
benzene	180	$4x10^6$	$4x10^6$	–
toluene	37	$3x10^5$	$3x10^5$	–
1-butene	7.4	26	93	–
isoprene	2.3	20	1.4	–
formaldehyde	19	–	–	5.5
acetaldehyde	12	5,600	–	2.7

Source: California Air Resources Board Toxic Air Contaminant Fact Sheets[4]

Data such as those in Exhibit 8-7 are available to some degree for many chemicals, and can help assessors to judge the distance scales over which to analyze air toxics impacts. As noted above, persistence on the order of less than a day suggests transport of about ten miles, while persistence for several days suggests regional transport (500-1,000 miles) before being substantially degraded. Atmospheric reactivity is not well-studied for some chemicals, requiring the use of assumptions about persistence that span a reasonable range of reactivity. For non-volatile air toxics that partition primarily into particles, physical processes (wet and dry deposition) may be the most important in determining overall atmospheric lifetimes.[4] Some chemicals that are very persistent in the atmosphere and in terrestrial and aquatic systems may require special consideration, as described in Chapter 17. Chapter 9 builds upon the discussion above and identifies how well available dispersion models address chemistry/physical removal.

References

1. U.S. Environmental Protection Agency. 2003. 2001 Toxics Release Inventory (TRI) Public Data Release Report. Office of Environmental Information, Washington, D.C., July. Available at: http://www.epa.gov/tri/tridata/tri01/index.htm.

2. U.S. Environmental Protection Agency. 2004. Technology Transfer Network. *Support Center for Regulatory Air Models*. Updated February 23, 2004. Available at: http://www.epa.gov/ttn/scram/. (Last accessed March 2004).

3. Rosenbaum, A.S., Ligocki, M.P., and Wei, Y.H. 1998. *Modeling Cumulative Outdoor Concentrations of Hazardous Air Pollutants, Volume 1: Text*. SYSAPP-99-96/33r2, Prepared for U.S. Environmental Protection Agency, Office of Policy, Planning and Evaluation, by Systems Applications International, Inc., San Rafael, CA.

4. California Air Resources Board. 1998. *Toxic Air Contaminant Fact Sheets*. Available at: http://www.arb.ca.gov/toxics/tac/tac.htm. (Last accessed March 2004).

Chapter 9 Assessing Air Quality: Modeling

Table of Contents

9.1 Introduction 1

9.2 Air Quality Modeling 1
- 9.2.1 The Overall Structure of an Air Quality Model 1
- 9.2.2 Types of Models: Scientific Principles 6
- 9.2.3 Modeling Deposition 7
- 9.2.4 Screening vs. Refined Models 8
- 9.2.5 Specific Data Required for Modeling 9
- 9.2.6 Sources of Air Quality Models and Information 11
- 9.2.7 Examples of Air Quality Models 12
- 9.2.8 Emissions from Soil 18

9.3 Air Quality Modeling Examples 18

References 19

9.1 Introduction

Models have been used for decades to approximate physical systems and make estimates about the nature of the system under study. The types of models most frequently used in air toxics exposure assessments are mathematically-based models, which attempt to approximate all of the important physical and chemical processes affecting contaminant fate and transport within the environment. The physical and chemical processes are described as a set of mathematical expressions which characterize the behavior of contaminants released into the environment.

One specific type of model, called an **air quality model**, is used by EPA to understand the impact of pollution on air quality for a variety of purposes. For example, under the Clean Air Act (CAA), EPA uses air quality models to facilitate the regulatory permitting of industrial facilities, demonstrate the adequacy of emission limits, and project conditions into future years. For several of the criteria pollutants, regulatory requirements call for the application of air quality models to evaluate future year conditions as part of State Implementation Plans to achieve and maintain the National Ambient Air Quality Standards (NAAQS). Model simulations are also used to assist in the selection of monitoring locations.

Air quality models, when combined with emissions inventory and meteorological data, can be used as part of risk assessments that may lead to the development and implementation of regulations or voluntary reduction measures. For example, under National Air Toxics Assessments (NATA), EPA has conducted a national-scale assessment using air quality models for some 33 priority air toxics (see Chapter 2) to identify broad national air toxics issues and to help focus efforts. This Chapter provides an overview of air quality modeling used in air toxics risk assessments.

9.2 Air Quality Modeling

A variety of methods, data, and tools used for modeling the fate and transport of air toxics released to the environment have been developed; for a summary of methods, the reader can refer to Chapter 3 and other parts of EPA's *Residual Risk Report to Congress*.[1] While the Report to Congress is oriented toward assessment of residual (i.e., post-Maximum Achievable Control Technology [MACT]) risks from facilities regulated by the Clean Air Act, it also provides a good, general overview of general modeling procedures for air toxics assessments at the local scale. Another key reference for air quality models is the EPA's Support Center for Regulatory Air Models (SCRAM) website (http://www.epa.gov/ttn/scram/).[2]

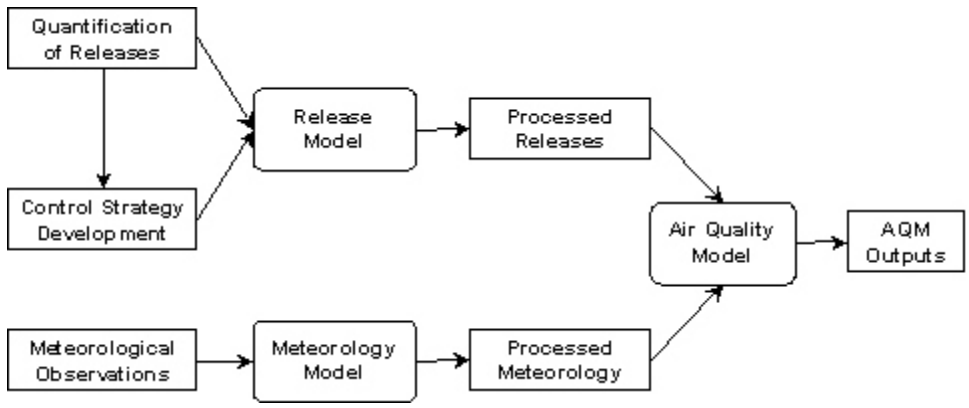
9.2.1 The Overall Structure of an Air Quality Model

Air quality models provide estimates of ambient air concentrations and/or deposition rates for one or more chemicals emitted from one or more sources. All air quality modeling systems are comprised of three major components (see Exhibit 9-1) which, when combined, provide a picture of predicted fate and transport of air toxics once released into the environment:

- An emissions (release) model (Chapter 7 discusses developing the emissions inventory);
- A meteorology model (Chapter 8 discusses atmospheric phenomena and physical properties that affect the fate and transport of air toxics after release); and

- An air quality model that predicts the movements of chemicals through the atmosphere along with any physical and chemical changes that may occur (e.g., chemical reactions that degrade the pollutant).

Exhibit 9-1. Basic Components of an Air Quality Modeling System



Specifically, the emissions and meteorology data are fed into the model (or the various components of the model) which are then run through various algorithms that simulate the physical and chemical processes in the atmosphere to provide estimated concentrations of chemicals (e.g., for inhalation exposure assessment, the exposure concentration at the point of exposure). Depending upon the specific model application being used, the release and meteorological data may simply be input to a single air quality model that includes both release and meteorological modules or the release and meteorological modules may be separated initially to "pre-process" the data and subsequently combined for the remaining calculations.

Air quality models provide estimates of ambient air concentrations at specific points distant from the source(s) being modeled. These are either predetermined within the model or selected by the analyst. In the simplest models (e.g., SCREEN3), the points are laid out along a vector (straight line) from the source. Many other models use a grid system to calculate ambient concentrations at specific exposure points at specified "nodes"(see Exhibit 9-2). The model does not always automatically provide an estimate of concentration at every desired location, and extrapolation to desired locations is often required. A discussion of where and how to choose exposure points is provided in Chapter 11.

Air Toxics Modeling Issues

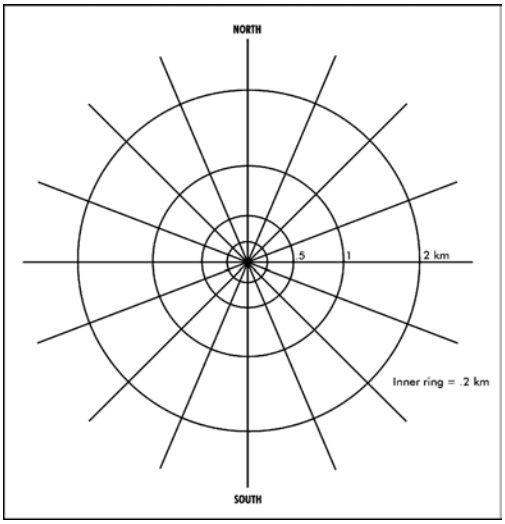
A recent study identified several issues that affect uncertainties associated with air toxics modeling, including:

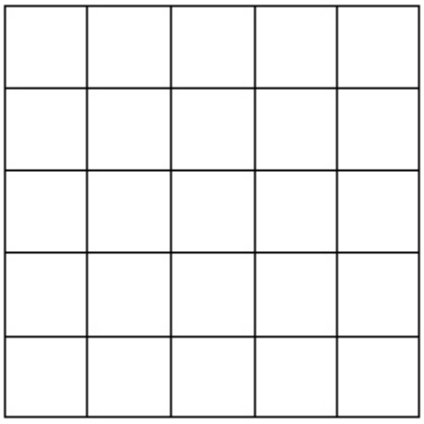
- Uncertainties associated with emissions;
- Meteorological conditions that are difficult to simulate (e.g., calm conditions, complex terrain, land/sea breezes, precipitation events);
- Spatial coverage, temporal resolution, and detection limits in monitoring data;
- Chemical transformations in the atmosphere;
- Removal via dry and wet deposition;
- Indoor sources; and
- Population activity patterns.

The study recommended a combination of modeling and monitoring for air toxics exposure assessments. For further information, see:

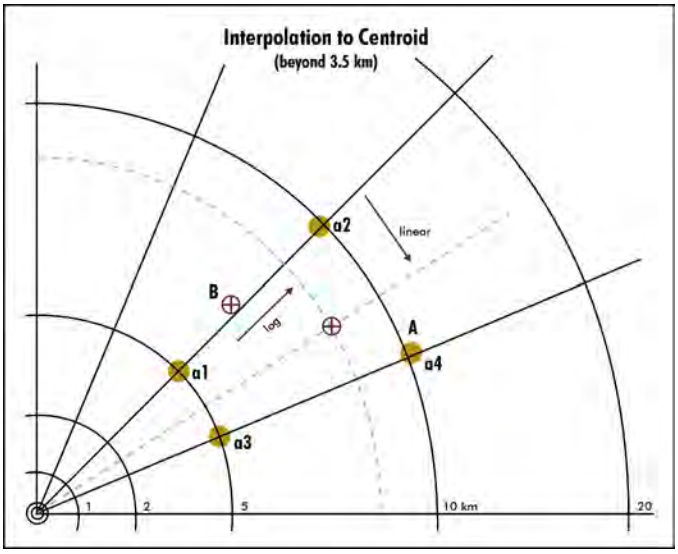
Coordinating Research Council and U.S. Department of Energy. 2002. *Critical Review of Air Toxics Modeling*, August 2002. CRC Project Number A-42-1, available at: http://www.crcao.com.

Exhibit 9-2. Model Grids and Interpolation





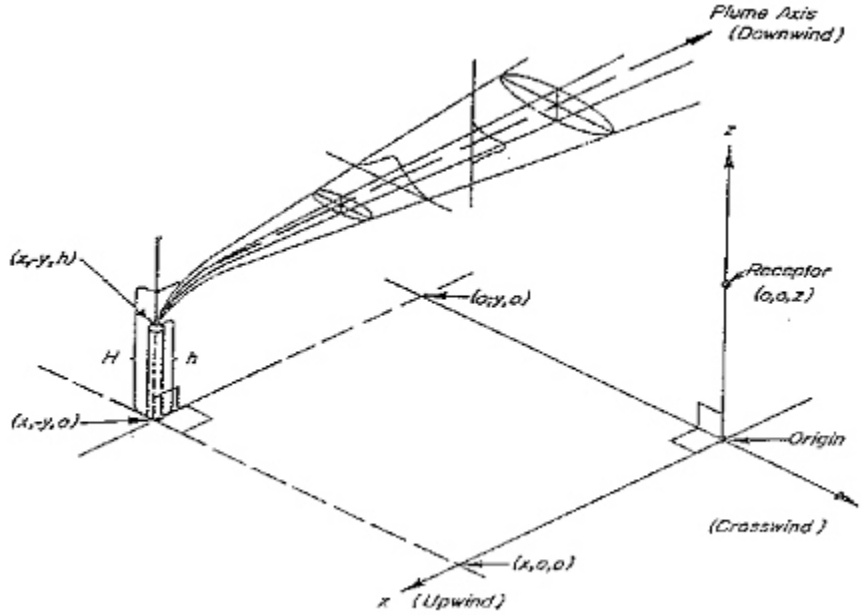
Many air quality models calculate ambient concentrations at specific exposure points at specified "nodes" using either a polar coordinate grid system (i.e., the intersections of a series of concentric circles and radial lines [above, left]) or on a standard Cartesian coordinate system (above, right). (Note that the nodes, in both of these types of grids, are simply the points where two lines intersect.) The locations of these nodes often do not fall precisely on the locations of interest for a given risk assessment.



In cases where the nodes and locations of interest do not align, a process of interpolation is used to estimate the ambient air concentration at the location. For polar grids, a two-step interpolation is used, starting with the modeled concentrations at the nearest locations (e.g., a1, a2, a3, and a4 in the graph to the left). The first interpolation is in the radial direction (i.e., along the two adjacent radial lines [a1,a2] and [a3, a4] in the graph). The concentration is estimated at the intersection of each radial line with the concentric circle that intersects the receptor location (at the same radial distance from the source as the internal point). This interpolation is performed under the assumption that the logarithm of the concentration decreases in proportion to the increase in the logarithm of the distance from the source (i.e., a log-log interpolation). The second interpolation is in the azimuthal direction (i.e., along the concentric circle that intersects the internal point). This interpolation is performed under the assumption that the change in concentration is proportional to the distance around the circle between the two radial lines (i.e., linear interpolation).

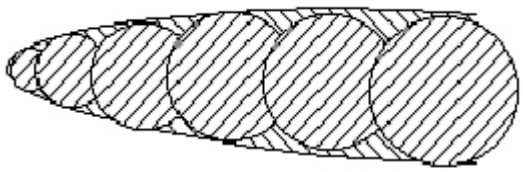
Illustrations of Three Common Types of Air Quality Models

Gaussian Plume Models: Model a continuous release downwind from a source

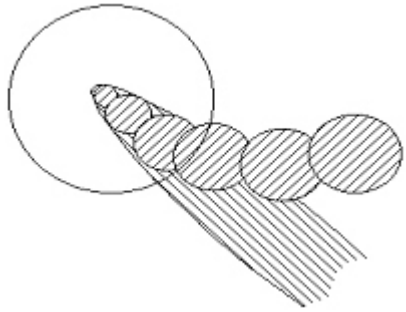


Gaussian plume models estimate the transport and mixing of pollutants in the dispersing plume as it moves downwind from the source. They assume that dispersion in the vertical and lateral dimensions will take the form of a normal Gaussian curve, with the maximum concentration at the center of the plume.[a]

Gaussian Puff Models: Model either Steady-state or Non-steady state releases



Steady-State Approach: Plume = Puff



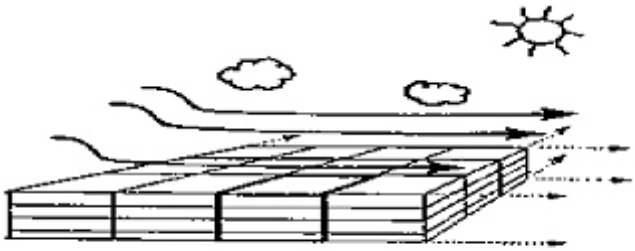
Non Steady-State Approach: Puffs follow Air

Puff models use a series of overlapping puffs to represent emissions. As shown by the illustration of the non-steady state approach, changes in wind direction over time and through space bring about changes in the plume's shape.[b]

Illustrations of Three Common Types of Air Quality Models (continued)

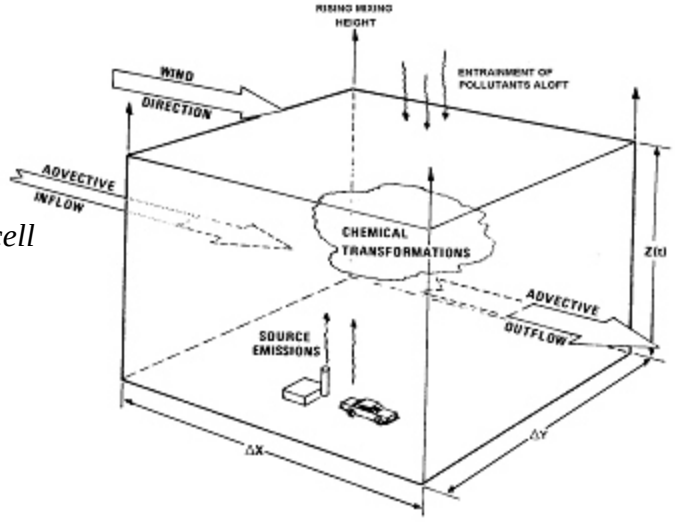
Numerical Grid Models: Model reactive pollutants in complex topography

Modeling Domain



Numerical grid models assume that emissions from area and line sources are mixed throughout the volume of each surface cell within the modeling domain. Emitted species react with each other and the incoming solar radiation with resulting chemical reactions taking place. Point source emissions, typically emitted from elevated stacks, are emitted into upper layers of the modeling domain based on a plume rise calculation. The point source emissions are then mixed throughout the volume of the elevated layer. Some models may modify this widespread dispersal by including a plume in grid module which acts to minimize the instantaneous mixing across the grid cell volume. These reactions are simulated to generate volume-average concentrations as a function of time within each cell.[c] The cells of the grid, representing discrete portions of the atmosphere, are superimposed on the modeling domain. [d]

Individual surface cell



[a] U.S. Environmental Protection Agency. 1970. Office of Air Programs, prepared by Turner, D.B. *Workbook of atmospheric dispersion estimates*. Publication AP-26. NTIS PB 191 482.

[b] National Oceanic and Atmospheric Administration. 2003. Prepared by Irwin, J.S. *Modeling Air Quality Pollutant Impacts*. Research Triangle Park, NC, 15 Oct. 2003. Available at: http://www.meteo.bg/EURASAP/40/paper1.html .

[c] U.S. Environmental Protection Agency. 1984. Office of Research and Development, prepared by Schere, K.L. and Demerjian, K.L. *User's guide for the photochemical box model (PBM)*.. Research Triangle Park. EPA-600/8-84-022a

[d] Systems Applications International. 1991-1993. Urban Airshed Modeling National Training Workshops.

Results from air toxic modeling are highly dependent upon the quality of data used as input to the models. The degree to which a user has reliable information on releases, meteorology, and setting will determine the accuracy of the modeled concentrations. Because model inputs are only estimates, even the most sophisticated models will have inherent uncertainties and will have the potential to underestimate or overestimate actual concentrations. (Monitoring data can assist in this regard as a way of evaluating the modeled results and to look for important gaps in the emissions inventory – see Chapter 10).

The uncertainty associated with the meteorology data includes measurement of key variables of wind speed/direction and atmospheric stability, and to a lesser extent, temperature and precipitation. Uncertainty is also associated with the terrain specification. Use of a model designed for flat terrain will likely provide inaccurate estimates of concentrations if the terrain is actually more complex (e.g., a facility located in a river valley modeled as being located on flat terrain).

In addition to the model inputs, uncertainties also arise from the model formulation used to describe the physical and chemical processes that take place in the atmosphere. In general, models are most accurate in simulating long-term averages of ambient concentrations and deposition rates in settings with simple topography.

9.2.2 Types of Models: Scientific Principles

In general, air quality models can be categorized as one of two types: **steady-state** and **non-steady state** models. The movement of mass away from the source (i.e., **advection**) and **turbulent diffusion** (e.g., dispersion) are modeled in both types of models. The steady-state model assumes that no variations occur over a certain time period (typically, one-hour); the non steady-state allows time-varying changes, but this capability imposes the need for additional model inputs, increased computation resources, and increased model formulation complexities. For additional information on air dispersion modeling, refer to NOAA's Real-time Environmental Applications and Display sYstem (READY) website.[3]

- **Steady-state models** are models which assume no time-varying processes occur over the period of interest. Hence, material released travels infinitely in only one direction over the time period (e.g., one hour). Often, these models assume that the material is distributed normally (also termed a "Gaussian distribution") and are thus called "Gaussian plume" models (see illustration above). The steady-state model typically uses meteorological information obtained near the source and assumes it holds true throughout the modeling region (e.g., a 50 kilometer radius). Wind direction, wind speed, and atmospheric stability are used to predict concentrations. This type of model is most widely used for stationary sources and for non-reactive pollutants (although models can take into account deposition and simple linear decay). The models are least applicable in areas with rapid time-varying conditions, over spatially varying terrain and land use, over large spatial scales (> 50 km), and where complex atmospheric chemistry takes place.

- **Non-steady state models** are models which can simulate the effects of time- and space-varying meteorological conditions on pollutant transport, transformation, and removal. The modeling region is typically divided into grid cells, and the model simulates movement of pollutants between cells by taking into account advection, degradation, and other physical

and chemical processes. These models are often used for chemically reactive pollutants or where there is complex topography or meteorology (e.g., complex sea breeze circulation). They require complex wind flow characterization and other detailed meteorological information for dispersion. For chemical transformation, they require information on the important chemical compounds as well as chemical kinetics to properly characterize the transformation and removal of air toxics. These models often take the form of grid models with the calculation of the physical and chemical processes taking place at each grid location. Other model types include "puff models" (illustrated above), which use a series of overlapping puffs to represent emissions. The calculations of the physical and chemical processes are made for each "puff."

Another type of non-steady state model, the atmospheric trajectory model, uses meteorological data and mathematical equations to simulate transport in the atmosphere. The position of a parcel of air with time are calculated based on externally provided meteorological data such as wind speed and direction, temperature, humidity, and pressure. Model results depend on the spatial and temporal resolution of the meteorological data used, and also on the complexity of the model itself. Simpler models may deal with only two-dimensional transport by winds assuming the material emitted into the parcel stays at the same level, while more complex models may include 3-dimensional chemical and thermodynamic processes such as aerosol formation, convection, and turbulent diffusion.

9.2.3 Modeling Deposition

Deposition is the transfer of chemicals from the plume to the earth's surface (i.e., to soil, water bodies, or living organisms such as plant surfaces). Although the primary route of exposure for many air toxics is inhalation of ambient concentrations, deposition rates can be important for the multimedia fate and transport assessments required for persistent bioaccumulative hazardous air pollutant (PB-HAP) substances (see Chapter 18). Air quality models all simulate ambient air concentrations, and many also simulate deposition. Based on the simulated ambient air concentration at a location, the **deposition flux** (i.e., mass of pollutant deposited per unit area) can be simulated based on a number of assumptions (see Chapter 8 for a discussion of mechanisms of deposition). Two types of deposition are usually modeled:

- **Dry deposition** is determined from the ambient air concentration and the deposition velocity. Particle-phase air toxics are the principal pollutants removed through dry deposition by particle settling. In addition, semi-volatile toxics (air toxics that exist both in the gas and particle phases) can also be removed through dry deposition. Dry deposition of some vapor-phase air toxics is also possible for some chemicals (e.g., divalent mercury).

- **Wet deposition** is determined from a combination of the ambient concentration and a **scavenging ratio**. The scavenging ratio accounts for the propensity of the modeled chemical to partition into precipitation in the atmosphere, based on physical and chemical characteristics of the pollutant, the nature of the precipitation (liquid or frozen), and the precipitation rate. The term "scavenger" is a general term that can apply to anything chemical or physical that removes a pollutant from the atmosphere. In this example, rain is a scavenger because it is removing (by dissolution) an air toxic from the atmosphere and transferring it to a surface.

9.2.4 Screening vs. Refined Models

The overall accuracy and precision of results determined by a model is generally proportional to the complexity of the model, which in turn affects input data requirements and overall resources.

- **Screening-level models** are designed to provide conservative (i.e., high) estimates, and are useful for applications such as identifying facilities and/or air toxics that appear likely to contribute the greatest risk among a group of sources and chemicals released. Data requirements are generally low (e.g., emission rates, some stack parameters), and running the models is generally easy and requires few resources.

- **Refined models** take into account more complex chemical behavior and a greater degree of site-specific information, generally producing more accurate results. Data requirements are higher (e.g., site-specific meteorology, terrain, chemistry data), and application of more refined models may require expert judgment in developing model inputs and setting model options. Some models can be used both as a screening model and refined model if additional site-specific information is used in the application.

The selection of a model for a specific application depends on a number of factors, including:

- The nature of the pollutant (e.g., gaseous, particulate, reactive, inert);
- The meteorological and topographic complexities of the area of concern;
- The complexity of the distribution of sources;

Exposure Concentrations: Units are Important

Air toxics exposure concentrations (ECs) should in general be reported as $\mu g/m^3$. Dose-response values often are reported as parts per million (ppm), parts per billion (ppb), or mg/m^3. In the risk characterization step, ECs are compared to dose-response values, and therefore the units for the EC must match the units for the dose-response values.

The conversion from mg/m^3 to ppm can be expressed as:

Concentration [ppm] = Concentration [mg/m^3] × 24.45 [L/mole] / MW

and the conversion from ppm to mg/m^3 is:

Concentration [mg/m^3] = Concentration [ppm] × MW / 24.45 [L/mole]

where MW is the molecular weight of the air toxic in g/mole and 24.45 is the volume in liters of one mole of an ideal gas at 1 atmosphere and 25 degrees Celsius.

Note also that ppb = 1,000 × ppm and that here, ppm is volume-based. Also, $\mu g/m^3$ = 1,000 × mg/m^3.

Tip: In the development of the analysis plan, stipulate that all laboratory and modeling results be reported in $\mu g/m^3$. This will save time (and reduce computational errors) in the remaining phases of the risk assessment.

- The spatial scale and temporal resolution required for the analysis;
- The level of detail and accuracy desired for the study and the amount of uncertainty that the analyst/risk manager is willing to accept; and
- The technical expertise of user.

For example, steady-state models are not considered appropriate for downwind distances beyond a 50 km range, primarily because the steady-state wind speed and direction over that distance become unrealistic over the typical one-hour simulation period. This is especially true where complex terrain or meteorology is present.

Because screening models are applied with fewer resources and data to provide conservative estimates of concentrations, screening models are often applied prior to any refined modeling in order to narrow the set of sources or air toxics to be modeled. Such an iterative approach is generally recommended by EPA, where screening results are used to generate a subset of potentially higher-risk sources or chemicals for more refined assessment. General guidance on screening-level modeling has been published by EPA.[4] Additional guidance on air modeling is incorporated into EPA's Guideline on Air Quality Models.[2]

Risk assessors generally work out the development of a modeling protocol to be used in the assessment during the planning/scoping and problem formulation phase of the assessment. Providing this protocol will help establish the modeling approach for not only review and comment by interested parties up front, but will help to establish technical credibility and provide for consensus building among all interested parties.

9.2.5 Specific Data Required for Modeling

As described above, meteorology, terrain, and emissions data are processed and used as primary input data for air quality models. Depending on the level of refinement of the model, the required input data for an air quality model will include (but not necessarily be limited to) the following parameters:

- **Emission rate**. In general, the rate at which emissions are released into the atmosphere are specified as a rate of release for each chemical in units of mass per unit time.

- **Physical/chemical characteristics of emissions**. These data are closely related to emission rates (i.e., from measurements and/or emission factors; see Chapter 7). For some models, the phase of emission must be specified (e.g., gas, particulate, or semi-volatile). For chemicals present as particulate matter or as semi-volatile substances, particle size distribution and fraction of particle phase as a function of temperature, for each chemical, may be necessary inputs. In some cases, information may only be available on the basis of total volatile organic compounds or total particulates. This information may be speciated based on the emissions source type through the use of sources such as EPA's SPECIATE database. (The most recent version of SPECIATE, Version 3.2, was last updated with new profiles in October 1999.) [5]

- **Type of release point**. The required input data, modeling approach, and model selected for assessment can depend on the type of release being modeled. Chapter 4 discussed types of sources from a regulatory perspective (e.g., stationary, mobile). The following discussion is focused on types of sources from a modeling perspective.

- **Point sources** (modeling sense) are releases from stacks and isolated vents, and typically have plume rise associated with the release due to the buoyancy or momentum of the effluent.
- **Area sources** (modeling sense) are sources which are usually low level or ground level releases with no plume rise (e.g., fugitive emissions from the summary of equipment leaks across a facility; uncontrolled emissions that escape from the windows along a building wall; releases of dust from a road or work site; slag dumps; storage ponds). Depending on the type of area source, the modeler may opt to evaluate it as emissions occurring from a two-dimensional surface (i.e., an area source in the modeling sense) or as a three-dimensional volume source (see below). If a large number of sources are to be modeled, a common approach is to spread these sources uniformly across the modeling domain if no appropriate spatial surrogate is available. Alternatively, these sources may be allocated based on spatial surrogates. Typical examples include census tract population and commercial, residential and industrial land-uses.
- **Volume sources** are releases that are modeled as emanating from a 3-dimensional volume (such as a box) . Examples include releases from conveyor belts or the collective releases from the gas pumps at service stations. Volume sources differ from area sources in that they have a vertical dimension to their release. Like area sources, they do not have plume rise.
- **Line sources** are releases that are modeled as emanating from a two-dimensional area. Examples include rail lines and roadway segments. Line sources differ from area sources in that they have aspect ratios (length to width) much higher than 10:1. Like area sources, they do not have plume rise.
- **Specialized release types** include multiple parallel release lines that result in increased buoyant dispersion (e.g., coke ovens, aluminum smelters); dense gas release; and exothermic gas release, jet-plume release and horizontal venting that may be defined and modeled using special techniques or models depending on the characteristics of the emission source.

- **Release point parameters**. Depending on the type of source being modeled, the user may need to specify the physical characteristics of the release point. Key parameters may include the following:
  - Release height above ground level (e.g., stack height, average height of fugitive emissions).
  - Area of the release point (for point sources, stack diameter; for area sources, length and width of the area across which releases occur).
  - Other stack parameters of the release stream for point sources that can alter the effective release height, which include temperature, stack orientation, the presence of obstructions to flow (i.e., rain caps), and exit velocity or flow rate. Flow rate is expressed in terms of the total volume of material released per unit of time. In general, most of the flow rate is made up of nontoxic exhaust gases, with a small fraction being composed of chemical contaminant.
  - Facility building dimensions, if building downwash (i.e., the effects on plume dynamics due to structures located near the source) is modeled.

- **Location of special receptors**. The location of known sensitive receptors (e.g., a school or day-care center) may be a critical input when determining where to model ambient concentrations. If these special receptor locations are not identified, the model will only

provide concentration estimates at the nodes of the modeling grid that is initially laid out around the source.

- **Information on the surrounding land-use and terrain heights**. For dispersion models, classification of the surrounding area as urban or rural is usually required (this classification can affect the rate of dispersion). In addition, more refined modeling that takes into account complex terrain (e.g., ground surfaces higher than release height elevation) will require terrain elevation data.

- **Chemical-specific data**. If transformation/removal is being modeled, rates of transformation or removal for the chemicals being modeled are required (transformation processes are discussed in Chapter 8).

- **Boundary or background concentrations**. Ideally, emissions from modeled source(s) are responsible for the modeled concentrations. However, background concentrations, or boundary conditions in the case of grid models, may be important contributors to the total concentrations. This is particularly relevant where modeled concentrations are compared to observed concentrations. There are three basic approaches to estimating background concentrations:

  – Default values based on supporting documentation from the literature (this is the simplest approach);
  – Data collected from monitoring stations within the study area; and
  – Estimates made from larger regional scale models that cover the study area.

  For grid type models, users should be aware that with a smaller modeling domain, there is more potential for the boundary concentrations to play a more important role in determining the total concentration.

In general, air quality modeling results will be most sensitive to the emission rate when studying a single or few release points. However, when studying multiple release locations over a broad area, source location becomes the most important parameter. For a Gaussian-type dispersion model (e.g., ISC3, AERMOD; see Section 9.2.7 below), the ambient concentration will be directly proportional to the emission rate (enabling the use of unit emission rates). Other inputs, especially stack height and distance to fenceline, can also affect the results because these parameters can have a direct impact on the location of higher ambient chemical concentrations and potential off-site receptors. In general, however, the sensitivity of air modeling results to specific input parameters can vary widely according to site-specific and chemical-specific factors. Site-specific analyses are generally required to derive accurate sensitivity results for a specific air modeling application. Additional discussion on sensitivity analysis can be found at the EPA Region 6 Air Modeling for Combustion Risk Assessments website.[6]

9.2.6 Sources of Air Quality Models and Information

Numerous models (both screening and refined) have been developed by EPA, other government agencies, and private sources. EPA models in particular undergo extensive evaluation and statistical measures of performance. Some private industry models are also available to the user at little or no charge. (If a public domain model is not available and a private model must be

used, the user should request information about the theoretical basis for the model and the result of any peer review.) Important sources of information include EPA's *Guideline on Air Quality Models*[2] and *Dispersion Modeling of Toxic Pollutants in Urban Areas: Guidance, Methodology and Applications*.[7] Both are available at EPA's SCRAM website (http://www.epa.gov/ttn/scram/), EPA's primary resource for Agency air modeling information.[2] At the SCRAM site, EPA maintains an up-to-date collection of the executable files, source codes, and user guidance for EPA air quality models. The EPA Office of Air Quality Planning and Standards maintains an on-line Air Pollution Training Institute (APTI) that is managed by the Education and Outreach Group (EOG) and offers additional information and training opportunities for air quality modelers.[8]

9.2.7 Examples of Air Quality Models

A variety of models are available for air toxics risk assessments, with some models having been designed for specific air toxics application. The SCRAM website provides detailed information regarding individual models, including software/code for each model, user's manuals, and other support documentation.

The extent to which a specific air dispersion model is suitable for the evaluation of air toxic source impacts depends upon several factors, such as the nature of the pollutant (e.g., gaseous, particulate, reactive, inert), the meteorological and topographic complexities of the area, the complexity of the source distribution, the spatial scale and resolution required for the analysis, and the level of detail and accuracy required for the analysis. For example, steady-state Gaussian plume models are not considered appropriate for downwind distances outside of the 0.1 km to 50 km range. Because of the assumption in Gaussian models of a steady wind speed and direction over the entire modeling domain for each hour, a > 50 km distance may be inappropriately long in many areas, especially where complex terrain or meteorology is present. In such cases, a non-steady state model would be more appropriate.

Exhibit 9-3 provides an overview of the key physical processes simulated in the most widely used air quality models oriented toward assessment of risks from facilities. Exhibit 9-4 shows the spatial and temporal scales over which these air quality models are typically applied. Exhibit 9-5 identifies some common applications for these air quality models.

> The Draft Guidance on the Development, Evaluation, and Application of Regulatory Environmental Models recommends best practices to help determine when a model, despite its uncertainties, can be appropriately used to inform a decision. The Knowledge Base (KBase) is a web-accessible database of information on some of EPA's most frequently used models. The draft guidance recommends what information about models to document, while the Knowledge Base is the repository where this information is documented. Both products are available at the CREM internet site at http://www.epa.gov/crem.

Finer scale models, such as CAL3QHC and CALINE4, are most typically applied to exposure studies from mobile sources. The UAM-TOX and CMAQ models are examples of models which can simulate photochemically active air toxic species, including secondary formation of pollutants like formaldehyde. Because the complex secondary formation processes are nonlinear and can occur at locations distant from the emission source, these models are designed to be applied to an exhaustive set of sources over a large region, rather than to individual facilities or small groups of facilities. The models more typically applied to single or

multiple facilities include SCREEN3, ISCST3, ISCLT3, AERMOD, ASPEN, CALPUFF, and UAM-TOX. Brief descriptions of these models are provided below. Some modeling studies have combined the application of a regional model with a neighborhood-scale model in order to address secondary and background concentration contributions, while capturing finer spatial resolution for primary pollutant predictions.

Community Multi-scale Air Quality (CMAQ) Modeling System

The CMAQ modeling system has been designed to approach air quality as a whole by including state-of-the-science capabilities for modeling multiple air quality issues, including tropospheric ozone, fine particles, toxics, acid deposition, and visibility degradation. In this way, the development of CMAQ involves the scientific expertise from each of these areas and combines the capabilities to enable a community modeling practice. CMAQ was also designed to have multi-scale capabilities so that separate models were not needed for urban and regional scale air quality modeling.

The target grid resolutions and domain sizes for CMAQ range spatially and temporally over several orders of magnitude. With the temporal flexibility of the model, simulations can be performed to evaluate longer term pollutant climatologies as well as short term transport from localized sources. With the model's ability to handle a large range of spatial scales, CMAQ can be used for urban and regional scale model simulations. By making CMAQ a modeling system that addresses multiple pollutants and different spatial scales, CMAQ has a "one atmosphere" perspective that combines the efforts of the scientific community. Improvements will be made to the CMAQ modeling system as the scientific community further develops the state-of-the-science. Additional information about CMAQ can be found at: http://www.epa.gov/asmdnerl/models3/cmaq.html.

SCREEN3

- Screening-level Gaussian dispersion model that estimates an hourly maximum ambient concentration based on an average, constant emission rate (concentration results can be scaled up to annual average using simple conversion factors as specified in EPA guidance;[4] results are not direction-specific (i.e., wind direction is not taken into account).

- Data requirements are relatively low; uses site-specific facility data (e.g., stack height, diameter, flow rate, downwash); does not use site-specific meteorology data.

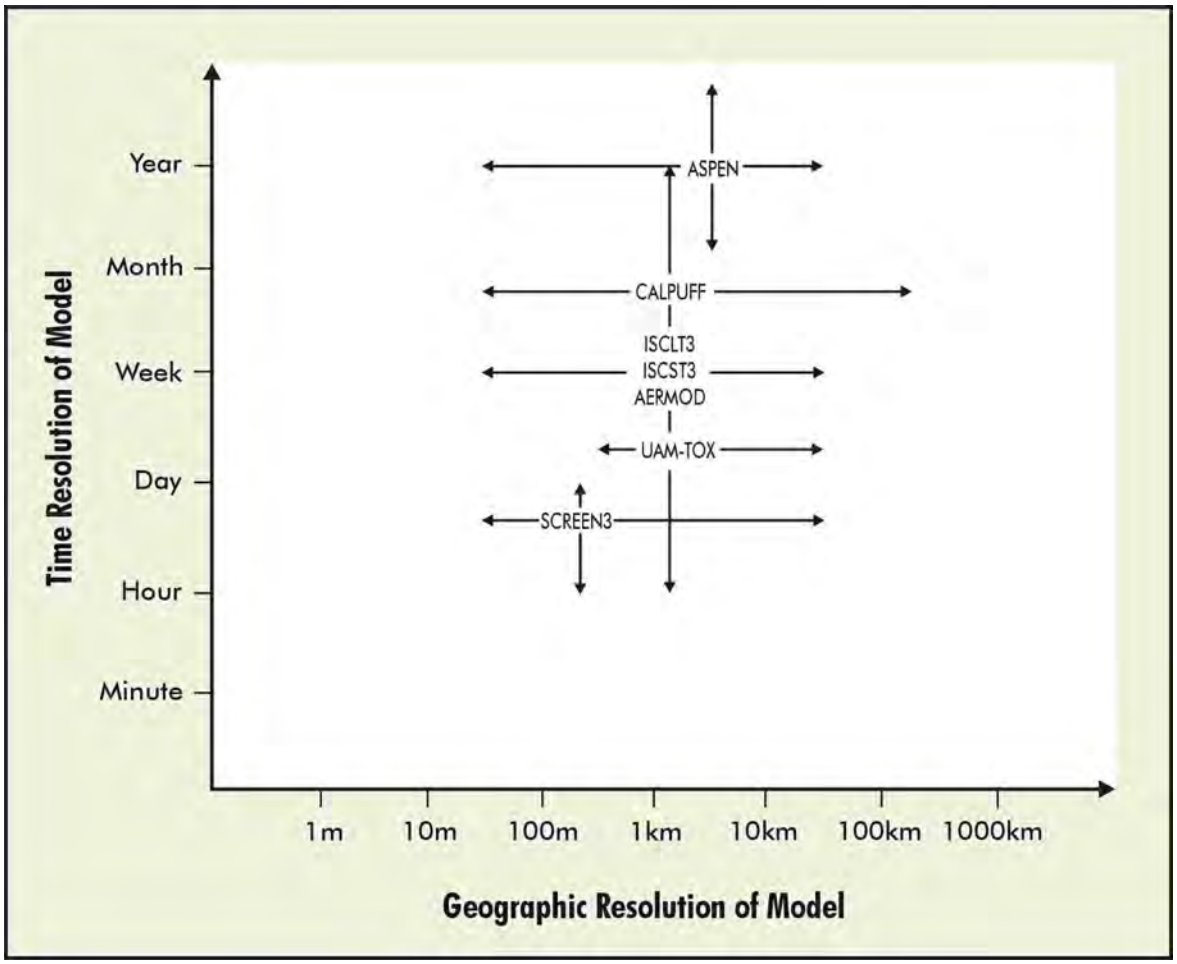
- Data processing requirements are low; easy to use for quick assessment of a single facility.

- Model does not estimate deposition rates.

Exhibit 9-3. Key Modeling Attributes of Some Widely Used Air Quality Models for Residual Risk Assessment							
Modeling Attribute	**SCREEN3**	**ISCST3**	**ISCLT3**	**AERMOD**	**ASPEN**	**CALPUFF**	**UAM-TOX**
Point	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Volume	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Area	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Meteorology	Built-in worst-case meteorology	Hourly (National Weather Service) or site-specific equivalent	Frequency array of meteorology data	Hourly (National Weather Service) or site-specific equivalent	Multiple hourly observations (National Weather Service or site-specific equivalent	Hourly user-defined 3-D fields, usually from a meteorological model with multiple meteorological stations	Hourly user-defined 3-D fields, usually from a meteorological model with multiple meteorological stations
Wet Deposition	No	Yes	No	Yes*	Yes	Yes	Yes
Dry Deposition	No	Yes	Yes	Yes*	Yes	Yes	Yes
Complex Terrain	Yes	Yes	No	Yes	No	Yes	Yes
Overwater Effects	No	No	No	No	No	Yes	No
Vertical Wind Shear	No	No	No	Yes	No	Yes	Yes
Building Downwash	Yes	Yes	Yes	Yes	Yes	Yes	No
Model Formulation and Plume Distribution	Steady-state, Gaussian	Steady-state, Gaussian	Steady-state, Gaussian sector average	Steady-state Gaussian stable & neutral conditions, bi-Gaussian in unstable conditions	Steady-state, Gaussian sector average	Non-steady-state, Gaussian puff	Non-steady-state grid model
Chemical Transformation	None	Simple decay	Simple decay	Simple decay (SO_2)	Difference between precursor inert and precursor decay	Simple psuedo-first-order effects	Complete chemical mechanism for most gas-phase toxics
Relative Complexity	Simple	Moderate	Moderate	Moderate	Moderate	Complex	Complex

*AERMOD version 02222 is now available for review and comment on EPA's SCRAM website (http://www.epa.gov/scram001/).
This version includes algorithms for dry and wet deposition as well as an improved downwash algorithm known as PRIME.

Exhibit 9-4. Spatial and Temporal Scales of Widely Used Air Quality Models



This figure illustrates the geographic and temporal resolution of several widely used air quality models. For example, the screening-level model SCREEN3 has a spatial resolution of 50 m to 50 km, but a temporal resolution of 1-24 hours. In contrast, ISCST3 has the same spatial resolution (50 m to 50 km), but has a temporal resolution from 1 hour to 1 year.

Industrial Source Complex - Short Term (ISCST3)

- Gaussian dispersion model (more advanced than SCREEN3); estimates average annual ambient concentration by modeling hourly emissions, and meteorology includes removal effects for wet and dry deposition flux for any locations specified by the user.

- Data requirements are higher than for SCREEN3; requires hourly, site-specific, processed meteorological data, physical characteristics of emissions, and terrain information. Model can accommodate variable emission rates.

- More expertise is required to use model (compared to SCREEN3); user should possess specific technical and computer skills.

Exhibit 9-5. Typical Applications for Common Dispersion Models						
	Averaging Period	**Terrain Type**	**Single Source**		**Multiple Sources**	
			Rural	**Urban**	**Rural**	**Urban**
Screening Models	Short Term (1-24 hour average)	Simple	SCREEN3	SCREEN3	ISCST3, AERMOD	ISCST3, AERMOD
		Complex	SCREEN3, ISCST3	SCREEN3, ISCST3	ISCST3	ISCST3
	Long Term (Monthly-Annual)	Simple	ISCLT3	ISCLT3	ISCLT3, ASPEN	ISCLT3, ASPEN
		Complex	ISCST3	ISCST3	ISCST3	ISCST3
Refined Models	Short Term (1-24 hour average)	Simple	ISCST3, AERMOD	ISCST3, AERMOD	ISCST3, AERMOD	ISCST3, AERMOD, UAM-TOX
		Complex	AERMOD, CALPUFF	AERMOD, CALPUFF	AERMOD, CALPUFF	AERMOD, UAM-TOX, CALPUFF
	Long Term (Monthly-Annual)	Simple	ISCST3, AERMOD	ISCST3, AERMOD	ISCST3, AERMOD	ISCST3, UAM-TOX, AERMOD
		Complex	CALPUFF, AERMOD	CALPUFF, AERMOD	CALPUFF, AERMOD	CALPUFF, UAM-TOX, AERMOD

Industrial Source Complex - Long Term (ISCLT3)[(a)]

- Similar to ISCST3, but uses seasonal frequency distribution of meteorological inputs rather than hourly data; runs more rapidly than ISCST3, but can only produce concentrations averaged over a relatively long period of time; not considered as accurate as ISCST3.

- Unlike ISCST3, it cannot simulate wet deposition or complex terrain (terrain higher than the stack height).

[a]EPA is no longer actively updating the model with improvements or additional capabilities. It still is one of EPA's preferred models and can be used in appropriate situations. For most single or limited source applications, the ISCLT3 model can be used without any overwhelming computational burden.

AMS/EPA Regulatory Model (AERMOD)

- Replacement model for ISCST3 using new or improved algorithms on the parameterization of the earth's boundary layer turbulence and state-of-the-science dispersion modeling; deposition algorithms should be available soon.

- Like ISCST3, is a Gaussian formulated model.

- Similar to ISCST3, but includes dispersion algorithm for both convective and stable boundary layers and allows plume penetration into elevated inversions.

- Incorporates new algorithms for building downwash.

- Unlike ISCST3, it simulates vertical profiles for wind, turbulence, and temperature.

- No wet or dry deposition (although planned future improvement).

- Requires surface characteristics as inputs (e.g., albedo, Bowen ratio, surface roughness), which allow user to differentiate between different types of terrain.

ASPEN

- A Gaussian dispersion model used to estimate toxic air pollutant concentrations over a large scale domain from regional to continental scale. (This is the model used for NATA risk characterization analyses.)

- Employs a dispersion algorithm similar to ISCLT3.

- However, unlike ISCLT3, it includes treatment of wet deposition for particles, and more detailed treatment of chemical transformation than ISCLT3 or ISCST3, although less detailed than UAM-Tox.

- In contrast to ISCLT3, ASPEN can utilize meteorological information from several locations, and includes a simplified treatment of secondary formation of gaseous air toxics.

CALPUFF

- A Gaussian puff model designed for long-range transport (> 50km) assessment, but may also be applied for near-source in situations with complex meteorology. As described previously, a puff represents a continuous plume as a number of discrete packets of pollutant material.

- Has all the functional capabilities of ISCST3, but also includes capabilities for including 3-dimensional wind fields, vertical wind shear, and overwater effects.

- Not as extensively evaluated and tested as ISCST3 model.

- Requires a substantially higher level of air quality modeling expertise to use the model (compared to ISCST3).

UAM -Tox (Urban Airshed Model - Toxics Version)

- A three-dimensional, grid-type model used to model pollutants in urban areas. Derived from the Urban Airshed Model (UAM), designed to calculate ozone concentrations under short-term, episodic conditions lasting three to four days resulting from emissions of oxides of nitrogen (NOx), volatile organic compounds (VOC), and carbon monoxide (CO).

- Simulates the most photochemically active air toxics (i.e., acetaldehyde, 1,3-butadiene, and formaldehyde), as well as secondary formation of acetaldehyde and formaldehyde, tracking primary and secondary fractions separately.

- Requires a substantially higher level of air quality modeling expertise to use this model (compared to ISCST3).

9.2.8 Emissions from Soil

In addition to the air quality models described above, it is sometimes necessary to model emissions of chemicals from soil. Emissions from soil may occur as a result of the volatilization of chemicals from contaminated soil or as a result of the resuspension of study area soils. Models that predict emission rates for volatile chemicals or dust require numerous input parameters, many of which are study area-specific. For volatile chemicals, emissions models are available from several EPA sources.[9] Emissions due to suspension of soils may result from wind erosion of exposed soil particles and from vehicular disturbances of the soil. To predict soil or dust emissions, a number of modeling approaches have been developed. These include EPA's fugitive dust model for a site-specific assessment.[10] For road dust, other techniques are generally used.[11] After emissions have been estimated or measured, air dispersion models can be applied to estimate air concentrations receptor points.

In addition, chemicals in contaminated soils and groundwater may also evaporate into homes and buildings through cracks in the floor. The models used to assess these types of exposures (often called "basement models" because this type of problem can be exacerbated when a room is buried in the contaminated medium) are commonly used by hazardous waste site cleanup risk assessors to determine whether people living on or near contaminated sites are being adversely affected by chemicals evaporating into their living or working spaces. This type of analysis is less common for ambient air toxics risk assessment of the type that will generally be performed in an urban setting or in the evaluation of source impacts on nearby populations. However, this issue does come up on occasion and the topic is mentioned here for completeness.

One of the primary vapor intrusion models is the Johnson and Ettinger model (http://www.epa.gov/oerrpage/superfund/programs/risk/airmodel/johnson_ettinger.htm), and EPA has developed a users guide for evaluating vapor intrusion into buildings through the use of this model (http://www.epa.gov/superfund/programs/risk/airmodel/guide.pdf).

Another chemical, radon, is also an issue for homes and buildings in certain parts of the country (see Chapter 2). EPA's Indoor Environments Division (http://www.epa.gov/iaq/) provides a comprehensive set of informational materials on risks associated with radon and mitigation methods (see http://www.epa.gov/iaq/radon/pubs/).

9.3 Air Quality Modeling Examples

EPA's Air Toxics Community Assessment and Risk Reduction Projects Database has been compiled to provide a resource of planned, completed, and ongoing community-level air toxics assessments across the country. The projects included in the database provide examples of the applications of air quality modeling at real-world sites. Project descriptions and related information can be obtained from the database website at: http://yosemite.epa.gov/oar/CommunityAssessment.nsf/Welcome?OpenForm.

Additional Reference Documents

Although the list of following documents are now somewhat dated in terms of computational limitations for application of the models, the documents do provide overall methodology and guidance on procedures to consider when conducting air toxic modeling:

Guidance on the Application of Refined Dispersion Models for Hazardous/Toxic Air Releases, USEPA/OAQPS, Research Triangle Park, NC, EPA-454/R-93-002, May 1993.

Air/Superfund National Technical Guidance Study Series, Volume V - Procedures for Air Dispersion Modeling at Superfund Sites, EPA/OAQPS, Research Triangle Park, NC, February, 1994.

Dispersion Modeling of Toxic Pollutants in Urban Areas, Guidance, Methodology And Example Applications, EPA/OAQPS, Research Triangle Park, NC, EPA-454/R-99-021, July 1999.

Guidelines on Air Quality Models. 40 CFR Part 51 and Part 52, Appendix W; Environmental Protection Agency, AH-FRL-5531-6.

References

1. U.S. Environmental Protection Agency. 1999. *Residual Risk Report to Congress*. Office of Air Quality Planning and Standards, Washington, D.C., 1999. EPA/453/R-99/001.

2. U.S. Environmental Protection Agency. 1994. *Volume V - Procedures for Air Dispersion Modeling at Superfund Sites.* Air/Superfund National Technical Guidance Study Series. Office of Air Quality Planning and Standards, Research Triangle Park, NC, February 1994. Available at: http://www.epa.gov/scram001/tt25.htm#guidance. (Last accessed March 2004).

   U.S. Environmental Protection Agency. 1999. *Dispersion Modeling of Toxic Pollutants in Urban Areas.* Office of Air Quality Planning and Standards, Research Triangle Park, NC, July 1999. EPA/454/R-99/021. Available at: http://www.epa.gov/ttn/scram/. (Last accessed March 2004).

   U.S. Environmental Protection Agency. 2003. *Guideline on Air Quality Models*. *Federal Register* 40 CFR Part 51 Appendix W, April 15, 2003. Available at: http://www.epa.gov/scram001/tt25.htm#guidance.

3. National Oceanic and Atmospheric Administration. 2003. Air Resources Laboratory. Realtime Environmental Applications and Display sYstems. Updated December 12, 2003. Available at: http://www.arl.noaa.gov/ready.html (Last accessed March 2004).

4. U.S. Environmental Protection Agency. 1992. *Screening Procedures for Estimating the Air Quality Impact of Stationary Sources, Revised*. Office of Air and Radiation. Research Triangle Park, NC. EPA/454/R-92/019. Available at: http://www.epa.gov/ttn/scram/. (Last accessed March 2004).

5. U.S. Environmental Protection Agency. 2004. Clearinghouse for Inventories & Emission Factors. CHIEF Software and Tools. Updated March 2004. Available at: http://www.epa.gov/ttn/chief/software/speciate. (Last accessed March 2004).

6. U.S. Environmental Protection Agency, Region 6. 2004. Air Modeling for Combustion Risk Assessments. Updated February 5, 2004. Available at: http://www.epa.gov/earth1r6/6pd/rcra_c/pd-o/comb_air.htm. (Last accessed March 2004).

7. U.S. Environmental Protection Agency. 1999. *Dispersion Modeling of Toxic Pollutants in Urban Areas, Guidance, Methodology and Applications*. Office of Air Quality Planning and Standards, Research Triangle Park, NC, July 1999. EPA/454/R-99/021. Available at: http://www.epa.gov/ttn/scram/. (Last accessed March 2004).

8. U.S. Environmental Protection Agency. 2002. *The Air Pollution Training Institute (APTI)*. Updated August 22, 2002. Available at: http://www.epa.gov/air/oaqps/eog/apti.html. (Last accessed March 2004).

9. U.S. Environmental Protection Agency. 1990. A*ir/Superfund National Guidance Study Series. Volume II: Estimation of Baseline Air Emissions at Superfund Sites*. 1990. EPA/450/1-89/002a. Available at: http://www.epa.gov/superfund/resources/soil/part_3.pdf.

10. U.S. Environmental Protection Agency. 1988. *User's Guide for the Fugitive Dust Model (FDM) (Revised).* EPA/910/9-88/0202R.

11. U.S. Environmental Protection Agency. 2003. Emission Inventory Improvement Program, EIIP Document Series - Volume IX. Updated February 11, 2003. Available at: http://www.epa.gov/ttn/chief/eiip/techreport/volume09/. (Last accessed March 2004).

Chapter 10 Assessing Air Quality: Monitoring

Table of Contents

10.1 Introduction 1

10.2 Air Toxics Monitoring: Recent Advances 2

10.3 Monitoring for Air Toxics Risk Assessments: Why Monitor? 4

10.4 Planning for Air Toxics Monitoring 9
- 10.4.1 General Planning Approach 10
- 10.4.2 Specific Planning Issues 14

10.5 Implementing Air Toxics Monitoring 19
- 10.5.1 Locating Monitors and Selecting Sample Size 19
  - 10.5.1.1 Locating Monitors 19
  - 10.5.1.2 Selecting Sample Size 24
  - 10.5.1.3 Setting Up a Monitoring/Sampling Program 25
- 10.5.2 Data Analysis and Reporting 28
- 10.5.3 The Use of Monitoring Data to Calculate Exposure Concentrations 29

10.6 Monitoring Methods, Technologies, and Costs 31
- 10.6.1 Ambient Air Monitoring 33
- 10.6.2 Sampling Costs 35

10.7 Archiving Air Toxics Monitoring Data 35

10.8 Using Air Monitoring Data to Evaluate Source Contribution 37

References 38

10.1 Introduction

In environmental investigations, the term "monitoring" describes the collection of actual samples of environmental media and then subjecting those samples (usually) to chemical analysis to determine the identity and concentration of the various pollutants in the sample. A distinction may also be made between sampling (i.e., stack testing) and monitoring (i.e., for ambient concentrations). In air toxics risk assessment, this process commonly consists of collecting air samples and either evaluating the samples at the monitoring station itself, or sending them to a laboratory for evaluation.

For air toxics risk assessments, monitoring and analysis can help determine the concentration of both those pollutants in air and those that have migrated into other media, such as soil, water, sediments, and biota. This chapter discusses the use of monitoring to evaluate pollutants in air. Chapter 19 discusses the use of monitoring in media other than air.

Many aspects of a monitoring program will depend on the spatial scale of the assessment being supported by the measurement program:

- **Micro-scale** – highly localized regions up to 100 meters in size; these might reflect city blocks or individual households.

- **Middle-scale** – regions of several blocks with sizes of 100 to 500 meters.

- **Neighborhood-scale** – an extended area with uniform land use (and, hence, relatively homogeneous receptor population), extending up to several kilometers in size.

- **Urban-scale** – overall city or county conditions, perhaps up to 50 km in size.

- **Regional- or national-scale** – a state, several states, or the entire nation.

Air toxics risk assessments often examine exposure to relatively large numbers of people over relatively large geographic areas (e.g., a neighborhood or urban area, county, or larger). In these instances, the risk managers and analysts must carefully use their planning and scoping activities to develop the questions they want to answer and identifying the types of data they will need to answer those questions. For some questions and data needs, monitoring is the preferred tool for estimating inhalation exposure concentrations for air toxics risk assessment, either as the primary way of determining concentrations in air or as a way to test and normalize model results (and look for gaps in the emissions inventory).

This chapter provides an overview of monitoring, including recent advances by EPA (Section 10.2); the reasons for monitoring (Section 10.3); how to plan a monitoring program (Section 10.4); implementation (Section 10.5); available air monitoring methods (Section 10.6); archiving monitoring data (Section 10.7); and using monitoring data to evaluate source contribution (Section 10.8).

10.2 Air Toxics Monitoring: Recent Advances

EPA recently published a draft *National Air Toxics Monitoring Strategy* that describes the structure of the national air toxics monitoring program, including its history, status, and expected products.[1] At the start of the program, EPA's focus was on "nationally pervasive" priority pollutants. In recent years, EPA has initiated local scale monitoring studies to address potential air toxics problem areas.

EPA's Ambient Monitoring Technology Information Center (AMTIC)

AMTIC (http://www.epa.gov/ttn/amtic/welcome.html) is centered around the exchange of ambient monitoring related information. Established in 1991 as an electronic Bulletin Board System (BBS), AMTIC has evolved with changing technology into a page on the World Wide Web. AMTIC is operated by EPA's Office of Air Quality Planning and Standards (OAQPS) through the Monitoring and Quality Assurance Group (MQAG). AMTIC contains information on all the Reference and Equivalent methods for the Criteria pollutants, the toxic organics (TO) Methods for air toxics and other noncriteria pollutant methodologies, Federal Regulations pertaining to ambient monitoring, ambient monitoring quality assurance/quality control (QA/QC) related information, information on ambient monitoring related publications, ambient monitoring news, field and laboratory studies of interest, and updates on any new or developing EPA Ambient Air standards.

EPA's air toxics monitoring is structured into four groups – national level, local scale, persistent bioaccumulative toxics (PBTs), and "other" EPA-specific programs.

- The National Air Toxics Trends System (NATTS) program is a network of monitoring stations at 22 urban or rural locations across the country (see Exhibit 10-1). The focus for these sites is on seven "nationally pervasive" priority pollutants (formaldehyde, arsenic, chromium, benzene, 1,3-butadiene, acrolein, and light absorbing carbon). All of the stations are expected to become operational in early 2004.

- Local scale monitoring studies are designed to complement NATTS, but they are shorter-term (less than 2 years) and have more flexible study requirements to go beyond the scope of the NATTS. Local-level studies provide information of urban/local interest that is not achievable with a single monitoring site at a city. For example, these studies may address specific source categories or better characterize pollutant levels associated with different locations in a metropolitan area. EPA plans to implement 10 to 15 local scale monitoring projects that are implemented by state, local, and tribal (S/L/T) air pollution control agencies.

- Programs that monitor atmospheric deposition of PBTs include (1) the National Atmospheric Deposition Program – Mercury Deposition Network (NADP – MDN), a multi-agency program with approximately 90 monitoring sites; (2) the Integrated Atmospheric Deposition Network (IADN), a partnership between EPA and Canada, which is measuring PBTs in the Great Lakes Region; and (3) the National Dioxin Air Monitoring Network (NDAMN), a 30-site research program.

- A variety of EPA Regional air toxics monitoring activities that existed prior to NATTS continue.

Exhibit 10-1. National Air Toxics Trends Stations (NATTS) Sites

January 2003 Startup	January 2004 Startup	Pilot Programs
Providence, RI	Chittenden County, VT*	Barcelona/San Juan, PR
Roxbury, MA	Rochester, NY	Providence, RI
New York, NY	Tampa, FL	Keeney Knob, WV*
Washington, DC	Chesterfield, SC*	Tampa, FL
Decatur (Atlanta), GA	Chicago, IL	Detroit, MI
Hazard, KY*	Mayville, WI	Rio Rancho, NM
Detroit, MI	Harrison County, TX*	Cedar Rapids, IA
Deer Park (Houston), TX	Phoenix, AZ	San Jacinto, CA
St. Louis, MO	La Grande, OR*	Grand Junction, CO*
Bountiful, UT		Seattle, WA
Grand Junction, CO*		
San Jose, CA		
Seattle, WA		* rural site

Source: EPA's *Latest Findings on National Air Quality*[2]

EPA has encouraged a significant effort over the past few years to increase reporting of air toxics sampling results to EPA's AirData database website (http://www.epa.gov/air/data). For example, the Lake Michigan Air Directors Consortium (LADCO), the Northeast States for Coordinated Air Use Management (NESCAUM), and the California Air Resources Board (CARB) "mined" existing data from approximately 300 existing monitoring sites across the U.S. to provide information about the spatial pattern, temporal profile, and general characteristics of air toxics compounds. EPA collected additional data for this analysis from a year long monitoring study carried out in four urban areas and six smaller city/rural areas. A number of reports, newsletters, and related documents describing EPA's air toxics monitoring efforts are available at EPA's Ambient Monitoring Technology Information Center website.[3]

10.3 Monitoring for Air Toxics Risk Assessments: Why Monitor?

Air toxics programs have long used monitoring to evaluate the concentration of chemicals in air. In general, monitoring (sampling and analysis) results may help:

- Identify and estimate current exposures to ambient concentrations of air toxics (outdoor and/or indoor) at a specific location of concern (e.g., a school or neighborhood). As an example, EPA tracks ozone concentrations at numerous locations around the country, with results available over the Internet (http://www.epa.gov/airnow/) for many locations, virtually in real-time. As another example, air toxics monitoring can be used to evaluate the impacts of a specific source on a nearby receptor ("source-oriented" monitoring).

- Develop or refine values for specific parameters needed by air dispersion models (for example, study-specific release data, meteorological conditions).

- Validate the predictions of a model in specified circumstances (e.g. validate that the location of highest exposure predicted by the model is correct, which increases confidence that a maximally exposed subpopulation has been identified – may be difficult to do without a very dense monitoring network).

- Track trends in air quality levels (e.g. to determine whether air pollution programs have generally been effective at reducing exposures).

- Identify gaps in emissions inventories (e.g., monitoring identifies an airborne chemical that is not reported in existing emissions inventories) or close gaps that might be present in existing data (e.g., concentrations of specific air toxics in specific releases).

- Determine compliance with air toxics legal requirements (e.g., permit limits at a factory, emissions limitations on motor vehicles).

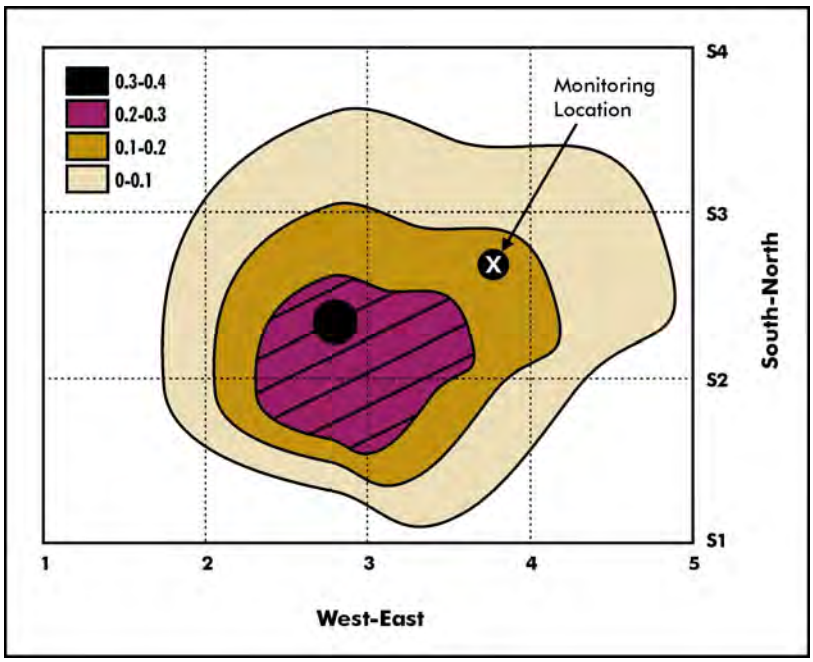
- Gather data in support of enforcement actions.

Ultimately, the choice of whether to monitor or model (or both) depends on the goals of the assessment, the exposure setting, other specific project circumstances (e.g., many communities want monitoring as part of a risk assessment), and the assessing entity. For example, to understand the exposure an actual individual receives as they move about their daily activities, personal monitoring is the best option because it reflects the pattern of this movement. However, such studies are rarely done outside of research settings. As another example, compliance with a permitted release rate may also require monitoring as the preferred method of measurement. Exhibit 10-2 provides a brief comparison of modeling versus monitoring.

Exhibit 10-2. Comparison of Modeling and Monitoring Approaches for Estimating Ambient Air Exposure Concentrations (ECs)	
Modeling	**Monitoring**
Modeling is relatively fast and inexpensive compared to monitoring. Many screening-level models can be run in spreadsheet formats and require relatively simple input parameters. Many dispersion models, along with technical reference manuals and other support documents, are available for free download from EPA's Support Center for Regulatory Air Models (SCRAM) website (http://www.epa.gov/ttn/scram/). Resources normally need to be expended to enhance the local air toxics emission inventories to make air toxics modeling more precise.	With monitoring, it takes time to build data, and there are methodological limits and logistical issues. How expensive monitoring is depends on what you are trying to do and how much you are willing to pay. Monitoring does not always require equipment purchase, and some states and local areas already have equipment. Some less expensive monitoring techniques are now available (e.g., passive samplers).
Modeling results can estimate concentrations over a large spatial area (e.g., a 50-km radius from a source) and can provide a "big picture" view of the assessment area. Modeling also allows for analysis of EC at multiple points throughout the assessment area. The downside of modeling, however, is that these are predicted concentrations.	Monitoring results provide actual measured concentrations. Multiple locations may be required to characterize concentrations over an area, although Geographic Information Systems (GIS) methods facilitate interpolation between locations. The downside is that the monitoring may not be representative of a large geographic area.
Screening-level models can provide a predicted estimate of whether significant concentrations are likely. A simple screening analysis may be sufficient to make a risk management decision that no action is required.	Monitoring can be used to identify and measure exposures for specific individuals at a specific location of concern (e.g., a school). This data can provide a quick screen to determine whether more extensive monitoring is needed.
Models can be used to identify areas where maximum concentrations are likely to occur, and thus where to focus efforts for additional tiers of the assessment. Uncertainties in model parameters and the discrete division of the wind field used in models (often with only eight wind directions) can result in incorrect identification of the locations of maximum concentration.	Monitoring can identify areas and actual levels of exposure occurring at the monitoring sites. Monitoring can also be used to indicate the point of maximal exposure if the monitoring is designed for that purpose. The selection of the monitoring locations is critical; if placed in the wrong locations, monitors can provide incorrect and misleading information about maximal exposures.
Models can be used to identify the subset of chemicals of potential concern (COPCs) and exposure pathways/routes that have the greatest contribution to risk. This can be helpful in focusing efforts for additional tiers of the assessment as well as determining appropriate risk management actions.	Monitoring can be used to confirm significant exposure pathways and routes. (Measured concentrations can be compared to risk-based screening levels). It also can be used to identify compounds that may not have been suspected and, hence, were not included in models (i.e., monitoring allows identification of gaps in the emissions inventory).
Models allow "what if" scenarios to be evaluated (e.g., what if a permitted emission were doubled?).	Monitoring can only evaluate current conditions.
More complex modeling may allow explicit predictions and estimates of variability in exposure.	A large number of samples generally is needed to characterize variability; this may be prohibitively expensive. Monitoring, however, provides a direct and reliable means to characterize variability.
Models often use simplifying assumptions and data inputs that may or may not be representative of the specific assessment area. This introduces uncertainty into model predictions.	Monitoring can be used to confirm actual exposure levels, to investigate assumptions or calibrate models to site-specific conditions, and to close gaps in data, reducing uncertainties.

Air toxics risk assessments, however, tend to examine potential exposures to hazardous air pollutants (HAPs) and other air toxics for a relatively large number of people over relatively large geographic areas (e.g., a neighborhood or urban area, county, or larger). In these instances, the risk managers and analysts must carefully use their planning and scoping activities to develop the questions they want to answer and to identify the types of data they will need to answer those questions. For some questions and data needs, monitoring is the preferred tool. For others, modeling is better. In general, most air toxics risk assessments will benefit from some combination of both modeling and monitoring to provide the depth and breadth of information that will be necessary to answer the assessment questions (see hypothetical example in Exhibit 10-3).

Exhibit 10-3. Hypothetical Example of a Combined Modeling and Monitoring Program



This figure illustrates a hypothetical set of isopleths for annual average air concentrations that a dispersion model predicted, assuming a single source (black dot) near the center of the geographic region. Note that the model predicts the point of maximal exposure to be somewhere within the area bounded by grid points 2, 4, S1, and S3, based on the existing information on release rate, wind direction, and effective release height. In this hypothetical example, a monitoring station was used to measure ambient concentrations as a means of evaluating the model predictions. Note that the monitoring location is not in the area of estimated highest concentration and, therefore, might not provide a better estimate of maximum exposure.

Indeed, most air toxics risk assessments that evaluate exposures to populations receiving impacts from one or more sources should generally consider using modeling as their primary tool to evaluate and characterize exposures and risks. In certain instances, assessors may use monitoring as the primary tool to evaluate exposure concentrations for potentially exposed populations. The utility of modeling for neighborhood and larger scale analyses is that it provides a better picture of the variation of exposure conditions over the assessment area domain (i.e., modeling provides spatial resolution) and allows a more straightforward approach to source allocation (i.e., what portion of the risk is caused by each of the modeled sources).

Monitoring, on the other hand, only provides estimates of concentrations at the point at which samples are taken, and it is often difficult to clearly define the spatial coverage that those measured concentrations represent. In addition, it is often difficult to use monitoring data for source allocation (especially for chemicals emitted by numerous sources). Monitoring plays a crucial role in identifying important chemicals that the emissions inventories may not have captured. In rarer instances, assessors can use monitoring as the primary tool to evaluate exposures for potentially exposed populations; however, this method carries a corresponding increase in the uncertainty of the results (see Section 10.4 on how to use ambient monitoring data to develop estimates of exposure concentration). (Note that, in limited circumstances, geostatistical techniques such as kriging are sometimes applied to estimate concentration variation between a set of monitors. This topic is beyond the scope of this reference manual; however, assessors are encouraged to carefully consider the uncertainties associated with this type of approach and whether alternate tools, such as air dispersion modeling, would provide a better understanding of concentration gradients across the study area. In addition, the average concentration of atmospheric pollutants across a study area is sometimes estimated by averaging the results of all the monitors in the area. However, since pollutant concentration can change rapidly across space and time, combining data across monitors may "average out" very important information about exposure at a particular monitoring location. It is for this reason that combining data across monitors is not commonly performed and assessors are encourage to carefully consider the pros and cons of attempting such an analysis. If monitors are combined, the results should, nevertheless, be reported alongside the results of each of the individual monitors.)

If assessors make the choice to implement a monitoring program, it is important to carefully design the sampling and analysis approach to provide meaningful input into the risk management decision. Because sampling and analysis are relatively expensive and time consuming, a well-designed monitoring program can ensure the efficient use of resources. Well designed and implemented monitoring programs quantify not only the concentrations but also information related to the associated data uncertainty. The study-specific conceptual model and analysis plan that assessors develop during the planning and scoping phase help ensure a well-designed sampling and analysis program that will yield results suitable for decision-making purposes. Monitoring programs are commonly designed to:

- Use a sampling methodology that results in scientifically defensible data and that meets regulatory criteria or other concerns – it is important to utilize methodologies that are scientifically defensible and acceptable within a regulatory context;

- Identify and quantify air toxics (or their breakdown products) of interest with respect to contribution to risk in all media of interest (including, in some cases, non-air media; see Chapter 19);

- Attain quantitation requirements (e.g., quantitation limits) sufficient to compare to dose-response values (e.g., the sensitivity should be sufficient to allow reliable measurements below concentrations anticipated to produce adverse health effects);

- Demonstrate acceptable confidence in the data set to be used for decision-making based on quality assurance benchmarks including benchmarks for precision, accuracy, representativeness, completeness, and comparability; and

- Provide for a clear and unambiguous data validation and reporting methodology so monitoring results can be tracked, verified, and validated when they are used in decisions.

The design of a monitoring program that meets data quality objectives (DQO) and quality assurance project plan (QAPP) requirements depends on the answers to four questions:

1. **What is the risk management decision to be made, and how will assessors use monitoring results in that decision?** Monitoring programs typically are a component of risk assessments that support risk management decisions; these decisions normally focus on how best to reduce risks from exposure to air toxics through reducing or otherwise limiting emissions.

Quality Assurance Project Plan (QAPP) and Data Quality Objectives (DQO) Process

As Chapter 6 introduced, a QAPP is part of the overall risk assessment analysis plan that ensures the quality of data used in decisions. Generally included in the data quality program is the DQO process, which establishes the criteria that must be met if data are to meet the needs of a decision-maker (e.g. it establishes the error bounds on data, which are related in turn to the uncertainties a decision-maker, can tolerate in reaching a defensible decision). Assessors can accomplish this goal through the following seven steps:[a]

1. State the problem.
2. Identify the decision to be made.
3. Identify inputs to the decision (i.e., which data are needed).
4. Define the study boundaries (i.e., what factors, scenarios, etc., will be included in the study to produce these data).
5. Develop a decision rule (i.e., how the data will relate to a specific decision to be made).
6. Specify limits on decision errors (i.e., how much uncertainty can exist and still allow a defensible decision to be made).
7. Optimize the design of the study to ensure the data quality meets the decision rule.

The QAPP specifies precisely how to collect and analyze the data to meet the goals established by the DQO process. The QAPP establishes specific procedures that assessors follow to meet DQOs. These DQOs include procedures for identifying reliable methods, choosing sample locations and frequencies, handling samples, calibration of equipment, recording and archiving of data, and analysis of the data. The DQO goal is to ensure that all members of the project team understand, and follow, procedures that will ensure the results of the study meet the data quality needs of a decision. Once these DQOs have been established, it is necessary to develop a plan as to how the participants will meet them in practice while collecting the data for the study.

[a]U.S. Environmental Protection Agency (EPA). 1994. *Guidance for the Data Quality Objectives Process*, EPA QA/G-4, Office of Research and Development, EPA/600/R-96/055; available at http://ww.epa.gov/swerust1/cat/epaqag4.pdf.

2. **How accurately must the results be to be useful in these decisions?** The reliability of monitoring program results must be adequate for the needs of the risk management decision. For example, risk assessors need to quantify air concentrations and/or exposures within some bounds of accuracy and/or precision. It is important to meet these criteria of accuracy and precision, but not necessarily to exceed them. As noted in Appendix H, the data quality objectives must provide results that allow reliable decision-making. However, resources that participants devote to one aspect of a monitoring program, such as choosing a larger number of sampling sites, will draw resources away from another aspect of the program, such as sampling for a larger number of air toxics. This is why it is essential to understand fully the decision that the given set of results will support, other results that assessors will need to support that same decision, and how participants can balance monitoring results across these different data needs to reduce the levels of uncertainty to acceptable levels. Assessors can achieve this goal by conducting a **sensitivity analysis**[a], which determines what aspects of a full monitoring program will require the greatest attention and resources; monitoring results that play the most significant role in a decision may require the greatest allocation of resources.

3. **What methodologies are available to monitor at a particular level of quality?** The choice of monitoring method depends on the specific air toxic(s) to be analyzed, the objective of the monitoring (as the DQOs specified), the time over which a result is to apply, and available resources. It is important to note here that there do not currently exist valid methods (either field, lab, or both) for a large number of chemicals that may be of interest; for methods that do exist, the achievable sensitivity may not match the DQOs (this is another reason that modeling is often used as the primary decision making tool since these issues are irrelevant to models).

4. **What resources are available for the monitoring program?** The choice of a monitoring strategy often depends primarily on available resources (e.g., time, money). These factors are of particular concern in air toxics monitoring because most studies of chronic exposure generally require a minimum of one full year of data to characterize chronic exposure. It is not uncommon to have a lag time of two years or more from the beginning of a monitoring study to a final report when one considers the time it takes to plan the monitoring study, obtain access to land, build the monitoring structures, run the study, analyze the samples, validate the results, and write the data report.

10.4 Planning for Air Toxics Monitoring

As noted above, planning is a critical part of any air toxics monitoring program. The discussion of planning below first describes a recommended general approach (Section 10.4.1) and then outlines several specific planning issues (Section 10.4.2). EPA has developed resources that provide additional details on operating procedures, with discussions of data quality issues, definitions, and applications to specific methodologies.[4]

---

[a] A sensitivity analysis shows the relative effect of uncertainty in each aspect of an assessment on the overall uncertainty in that assessment. Ideally the data quality objectives will be more stringent for those measurements that play a larger role in the final decision, since narrowing the uncertainty in these measurements significantly reduces uncertainty associated with the decision.

10.4.1 General Planning Approach

Planning an air toxics monitoring program involves a step-wise integration of sampling protocols with data quality criteria and data analysis processes that are consistent with the study-specific conceptual model (CM), QAPP, and DQO processes. Although presented step-wise, the process is actually iterative, and decisions at one step may require verification or modification of assumptions or decisions made at previous steps.

1. **Understand the problem**. As noted above, assessors may design monitoring programs to support a number of different types of management decisions. For risk assessments, the CM can focus participants' understanding of both the scope and the breadth of the problem that the sampling and analysis are to address. The ***most important questions to answer*** immediately are: whether assessors will use monitoring results to characterize exposure and risk, whether they will use results to evaluate air quality model performance and look for gaps in the emissions inventory, or whether they will use results for both reasons. This is a ***critical question for participants to answer***, because the data needs can be drastically different, depending on how the assessors will use the monitoring data.

2. **Identify existing data**. Sampling and analysis for risk assessment may not be necessary if the information to be developed is already available from other sources and meets the quality requirements for decision making. The data sources discussed in Chapter 4 may provide sufficient information for the risk management decision.

Examples of Study-Specific Questions

- What is the maximum plausible value of EC for the population in a geographic region, taking into account spatial and temporal variability and uncertainty?
- What is the location of this maximal value within the geographic region?
- Which air toxics are found at the highest concentrations with respect to their dose-response values (e.g., which air toxics have the greatest potential to produce a hazard quotient above one)?
- Do monitoring results generally agree or disagree with the value of air concentrations identified by existing models?

3. **Itemize data needs**. Where existing data are insufficient to answer the study-specific questions, it will be necessary to obtain new data through monitoring. Potential data needs include: filling gaps in emissions inventory data; providing input data for models and validating modeling results; generating new data to more fully characterize exposures in areas, populations, or pathways; establishing trends over time; or supplementing a body of data to increase their quality for the risk management decision. The process for itemizing data needs includes articulating critical decision criteria (which may drive data quality needs and/or selection of specific methods), applying these criteria to determine areas where existing data are insufficient, and identifying the manner in which new data can supplement existing data to meet the decision criteria. In many ways, the identification and enumeration of data needs acts a bridge between the conceptual model and the DQO process.

4. **Define data quality needs**. The reliability (e.g., accuracy and precision) of monitoring results must be adequate to meet the needs of the risk management decision. However, given finite resources, even well-designed studies may not be able to achieve all quality criteria. That limitation makes it important to determine which criteria are essential for addressing the

study-specific decision problem and for focusing resources on meeting (and not necessarily exceeding) those criteria.

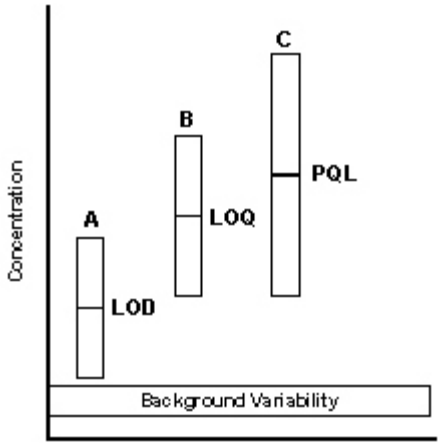
The DQO process determines general data quality objectives to meet specific needs. This process can be informed both by a well specified decision statement and by a sensitivity analysis to determine which aspects of a full monitoring program will require the greatest attention and resources to support that decision. Identification of data quality needs at this level is targeted on the specific problem identified in Step 1, but is independent of the specific methods to be applied. It is important to base data quality criteria at this step on what is required to answer the problem identified in Step 1, not on impressions of best available analytical methods, approaches used in the past, or consideration of questions that might be of general scientific interest but are not of direct use in the decision problem. A common approach is to consider all aspects of sample and data handling from collection to data report writing, as these affect the confidence with which decisions can be made through the introduction of random or systemic errors. A number of factors affect data quality, including bias related to sampling error (e.g., taking only a single sample at one location, which may or may not be representative of actual ambient concentrations) and relative precision related to analysis methods.

5. **Select monitoring methods to meet data quality needs**. The choice of monitoring method depends on the scale of the assessment, specific contaminant(s) to be analyzed, the sampling time over which the result is derived (e.g., a sample collected over 15 minutes versus a sample collected over 24 hours), the decision criteria or other reporting limit needs, and the resources available (see Section 10.3). Methodologies include the sampling methods and techniques, sampling program design (i.e, sampling frequency, coverage, and density), as well as analytical methods. The data quality needs identified in Step 4 represent the total data quality requirements of all aspects of the sampling and analysis process necessary to support risk-based decision-making. Therefore, evaluation of all aspects of sampling and analysis with respect to data quality needs is necessary for proper method selection.

   The QAPP process involves balancing decisions for method selection to meet data and quality needs. Selection of the methods for both sampling and data analysis defines the approach and defines what is termed the **measurement quality objectives**. Although there is a natural tendency to select sampling and analysis methods based on previous data, it is important that the benefit of consistency and likely improved comparability are not outweighed by data gaps that Step 3 identified. For example, in a risk assessment for chlorinated volatile solvents, the presence of fluorinated volatile solvents may cause assessors to overestimate chlorinated concentrations due to analytical interferences. The method selection generally takes into account the known or suspected presence of other chemicals having similar toxic effects, symptoms, and mechanisms, and/or that which otherwise may affect sampling and analysis results. To take this into account, the study may require adding chemicals to the target analyte list, selecting a method where these compounds are not potential interferents, or limiting the scope of the study with stated assumptions about contributions from these undefined factors (e.g., stating only that the measured concentration is the sum of a defined set of analytes and not applicable to any one analyte in the mixture).

Detection Limits and Limits of Quantitation

The **detection limit** is the minimum concentration that an analyst can reliably expected to find (i.e., detect) in a sample, if it is present. For any given method (e.g., the method to analyze for volatile organic compounds [VOCs] in air), this limit is established in each lab for each instrument and is called the **method detection limit** or **MDL**. An MDL of 1µg/m^3, indicates that a field sample that contains 1 µg/m^3 or below of contaminant will probably not be detected by the instrument in question. The **limit of quantitation (LOQ)**, on the other hand, is the minimum concentration for which the analyst can reliably say that the substance is present in the sample and at a specific concentration within some pre-established limits of precision and accuracy. If the limit of quantitation is 2 µg/m^3, then measurement results above 2 µg/m^3 may be reported as not only indicating the presence of the substance in the sample, but as indicating the specific concentration measured (i.e., positive identification, certain concentration). Measurements between the MDL and the LOQ , indicate the presence of the substance in the sample, but analysts can only make an estimate of the concentration (i.e., certain identification, uncertain concentration). NOTE: It is common (but incorrect) to refer to the quantitation limit as the detection limit. The LOQ, practical quantitation limit (PQL), estimated quantitation limit (EQL), and sample quantitation limit (SQL; see below) are all limits of quantitation, not detection. Thus, when one says "benzene was not detected at a detection limit of 5 µg/m^3," this most likely actually means "benzene was not detected; the limit of quantitation was 5 µg/m^3." Likewise, when a lab reports a measurement as "<5 µg/m^3," this most likely means "not detected; the limit of quantitation was 5 µg/m^3." There is much confusion on this point and analysts must clarify with the laboratory exactly what they mean in their lab reports (and what the analyst needs to have reported to them for their risk assessment activities). For air toxics risk assessments, the MDL is largely irrelevant for purposes of estimating exposure and the limit of quantitation is the critical information that needs to be reported (see Chapter 7).



In establishing limits of detection and quantitation, it is necessary to give the confidence level associated with the detection limit and the limit of quantitation. In this figure, the confidence level is 99 percent. The *Limit of Detection* (LOD) is then the minimum concentration that has a 99 percent probability of producing a result above background noise (background is shown in the figure as a horizontal bar) using a specific method. The LOD includes two considerations: an *instrument detection limit*, accounting for variation in the instrument when it is presented with repeated samples at the same concentration, and additional variation caused by the need to sample, handle the sample, etc. (which can cause variations in the relationship between the concentration in the environmental medium and the concentration presented to the instrument). The LOD is the horizontal line in the bar marked A. Note that the range of variation of results from a concentration at the LOD (shown as the bar marked A), and the lower end of this range just barely avoids moving into the range of background variability.

Detection Limits and Limits of Quantitation (continued)

The LOQ assumes best practice in performing the measurements. It also is of interest to ask what the LOQ would be using more common, routine practice. The *Practical Quantitation Limit* (PQL) is the minimum concentration that has a 99 percent probability of producing a result above the LOD under routine lab conditions (shown as the bar marked C). Under these conditions, the variation will be larger than under ideal conditions, and so the PQL is higher than the LOD. Each lab must establish these parameters for each method on each analytical instrument. When actual environmental samples are evaluated on an instrument, the actual PQL reported for any given sample may vary (for example, if a sample is highly concentrated and needs dilution before analysis, the resulting PQL for that sample will be elevated by an amount proportional to the dilution). It is for this reason that PQLs reported for actual samples are referred to as a **sample quantitation limits** or **SQLs**. When using analytical monitoring data for air toxics risk assessment purposes, ***the MDL is irrelevant***. The SQL is the key factor in developing exposure concentrations (see Chapter 7).

Having established these terms, some system then is needed to "flag" results as being either usable or unusable for the purposes of decision-making. For example, in the Superfund program,[a] results are flagged *"R"* if the data are unusable for some reason and *"J"* if the data fall between the SQL and the MDL. A more thorough description of data qualifiers if presented in Appendix I.

[a]U.S. Environmental Protection Agency. 1992. *Guidance for Data Usability in Risk Assessment (Part A)*. Office of Emergency and Remedial Response, Washington, D.C. EPA Publication 9285.7-09A; available at http://www.epa.gov/superfund/programs/risk/datause/parta.htm.

6. **Develop systems to ensure that data meet decision requirements**. Setting the objectives and selecting sampling and methods capable of meeting the DQOs are the prelude to determining whether and to what degree the data may support risk management decisions. Having collected and analyzed the data, it will be necessary to determine whether decisions can now be made with the desired confidence. For example, the actual data collected must be assessed for quality and compared against any decision criteria such as toxicity dose-response values. Where the quality is insufficient to support the decision (e.g., insufficient to determine whether the benchmark is or is not exceeded), the previous steps may need to be re-assessed.

   It is also important to evaluate the contribution to uncertainty that is related to sample collection and sample program design as well as analytical method uncertainty. Sampling uncertainty is decreased when sampling density increases, however resource limits often constrain sample density. Typically, errors in the collection of field samples are much greater than errors introduced by preparation, handling, and data analysis; yet, most sampling studies have devoted resources to assessing and mitigating laboratory errors. Ultimately, the proper use of a QAPP that considers the entire process (sample collection through lab data reporting) allows for evaluation of and reduction in uncertainty across all the activities of the monitoring program, focusing resources on those aspects contributing most significantly to uncertainty affecting decision-making.

7. **Develop documentation**. The QAPP and other planning documents must record the results of the environmental data collection design process. Information to be documented includes the assumptions, findings, outliers, biases, data confidences, and other factors that are critical to implementation, as well as evaluation and eventual interpretation of the data collected. Data collected and analyzed is often reviewed thoroughly to ensure they are adequate to support decisions; sufficient documentation allows such a review.

10.4.2 Specific Planning Issues

The design of the monitoring program also raises some specific issues:

- **Select appropriate monitoring or sampling methods for the chemical(s) to be measured**. In general, it is important that the methods selected have the sensitivity needed to monitor at concentrations likely to be of health and/or regulatory concern. At a minimum, the PQL or SQL should be below any relevant health benchmarks (e.g., the human health dose-response values discussed in Chapter 12). For some chemicals, the limit of the current technology may not allow for a PQL or SQL that is below a health benchmark (or, that level may be reached, but at a higher cost). In such instances, the planning and scoping team must decide how best to balance resources to support data quality needs.

- **Select appropriate monitoring sites, sample collection frequency, and length of sampling time for the spatial and temporal variation of the scale being assessed and for the objective of the air toxics monitoring being conducted**. The way monitoring captures this variation depends on the particular measure(s) needed to support the risk management decision. For example, the monitoring goal might be to estimate the average long-term exposure to people spread over a large geographic region (e.g., the average urban exposure for a typical resident in a town). In this case, measurements spaced on a grid throughout that region, or selected with a spatial density proportional to population density, may be appropriate. On the other hand, if the goal is to identify or verify the maximum modeled exposure or to perform a screening-level assessment in a population living down-wind from an industrial source, sampling should be performed at the location likely to represent the highest exposure, or in several different regions to identify the site representing the highest exposure. Again, issues such as atmospheric photochemistry and differential settling of metals are important considerations.

  Assessors often make similar decisions when considering temporal variation. For example, samples may vary over time due to fluctuations (e.g., emission rates from a facility may fluctuate over time) or a systematic temporal trend (e.g., a facility might change its production methods or products over time). In the former case, it is necessary to obtain enough samples spread over a large interval of time to estimate the mean over the measurement interval. In the latter case, the samples must be spaced in time so as to capture the trend (i.e., a **time-trend study** must be performed). In addition, the objective of a study may be to capture high short-term spikes in chemical concentrations. In this case, samples collected over a 24-hour period may "dilute out" these spikes, and frequent shorter term samples (e.g., collected over 15 minutes) may be required.

A recent evaluation of many of the issues regarding variability was recently published using data from a wide range of monitoring sites throughout the United States (see also Exhibit 10-4).[5] These results support the conclusions that: (1) environmental variability is a more important source of uncertainty than analytical uncertainty, emphasizing the need to carefully select the location and timing of monitoring; (2) temporal variability dominates data variability, emphasizing the need to not only carefully select the timing of monitoring, but to ensure that results are properly averaged over relevant exposure periods; and, (3) analytical uncertainty becomes a more significant contributor to overall uncertainty as ambient concentrations approach background levels.

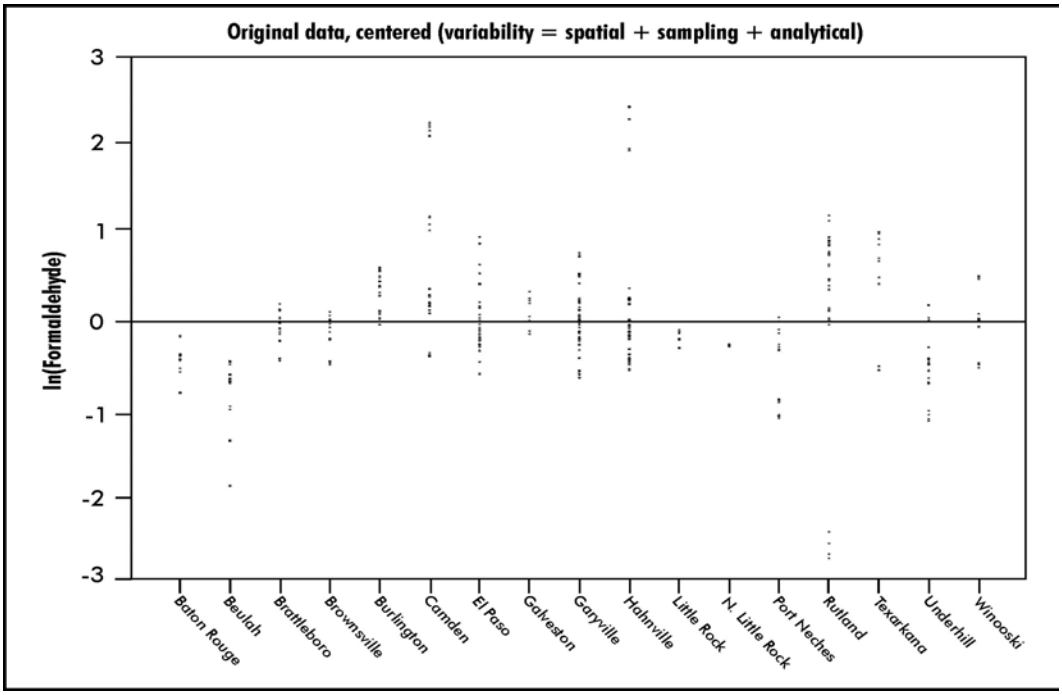
- **Most often, the monitoring efforts address the four main sources of variability in measurements**. These four sources are:

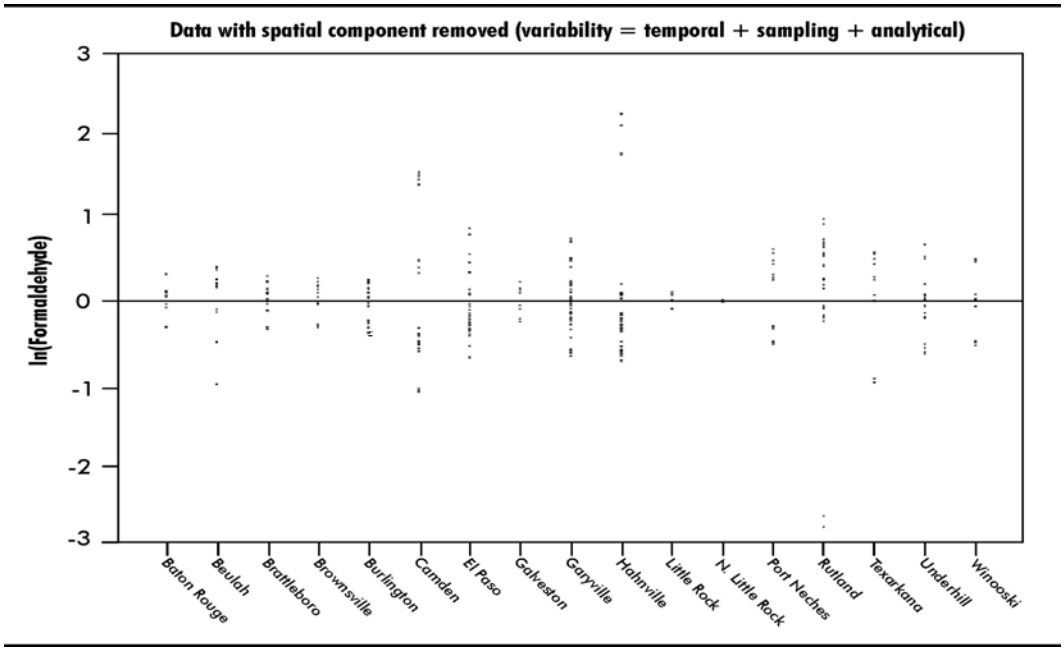
  – **Analytical**. The same sample analyzed repeatedly yields different concentrations.
  – **Sampling**. Duplicate samples collected using two identical monitoring devices from the same location and time yield different concentrations. This type of duplicate sampling is often performed to determine the precision of the method. In general, a minimum of 10 percent of the measurements in a monitoring program should be co-located to collect duplicate samples.
  – **Temporal**. Repeated samples at different times at the same location yield different concentrations.
  – **Spatial**. Samples from different locations at the same time yield different concentrations.

  Ideally, assessors allocate monitoring resources in a manner that is consistent with the relative contribution of these four sources to uncertainty. However, uncertainty may not be evident prior to establishing the sampling program. Some insights on the relative contributions can be obtained from the recent study of monitoring variability,[5] but it generally will be necessary to perform an analysis of the analytical uncertainty, the precision, and the degree of spatial and temporal variability before a firm judgment of the relative contributions can be made.

  As noted previously, ambient air monitoring data may not provide a completely accurate picture of exposure. There are several reasons for this limitation. First, air toxics monitors usually are physically located to provide an estimate of air concentration at a specific location. The assessor must then determine how representative the results are to populations in the geographic area around the monitor. For some chemicals, monitoring results can be reasonably representative, especially if the concentration does not show high levels of spatial variability. For other chemicals, results may not be very representative at all, especially at some distance from the monitor. In addition, because people move around outside, their exposures are an average of the ambient air concentrations over the geographic regions in which they move; this exposure may not correspond to the average at any particular monitoring location. People also receive protection from the ambient environment, either in vehicles or by moving indoors or through filters. Thus, ambient air concentrations measured through monitoring and analysis can be taken as an indication of the potential for exposure at a given location.

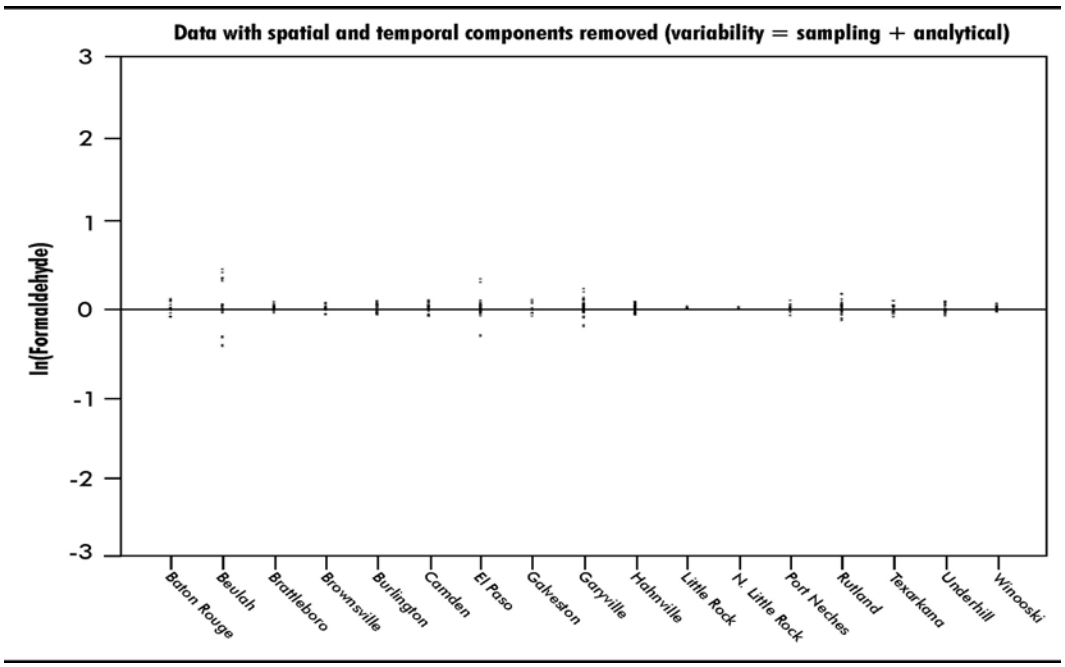
Exhibit 10-4. Temporal and Spatial Sources of Variability in Formaldehyde Sampling

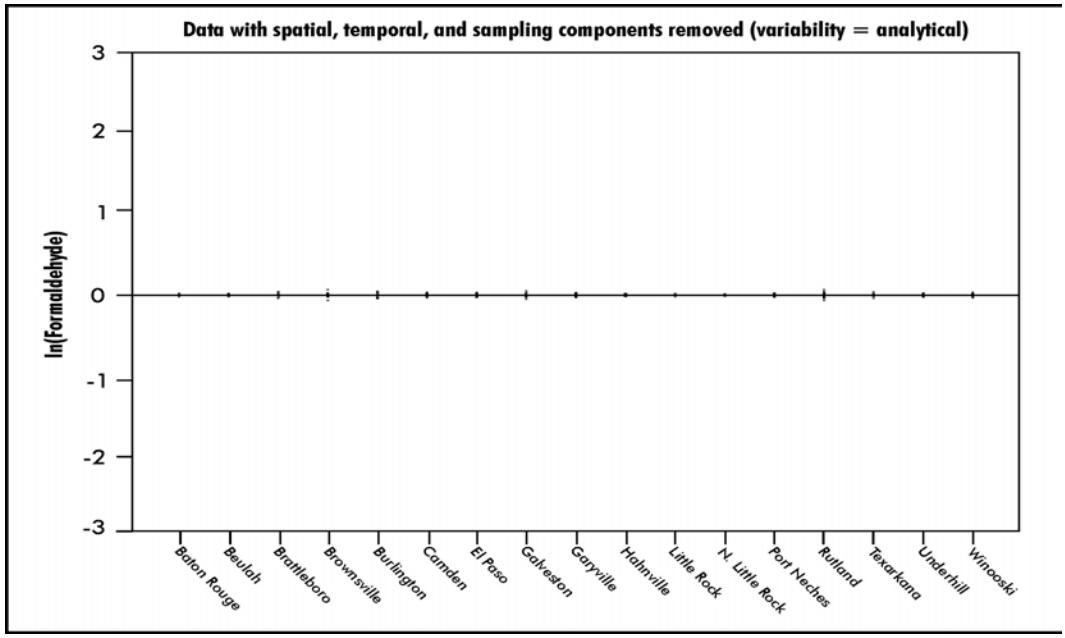




The four graphs in this exhibit summarize the results of Bortnick and Stetzer,[4] obtained by sequentially removing sources of variability. Note that the analytical variability is the smallest source of variability in this case, followed by sampling variability and temporal/spatial variability. Clearly in

Exhibit 10-4 (continued)





this case, a choice of sampling focused on temporal and spatial contributions to variability is needed; since temporal variability dominated, primary attention would focus first on that component in each sampled geographic region.

- **Follow and define standard operating procedures**. Risk assessors follow and define standard operating procedures both in the field (during sample collection and transport to the laboratory) and in the laboratory (during sample analysis). Procedures include those related to sample collection, sample transport, sample storage (including prevention of sample degradation), and chain of custody procedures, as well as sample analysis, validation, and data reporting. Procedures to identify potential problems are put in place. Periodic audits (both field and lab) are commonly performed to ensure procedures are being followed and that measurement and analytical devices are working properly.

- **Determine quantitation and compare limits**. A common approach is to determine quantitation limits and compare them against relevant decision needs, including health benchmarks and likely environmental levels. These quantitation limits should be below the health benchmarks and environmental levels to provide data of use in risk-based decisions.

- **Properly calibrate measurement processes**. One way to ensure the accuracy of the method is to properly calibrate measurement processes. To accomplish this, assessors perform calibration on a time schedule shorter than the time needed for the equipment to "drift"[(b)] further than is permitted under the criteria of accuracy and precision. It is for this reason that it is essential that systems be re-calibrated periodically, on a schedule that is related to the data quality objectives. In addition, it is desirable to cross-calibrate measurement methods by comparing results from several individuals and labs. In an inter-laboratory comparison, split and duplicate samples are submitted to several labs simultaneously, the results are collected, and variation between labs are assessed. Ideally, sample analysis in a monitoring study would be conducted at a laboratory that has participated in such an inter-laboratory comparison and has been certified to produce results within acceptable data quality limits.

- **Adequately record and archive results**. The best monitoring program can fail due to improper record-keeping. A periodic, random check of the archived records (e.g., computer files) is commonly made against "hard copies" to ensure the integrity of the process of recording the data. The recording of all results, including a description of the QA/QC and Data Quality Indicators, is essential because risk managers will use the results in their decisions.

- **Match measurement intervals to the relevant modeling assumptions or health endpoints**. Different health effects require varying averaging time-periods. Cancer and other chronic effects generally require averages over relatively long periods such as a year or more (up to a lifetime). In this case, samples may be taken randomly or systematically throughout the year, with the criterion of obtaining an accurate estimate of the mean. Acute effects, however, require an understanding of the temporal variability over short periods of time. For example, monitors need to measure benzene concentrations within shorter time intervals (e.g., 15 minute, one-hour, 24-hour) for comparison with a health benchmark reflective of the same time period.

---

[b] "Drift" refers to the fact that monitoring systems that are calibrated generally change their electronic and other characteristics in time, so the calibration factor also changes in time.

- **Ensure that temporal sampling reflects diurnal (time-of-day) and seasonal variability**. It is important to recognize that source terms and meteorological conditions can vary systematically both over a day and throughout the seasons. Monitoring programs commonly reflect this pattern, providing proper averages throughout a day (by sampling at selected time points in a day) and between the seasons (by sampling in the different seasons).

In general, most monitoring schemes that are designed to attenuate and validate a model will collect samples and analyze a relatively short list of "indicator compounds." If attenuation and validation are the primary motivation for sample collection, it may not be necessary to measure every compound being modeled, as long as it can be assumed that unmodeled compounds would be expected to behave similarly. However, the amount and type of data collected in the monitoring program designed to validate predicted model results should match the assumptions of the modeling program. For example, if the goal of the modeling program is to estimate long term (usually annual average) concentrations, then monitoring data must also be collected in sufficient quantity to develop an annual average value to compare to the model results. (In general, monitoring samples collected every six days for a year are required to develop a stable estimate of annual average.)[2]

10.5 Implementing Air Toxics Monitoring

Implementing a monitoring program raises two issues in addition to the items above that relate to planning for a monitoring study. These include selecting the actual location of monitors and selecting methods for data analysis and reporting. Each is discussed in a separate subsection below.

10.5.1 Locating Monitors and Selecting Sample Size

Determining the location of an air toxics monitor depends on a number of factors, including the specific purpose of the monitoring (e.g., confirm modeled concentrations at a specific location, estimate background concentrations), meteorological and terrain constraints, and the relative magnitude and location of the source(s) of concern versus other emissions sources that might contribute to measured air concentrations. For example, locations too close to a source may underestimate exposure concentrations if the plume has not yet reached ground level where people can come into contact with the contaminants. Locations too far from the source may also underestimate exposure concentrations for large groups of people due to the dispersion that takes place between the point of touch-down of the plume and the point of monitoring.

10.5.1.1 Locating Monitors

EPA's *Quality Assurance Handbook for Air Pollution Measurement Systems*[6] provides a set of consistent QA practices that will improve the quality of the nation's ambient air quality monitoring data and ensure comparability among sites across the nation. Although these practices were developed specifically for criteria air pollutants, they provide useful guidance for air toxics risk assessments. Exhibit 10-5 summarizes some of the *Handbook's* guidance on the relationship between topography, air flow, and the location of monitoring locations. The following factors are usually considered when siting monitors:

- **Perform measurements at locations that are representative of exposure**. Determining the location will depend on whether the goal is to quantify exposures in general, or exposures to the maximally exposed individual. In the latter case, locations too close to a source may underestimate exposure if the plume has not yet reached ground level where people can come into contact with the contaminant. Locations too far from the source may also underestimate exposure to large groups of people due to the dispersion that takes place between the point of touch-down of the plume and the point of monitoring. Exhibit 10-3 above presented an example of this issue. In that hypothetical example, the area of maximum concentrations predicted by the air quality model falls somewhere within the area bounded by grid points 2, 4, S1, and S3. If the goal of monitoring is to verify these maximum concentrations, then the ideal location for the monitor would be on the plume centerline at the exact point of touch-down of the plume. However, if the goal of monitoring is to verify maximum concentrations at the point of actual exposures, location at the site indicated in Exhibit 10-3 may be more appropriate (measurements at the point of plume touch-down may overestimate maximum actual exposure if there are no individuals within that area). It is essential to determine whether monitoring will estimate exposures to existing individuals or to hypothetical individuals who might move into currently unoccupied areas.

Exhibit 10-5. Relationships of Topography, Air Flow, and Monitoring Site Selection

Station Category	Characterization
A (ground level)	Heavy pollutant concentrations, high potential for pollutant buildup. A site 3-5 m (10-16 ft) from a major traffic artery that has local terrain features restricting ventilation. A sampler probe that is 3-6 m (10-20 ft) above ground.
B (ground level)	Heavy pollutant concentrations, minimal potential for a pollutant buildup. A site 3-14 m (15-50 ft) from a major traffic artery, with good natural ventilation. A sampler probe that is 3-6 m (10-20 ft) above ground.
C (ground level)	Moderate pollutant concentrations. A site 15-60m (5-200 ft) from a major traffic artery. A sampler probe that is 3-6 m (10-20 ft) above ground.
D (ground level)	Low pollutant concentrations. A site $\geq$ 60 m ($\geq$ 200 ft) from a traffic artery. A sampler probe that is 3-6 m (10-20 ft) above ground.
E (air mass)	A sampler probe that is 6-45 m (20-150 ft) above ground. Two subclasses: (1) good exposure from all sides (e.g., on top of a building), or (2) directionally biased exposure (probe extended from a window).
F (source-oriented)	A sampler that is adjacent to a point source. Monitoring that yields data directly relatable to the emissions source.

Source: Table 6.5 of EPA's *Quality Assurance Handbook for Air Pollution Measurement Systems*[6]

When source location is the goal of monitoring, the siting of a monitor depends on the meteorological conditions and the spatial locations of suspected sources. Again, the hypothetical example in Exhibit 10-3 provides some insights. If the source is suspected to be at the center of the geographic area, and if the wind direction is predominantly towards the east (as it is in that example), the monitor or sampler would be located to the east of the source and operated both at times when the wind blows towards the east and when the wind blows in the opposite (or another) direction. Support for the claim that the source is located

at the origin, and dominates exposures in the area around the monitor, would then be strongest if the ambient concentration increases significantly when the wind blows towards the east and drops significantly when it blows in other directions. If the data did not indicate this effect, then the source is not at the center, or there is an additional, and perhaps more significant, source in the area.

- **Take into account shielding and concentrating effects.** Buildings, hills, and trees can have shielding and concentrating effects. These effects may cause assessors to underestimate exposure if either measurement sites are shielded from normal air flow or if these same structures produce high concentrations downwind due to lee effects. Unless there is a pattern of movement of people that make sites near buildings and other structures of particular interest, assessors should perform measurements away from the influence of these structures. It is particularly important to locate monitors away from such structures if the goal is to locate sources, as the flow patterns for air are highly complex near these structures, greatly complicating the ability to identify the source location from monitoring data.

- **Be aware that sources of air toxics from mobile sources (cars, trucks, etc.) can complicate measurements of ambient air concentrations produced by stationary sources**. For the estimates of exposures from stationary sources, it may be preferable to make measurements at locations away from roads. Monitoring should occur at distances ranging from 3 to 61 meters from a major traffic artery (see Exhibit 10-5). These roads provide, in a sense, a "background" level, or noise, above which the source must rise to create a discernible signal. Of course, if total ambient exposure from all sources is to be estimated, and the exposed population spends a significant fraction of time near roads, this factor may be captured by selecting a sample of sites near those roads.

- **Make sure that the heights of monitoring and sampling devices are consistent with the breathing zones of people when public exposures are being evaluated**. This is generally between 1 and 2 meters (the lower end being for children and the upper end for adults). While less important for highly dispersed gases (i.e., gases with high diffusion coefficients), this consideration can be important for heavy gases and particulates, which produce significant vertical gradients of concentration.

- **Keep in mind that background concentrations can be difficult to determine**. Although background concentrations can be difficult to determine, it is important to estimate this factor as accurately as possible at the location of measurement (see below for a discussion of background concentrations). Unfortunately, even background levels can vary dramatically over time and over a geographic area, and so assessors should exercise caution in using past studies and studies from other geographic areas in establishing background for a measurement location. Meteorological and pollutant source information must also be carefully considered in selecting an appropriate background monitoring location. The location must not be near major sources of the contaminant, or in the predominant down-wind direction of those sources. The number of background samples should be determined during planning/scoping/problem formulation stage, and be based on statistical testing criteria specified in the DQOs.

The choice of monitoring or sampling locations depends on the spatial scale of the assessment being supported by the measurement program (i.e., micro, middle, neighborhood, urban, regional, or national). Note that samples collected (generally) at the micro-scale, middle-scale, or neighborhood-scale for the specific purpose of determining the impact of a source or co-located groups of sources on a specific population are called **source-oriented monitoring samples**.

In each case, selection of sites for the monitoring program should consider whether:

- A **mean value** is needed for a region (in which case, the sampling must be sufficient to allow interpolation of a surface concentration across that region, from which a mean may be estimated, or a mobile monitor/sampler must be used while moving throughout the region).

- A **mean value** is needed for an area. In this case, the monitor would be placed so as to capture the average of all the sources in the area (i.e., it is usually not oriented towards one source).

- A **maximum value** is needed (for example, for a screening assessment or an estimate of the maximum exposure to an individual from a particular source or co-located groups of sources; in this case, the task is to identify a location as close as possible to this point of maximal exposure).

- A **distribution of exposures** across the population in the region is needed, in which case sampling might be performed across a region. Information on the number of monitoring stations needed to perform this analysis with an acceptable level of accuracy/precision was recently evaluated and discussed by the Lake Michigan Air Directors Consortium (http://www.ladco.org/toxics.html).

- A **test of a model** is being conducted (in which case the location is selected to provide the most meaningful and unambiguous test of the model predictions under established source term and meteorological conditions).

In all five cases above, it is important to determine compounds that might interfere with the measurement of target compounds and, to the extent feasible, locate sampling devices in areas where such interference is small (without compromising the need to cover a geographic region). It also is important to establish one or more "background" and/or "control" locations so the elevation of concentrations or exposures at sampling locations due to sources not located in the assessment area can be determined.

In each case, site selection can improve through use of release data (source terms) and dispersion models. An accurate estimate both of average exposures and distributions of exposure (i.e., concentration measured across different monitors) generally will require adequate sampling in geographic regions characterized by the highest concentrations in addition to sampling in less impacted areas. Since such regions may represent a small fraction of the area in the overall study region, it may be necessary to "over-sample" in the highest exposed areas to ensure the points of maximal exposure are not missed. This process might be accomplished, for example, by sampling on a grid, with the grid density higher in the area surrounding the suspected point of maximal exposure; this will be particularly important if initial monitoring/sampling indicates high spatial variability in the area around the point of maximal exposure. For example, regions

near known, large emissions sources, and downwind of the predominant wind direction, should probably receive increased attention in sampling if a distribution of concentration is being developed across a larger assessment area. If samples were taken only in relatively non-impacted areas, the resulting distribution might not reflect the actual exposure of many area residents. (Ultimately, this is one of the prime reasons for using modeling to evaluate exposure; namely, that models can estimate exposure concentration at as many geographic points in a assessment area as the analyst wishes and for which sufficient emissions inventory data and computing power are available. Thus, modeling obviates these monitoring concerns.)

Background and Control Samples

Background monitors are monitors that are place in the predominant upwind direction (relative to sources) in the assessment area to measure the concentrations of the COPC in air that is moving into the assessment area. The results of such monitoring is helpful in understanding the monitoring results obtained in the assessment area; however, background monitoring results should not be subtracted from assessment area monitoring results because of the uncertainties in the background monitor as a truly representative measure of long term ambient background concentrations. Instead, EPA recommends bar charts that compare contemporaneous concentrations of a chemical in a background monitor to the same chemical at assessment area monitors; these charts provide a sense of the potential influence of background concentrations on the assessment area.

Unlike a background monitor, which is located upwind of the assessment area, a **control monitor** is located within the assessment area and is sited in such a way as to determine the average concentration of all pollutant sources, once mixing has occurred (including chemicals blowing into the assessment area from outside sources, mobile source emissions, and stationary source emissions within the assessment area). Control monitors should be located away from direct influence of any one or group of sources in the assessment area. Similar to background monitoring results, control monitor results should not be subtracted from other assessment area monitoring results (or modeling results). Instead, a simple bar chart comparison is usually adequate to compare the general "urban soup" to more focused monitors.

For the case of model testing, random sampling is not required or even desired. Instead, sampling is performed specifically in one or more locations where the conditions of emissions and dispersion are well established, and where there are no interfering sources or compounds. An ideal situation is a single, known source and a stable wind pattern during the period of sampling. Even in such cases, however, it will be necessary to provide a sampling grid covering the plume dimensions, since small errors in assigning wind direction can result in significant differences between model results and measurements. By sampling at a variety of locations in the plume, it is possible to adjust the model to determine whether a better fit might be obtained by more accurate information on the wind field, effective stack height, and other parameters.

As part of the national-scale assessment component of the 1996 National-Scale Air Toxics Assessment (NATA) activities, EPA compared monitoring to modeling results by using selected locations and compounds (seven HAPs) throughout the U.S. (see www.epa.gov/ttn/atw/nata/mtom_pre.html). The comparison goal was to assess the closeness of modeling and monitoring results, which would expose the overall uncertainty in estimating exposures. They found, for example, that modeled results generally underestimated results at monitors when the modeling was performed to predict air concentrations at the precise location

of the monitor; however, results were more comparable when the maximum concentration that the model predicted was compared against the maximum monitor concentration, without the requirement that modeling and monitoring be at the same location. These results indicate that uncertainties in the modeling produced errors that shifted the location of the point of maximal exposure, but not necessarily the magnitude of maximal exposure. A significantly more detailed uncertainty analysis currently is underway, with results expected in 2004 (these will be available at the NATA website).

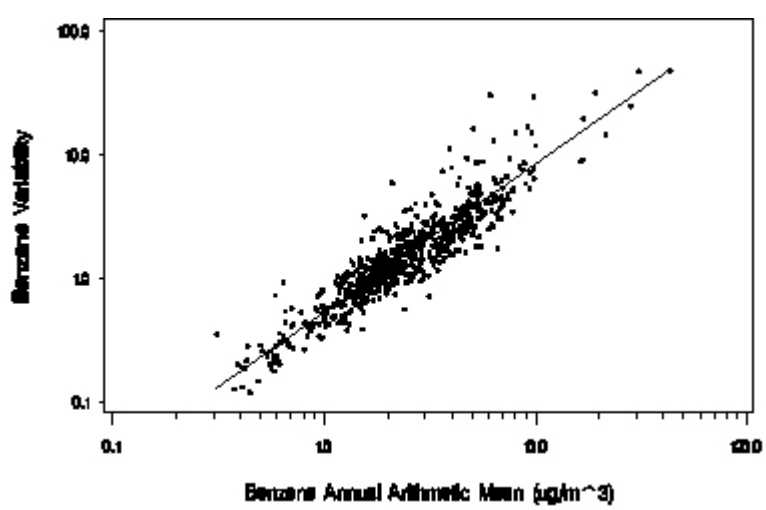
10.5.1.2 Selecting Sample Size

With respect to determining the quality of any estimates of mean concentration or exposure at a location, the coefficient of variation (CV) should be calculated to determine the number of samples needed to meet DQOs established by the decision problem. If σ is the standard deviation of a set of N measurements performed randomly throughout a geographic region and randomly in time, and μ is the mean for that sample set, the value of CV is:

$$CV = \frac{\frac{\sigma}{\sqrt{N}}}{\mu} \qquad \text{(Equation 10-1)}$$

The target value of CV depends on the decision criteria establishing the needed accuracy of an estimate of concentration or exposure, but a general target of less than 0.5 (50 percent) is suggested and a value of 0.2 or less should be possible. (This discussion assumes that the samples are representative of the geographic area and time period for which the average is being calculated.)

The above calculation of CV requires knowledge of σ and μ, which can only be obtained after the sampling program has been underway. It is possible, however, to estimate σ from an initial guess of the mean concentration or exposure, μ, through regression functions such as those established by Bortnick and Stetzer.[5] An example of such a regression is shown below based on a scatter plot of data from benzene monitoring.



Note that σ increases as μ increases. The authors use a lognormal relationship between σ and μ:

$$\ln \sigma = \ln a + \ln \mu \quad or \quad \sigma = a\mu^2 \qquad \text{(Equation 10-2)}$$

They perform a weighted least-squares regression (solid line in the figure above) and obtain for the case of benzene:

$$CV = \frac{\frac{0.54}{\sqrt{N}}}{0.2\mu} \qquad \text{(Equation 10-3)}$$

The approximate size of N needed to produce the desired value of CV may then be estimated from the above equation if an estimate of μ is available from either past monitoring data, similar geographic regions, or models.

10.5.1.3 Setting Up a Monitoring/Sampling Program

While the design of a monitoring program will depend in many ways on the kind of monitoring to be conducted, there are some general aspects of all monitoring programs that assessors should consider. EPA guidance describes many of these issues in detail.[7]

The general aspects related to designing a monitoring program that supports risk assessment are developed and written down in the planning, scoping, problem formulation phase (particularly, much of the following information is included in the study-specific conceptual model and the analysis plan and QAPP for monitoring activities). This activity involves three steps: (1) identify the sources, including the contaminants, the concentrations, the timing and locations of releases, as well as the hypotheses you want to test (e.g., whether a source exists, its relative contribution to overall exposures, etc.); (2) determine the exposure pathways (which in the case of air monitoring is inhalation and perhaps dermal absorption through immersion in air); and (3) determine the receptors of interest, including any sensitive subpopulations, their locations, how they are exposed, and relevant health benchmarks (e.g., IURs or RfCs). The conceptual model can be used to identify where significant exposures are likely to occur to receptors of interest, which in turn helps to guide the selection of monitoring sites. The following steps are then often used to develop, conduct, and evaluate the results of monitoring:

1. Collect and review existing air monitoring information for the site. This information should include data on concentrations, sources, locations of receptors, and other environmental data (e.g., meteorological data) needed to guide decisions. The sources of these data will depend on the location of the site, but a good start is to consider results from some of the national monitoring networks.

2. Determine the level of sophistication needed by the monitoring program. This level is established in the QAPP and the DQOs. The sophistication might range from simple screening procedures (e.g., to determine whether there are any exposures of concern) to more sophisticated methods intended to develop accurate maps of exposure across the region.

3. Develop a clear air monitoring plan, including determining the following: types of air monitors (these depend on the compounds identified as being of interest); the number and

location of monitors; the frequency and duration of monitoring, sampling and analysis of samples; and any QA/QC procedures that must be in place to meet DQOs.

4. Develop a detailed, written plan for day-to-day activities related to how equipment will be maintained and calibrated, and how to document results and QA/QC procedures. The data maintenance plan should include development of a system of logbooks for entering data, along with procedures to ensure the data are entered correctly and the logbooks are archived. There should be a clear procedure for maintaining chain-of-custody for both the samples and the logged results.

5. Evaluate the air monitoring results for their validity and reliability, including summary indicators of data quality (e.g., the data qualifiers discussed elsewhere in this chapter), and summarize these results so decision-makers can understand this quality and ensure the quality meets decision needs. This evaluation should include a summary of the statistical procedures used and the air concentration results, and an estimate of uncertainty in results deemed usable by the analyst (including uncertainty due to monitoring equipment, handling of samples, and sample analysis).

There are a number of specific issues that arise in Step 3 above that relate to the development of the monitoring program. These issues are summarized here in roughly the order in which they would be approached in developing a real program:

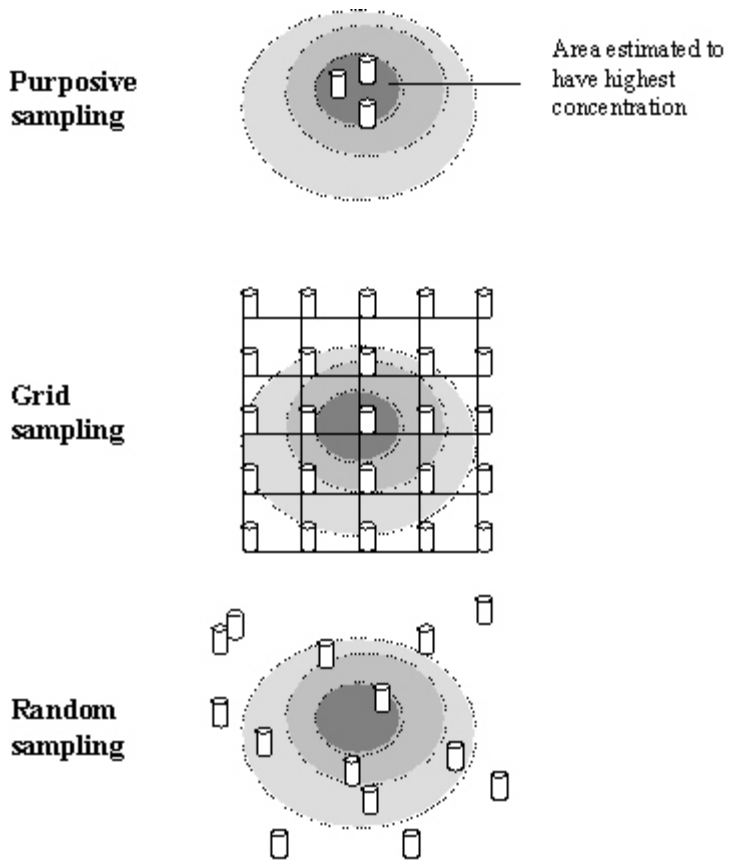
- **Establishing sampling locations**. Sampling may be purposive, random, or systematic. Purposive sampling refers to locating the monitor at a particular location because that location is of special interest. While such sampling can be useful to address specialized questions (such as the impacts of a specific source, or the reliability of model results), they generally are less useful for risk assessment purposes, and care should be taken when averaging the results along with results from the other forms of sampling. Random sampling involves selecting monitoring locations in a random and unbiased manner, with no correlation between locations (other than, perhaps, the fact that they are all in a defined region). Assessors could establish locations by creating a grid, and then randomly selecting the two coordinates (x and y) in that grid. Random sampling has the advantage of well established and relatively easy to apply statistical methods for evaluating results, but runs the risk of missing some "hot spots" of exposure. Systematic sampling involves establishing a grid and placing monitors systematically on the grid nodes. This ensures that sampling is uniform across an area, although statistical analysis is more complex because the samples are not truly random. Exhibit 10-6 illustrates common types of sampling programs.

A typical monitoring station, located at a site with easy access, power, and protection for the equipment

There also are practical considerations in selecting locations, regardless of which of the three procedures above is used. Monitors and samplers will require access to land, both in terms of

permission to locate the equipment and the ability to reach the site. It must also be possible to provide electrical power, and some protection of the equipment against theft, vandalism, and other disturbance; therefore, a fence may be needed.

Exhibit 10-6. Common Types of Sampling



Purposive sampling focuses the sampling effort in specific locations (in this example, the area estimated to have the highest concentration). Most air quality sampling is purposeful (i.e., monitoring stations are located in areas where monitoring is feasible (e.g., locations that are accessible), areas of direct (e.g., maximum) impact, or where concerns about potential exposures have been raised). Grid sampling consists of regularly-spaced samples in a predetermined grid. Random sampling consists of samples in locations selected by chance.

- **Determining the types of equipment and samples**. The sampling/monitoring method will depend on the compound being sampled, as well as the need for grab samples or composite (continuous) monitoring. See Section 10.6.1 for more detail on this issue.

- **Conducting field screening**. Before establishing the monitoring site, it is useful to conduct some limited screening of the region using relatively simply methods. This will help identify locations likely to be of interest (e.g., likely locations of maximal exposure). If this isn't possible, modeling results might be used. Guidance on this issue can be found in EPA's *Field Screening Methods Catalog*.[8] These results generally should not, however, be used in

the risk assessment of chronic exposures because a small number of samples taken over a short period of time will not provide an accurate estimate of long term exposure.

- **Accounting for temporal and meteorological factors**. Sampling must account for the fact that concentrations will fluctuate in time, in part because of meteorology (e.g., the wind blows in different directions during the day, carrying the contaminant to different locations). Where variability is high, a larger number of samples will be needed to achieve a desired level of accuracy. The sampling program should include a full annual cycle covering the seasons for a chronic exposure assessment. Where this is not possible due to limits on resources, the sampling should at least include two temporal extremes (e.g., under windy conditions blowing from major sources to the monitor, and under calm conditions). It is essential to include the variability of the samples in any estimates of accuracy for the monitoring location.

- **Implementing QA/QC measures**. It is essential that well-established, clear and documented methods for assuring the quality and reliability of data be developed. Many of these issues are described in the text box on the QAPP discussed in Section 10.3. A **sampling protocol** must be developed detailing (1) conditions under which samples are collected; (2) how training of individuals will be conducted; (3) how the precision and accuracy will be ensured so results are obtained reproducibly; and (4) the analytical strategies that will be used to ensure quantitation limits are met. Measures are also put into place to ensure that samples are handled appropriately from collection through analysis (e.g., chain-of-custody requirements, allowable sample holding times).

Field Blanks

A field blank is a clean sample, carried to the sampling site, exposed to sampling conditions, returned to the laboratory, and treated as an environmental sample. Field blanks are used to demonstrate that:

- Equipment cleaning has adequately removed contamination introduced by sampling at previous sites;
- Sampling and sample processing have not resulted in contamination; and
- Sample handling and transport, lab transport, and lab measurement have not introduced contamination.

Sampling devices used to collect, store, preserve, and transport samples must not alter the sample in any way that complicates analysis. Samples should be stored in a way that keeps the concentration as close as possible to that in the field. QC samples must be collected, stored, transported, and analyzed in a way that is identical to the treatment of site samples. For example, both field and trip blanks, which are sampling devices that have not been used for sampling in the field but otherwise are brought through all of the other procedures to which field samples will be subjected, must be treated identically to the actual field samples. These field and trip blanks provide information on the extent to which samples might become contaminated by non-site-related materials during handling in the field (field blanks) and subsequent transport back to the lab for analysis (trip blanks).

10.5.2 Data Analysis and Reporting

As Section 10.4.1 mentions, adequate data analysis, recording, and archiving is essential to the design and conduct of a monitoring program. It is important that assessors enter each data point

into a file with relevant qualifiers, including location of sample; date and time of sample; method of sampling and relevant operating characteristics (e.g., flow rate); transfer process; storage time; analysis method; and identity of people performing all stages of the measurement. The integrity of this database should then be assessed periodically by comparing a random sample of file information against hard copies (e.g., laboratory books) to ensure reliability of transcription. For results using a common methodology, there should be a record of several key aspects of the method that assure reliability:

- A description of the calibration process, including certification of any standards used in that calibration.

- Results of any inter-laboratory comparison of uses of the method, and certification that the laboratory performing the analysis for the sampling program falls within a reasonable range of these inter-laboratory results.

- A record of background levels and levels in blanks, allowing a comparison of these against sample results.

- A summary of the frequency of "detects," or fraction of samples with values above the MDL or SQL (see Exhibit 10-7). If this fraction is small and the chemical is thought to be present, it may indicate that improvements in the method are needed. Of course, if the SQLs are well below any health benchmark, a small fraction of detects or quantifiable results need not trigger a call for improvements.

- A policy on significant digits and how these are related to the accuracy of the method. All results should be reported only with a number of digits consistent with this accuracy. In addition, rounding rules should also be established and followed.

- A description of how summary quantities such as means are calculated. This description includes such factors as how outliers are identified and dealt with, the possible influence of this process on sample mean and variance, and how results below the SQL are handled. For example, some laboratories will report a chemical that they detect below the SQL as "not detected" simply because it is below the SQL and they cannot accurately quantify it. Other labs will report such a chemical as detected, but with an estimated concentration and qualify the value as "J." In general, labs should report detected chemicals, regardless of whether they can accurately quantify their concentration. The use of J-qualified data for risk assessment purposes is described below.

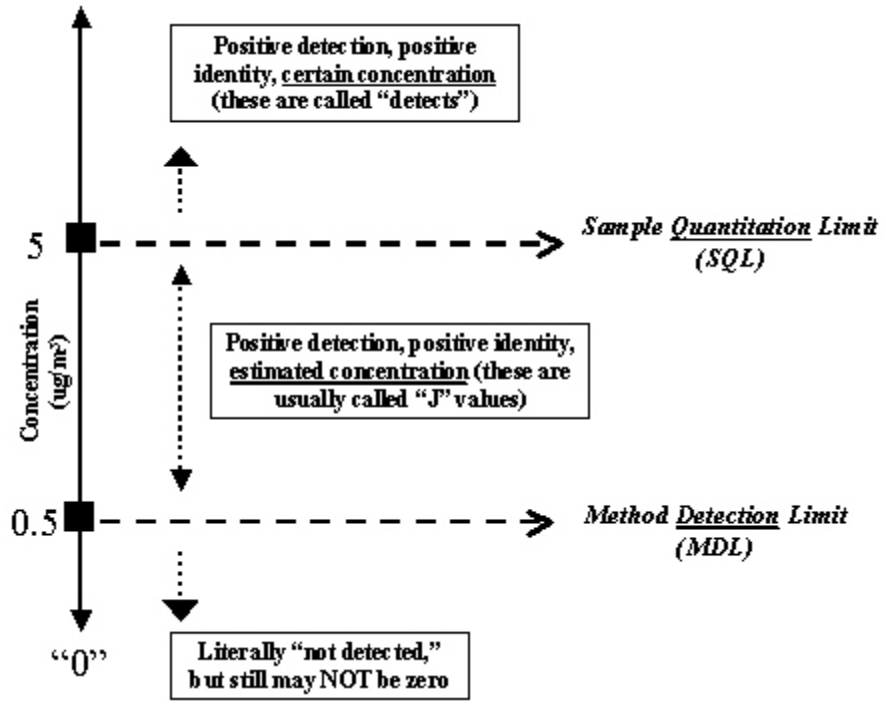
- A detailed description of the QA/QC flags that are used by the lab to report data and a clear description of how the lab deals with samples that are associated with blanks that are contaminated.

10.5.3 The Use of Monitoring Data to Calculate Exposure Concentrations

As the above noted, monitoring data can, under limited circumstances, be used to estimate exposure concentrations in the vicinity of the monitor. Some general rules that apply to this activity are as follows:

- Data from different monitors should not be combined to estimate exposure concentrations (with the exception of co-located duplicate monitors – see below).

Exhibit 10-7. Illustration of SQL and MDL



People often refer to the SQL as the "detection limit." When a lab reports a result as "undetected" (U) or "not detected" (ND), this is the level to which they are usually referring. The SQL is really a limit of quantitationthat can vary between samples (the MDL, on the other hand, is a true "detection limit"). For example, a result of "5U µg/m3" usually means "not detected at a sample-specific quantitation limit of 5 µg/m3." When a chemical is detected, but below the SQL (i.e., a "J" value), risk assessors often use the J value as is (i.e., the J value is used with no modification). When a chemical is not detected in a sample, but there is reason to believe it may be present, even at very small amounts (e.g., the chemical is found in some samples, but not in others), risk assessors often use ½ the SQL as a surrogate concentration for risk assessment purposes (in the example above, half of "5U" is 5/2 = 2.5). It is usually not appropriate to use ½ the MDL as a surrogate for concentration for exposure assessment purposes. The process of assessing and combining monitoring data for exposure assessment purposes is discussed in more detail in Appendices H and I.

- Monitoring data at a location are not generally used to describe variation of exposure concentrations experienced by individuals in a population of people, although temporal differences for the population as a whole (e.g., exposure to the population during the winter versus exposure to the population during the spring) may be appropriate. Variation in exposure concentration within a population is preferably described by looking at exposure concentrations across a set of monitors in the assessment area.

- The representativeness of the exposure concentrations, as represented by any one monitor's data, depends on the amount and quality of the data collected, and the individual chemicals involved. For example, some pollutants may be "regional" in nature, meaning that their concentration tends to be relatively homogeneous over a large area. In that case, a given monitor may be broadly representative of ambient concentrations throughout the region. Some compounds, on the other hand, show sharp

concentration gradients over space and the monitor may only be reflective of exposure concentrations for people living very near to the monitoring station.

- To assess acute exposures with monitoring samples, the results from the individual samples (not their average) should be compared to acute health benchmarks, and the sampling time should match the averaging time of the acute health benchmark (see Chapter 13).

- For chronic exposure assessment, all the valid samples collected and analyzed for a monitor (taken routinely throughout the course of at least one year) are averaged (see below) to provide an estimate of the long term exposure concentration.

Appendix I provides a general overview of how monitoring data should be evaluated, processed, and displayed to develop estimates of exposure concentration.

10.6 Monitoring Methods, Technologies, and Costs

EPA has developed a number of methods to measure the concentration of air toxics in ambient air. The majority of this information is found on EPA's Ambient Monitoring Technology Information Center (AMTIC) website (Exhibit 10-8), and assessors involved in monitoring should become familiar with this website and its contents. Given the breadth and scope of this website's contents, it is not possible here to fully review all of the information here. This section only provides an introduction to the methods. Appendix E summarizes relevant information from two key EPA compendia of methods, primarily for ambient air monitoring. In addition, this chapter does not examine indoor air measurements, as EPA has provided monitoring recommendations only for radon.

EPA has developed a *Compendium of Methods for the Determination of Toxic Organic Compounds in Ambient Air* to assist federal, state, and local regulatory personnel in developing and maintaining necessary expertise and up-to-date monitoring technology for characterizing organic pollutants in the ambient air (Exhibit 10-9).[9] The Compendium contains a set of 17 peer-reviewed, standardized methods for the determination of volatile, semi-volatile, and selected toxic organic pollutants in the air. The Compendium, along with updates and addenda, is available at EPA's AMTIC Website at http://www.epa.gov/ttn/amtic/airtox.html.

Exhibit 10-8. EPA's Ambient Monitoring Technology Information Center (AMTIC)
Information on ambient concentrations for a wide variety of compounds can be found through AMTIC (http://www.epa.gov/ttn/amtic/welcome.html). This Center facilitates the exchange of ambient monitoring-related information collected throughout the U.S., and can provide valuable insights into the selection of monitoring methods. Established in 1991 as an electronic bulletin board system (BBS), AMTIC has evolved with changing technology into a page on the World Wide Web. It is operated by EPA's OAQPS through the Monitoring and Quality Assurance Group (MQAG). The database contains information on all the Reference and Equivalent Methods for the criteria pollutants, the toxic organic (TO) Methods for air toxics and other noncriteria pollutant methodologies, Federal Regulations pertaining to ambient monitoring, ambient monitoring QA/QC related information, information on ambient monitoring related publications, ambient monitoring news, field and laboratory studies of interest, and updates on any new or developing EPA Ambient Air standards.

Exhibit 10-9. EPA's Toxic Organic (TO) Monitoring Methods	
Method	**Description**
TO-1	Method for the Determination of Volatile Organic Compounds (VOCs) in Ambient Air using Tenax® Adsorption and Gas Chromatography/Mass Spectrometry (GC/MS)
TO-2	Method for the Determination of VOCs in Ambient Air by Carbon Molecular Sieve Adsorption and Gas Chromatography/Mass Spectrometry (GC/MS)
TO-3	Method for the Determination of VOCs in Ambient Air using Cryogenic Preconcentration Techniques and Gas Chromatography with Flame Ionization and Electron Capture Detection
TO-4A	Determination of Pesticides and Polychlorinated Biphenyls in Ambient Air Using High Volume Polyurethane Foam (PUF) Sampling Followed by Gas Chromatographic/Multi-Detector Detection (GC/MD)
TO-5	Determination of Aldehydes and Ketones in Ambient Air Using High Performance Liquid Chromatography (HPLC)
TO-6	Determination of Phosgene in Ambient Air Using High Performance Liquid Chromatography (HPLC)
TO-7	Method for the Determination of nitrosodimethylamine (NDMA) in Ambient Air Using Gas Chromatography
TO-8	Method for the Determination of Phenol and Methylphenols (Cresols) in Ambient Air Using High Performance Liquid Chromatography
TO-9A	Determination of Polychlorinated, Polybrominated, and Brominated/Chlorinated Dibenzo-p-Dioxins and Dibenzofurans in Ambient Air
TO-10A	Determination of Pesticides and Polychlorinated Biphenyls in Ambient Air Using Low Volume Polyurethane Foam (PUF) Sampling Followed by Gas Chromatographic/Multi-Detector Detection (GC/MD)
TO-11A	Determination of Formaldehyde in Ambient Air using Adsorbant Cartridge Followed by High Performance Liquid Chromatography (HPLC)
TO-12	Method for the Determination of Non-methane Organic Compounds (NMOC) in Ambient Air Using Cryogenic Preconcentration and Direct Flame Ionization Detection (PDFID)
TO-13A	Determination of Polycyclic Aromatic Hydrocarbons (PAHs) in Ambient Air Using Gas Chromatography/Mass Spectrometry (GC/MS)
TO-14A	Determination of VOCs in Air Using Specially Prepared Canisters with Subsequent Analysis by Gas Chromatography
TO-15	Determination of VOCs in Air Collected in Specially-Prepared Canisters and Analyzed by Gas Chromatography/Mass Spectrometry (GC/MS)
TO-16	Long-Path Open-Path Fourier Transform Infrared Monitoring of Atmospheric Gases
TO-17	Determination of VOCs in Air Using Active Sampling Onto Sorbent Tubes

10.6.1 Ambient Air Monitoring Methods and Technologies

The term "monitoring method" is a comprehensive term that includes everything from the sample collection devices to analytical laboratory methods. These methods fall into three broad categories related to the **time scale over which concentration will be averaged**:

- **Grab samples** provide a quasi-instantaneous measurement of a concentration. They generally are obtained in the field usually over a period of 24 hours or less and then returned to the laboratory for analysis. The sampling may be automated, allowing a time-series of samples to be drawn, but all samples still are generally returned to the laboratory for analysis. In rare instances, a mobile laboratory may be co-located with the sampling location, in which more "real-time" data is possible.

- **Continuous monitors** provide a time series of measurements in the field, with a stream of data at selected intervals (e.g., once each 24 hours). These monitors may be fully automated versions of grab sampling, taking samples at a set interval but then analyzing the samples internally rather than returning to the lab. An alternative is a continuous flow monitors, which draw ambient air through a chamber and analyzes it in real time (e.g., the semi-continuous formaldehyde monitor developed by the EPA, which runs through one complete cycle of sampling and analysis in 10 minutes).

- **Time-integrated samples** are collected over an extended period of time. Only the total pollutant collected is measured, and so only the average concentration during the sampling period can be determined. As with grab samples, these measurements generally are obtained in the field and returned to a laboratory for analysis.

Monitoring methods/systems can also be divided into a different set of categories based on the **method of collection**:

- **Integrated air sampling devices** use a pump to draw air continuously into the sample chamber, over a reactive medium, or through a filter during a prescribed period of time; the sample is returned to the laboratory for analysis.

- **Direct-read monitors** draw air through a measurement system and provide a direct reading of the concentration without returning samples to the lab.

- **Automated monitoring systems** collect samples, perform the analysis, and report results at regular intervals in the field.

- **Air deposition monitors** rely on deposition properties of compounds (e.g., particulates), and may consist of active and/or passive, wet and/or dry sampling methods.

- **Passive monitors** allow the compound to diffuse into contact with an active material; these generally are analyzed in the lab, although some indicate the presence of a compound by a color change.

- **Grab sampling devices** use an essentially instantaneous sampling method, such as an evacuated chamber into which ambient air is allowed to enter at a fixed rate; the sample collected is returned to the laboratory for analysis.

In some circumstances, grab samples may be collected by volunteers (for example, when residents near an industrial complex organize to capture samples when a strong odor is present). This process is commonly referred to as a "bucket brigade." Bucket brigades may provide useful information that a problem may exist that warrants more in-depth evaluation. They are also helpful, in some circumstances, to help the affected community become more involved in the air toxics evaluation process. Nevertheless, care should be taken to ensure that all of the necessary sampling and analysis protocols and QA/QC are established, understood, and followed by the bucket brigade team members to ensure that the grab samples are of sufficient quality to be used for decision making purpose at hand.

Mobile air monitoring platforms are sometimes used to evaluate air quality parameters. A "mobile platform" can be anything from a VOC sampling apparatus on a movable trailer to a sophisticated multi-pollutant sampling and analytical mobile trailer. The utility of mobile platforms is that they can be moved from place to place relatively easily (e.g., for hotspots analysis) and may only require a place to park the platform and an electrical hookup (as opposed to the more difficult process of establishing fixed monitoring locations, which requires access to land, often by establishing a leasing agreement, and permanent security measures, such as fencing).

Most existing air toxics monitoring programs have focused on the 188 HAPs, and especially on the 33 urban HAPs identified by OAQPS on a nationwide basis (Exhibit 10-10) as generally presenting the greatest contribution to risk to public health from air toxics in urban areas. Note that the highest-risk HAPs in a specific region or community may differ from this list. A significant database exists on national exposures to these compounds, especially those monitored by the National-Scale Air Toxics Assessment (see Chapter 2 and the website at www.epa.gov/ttn/atw/nata). A general starting point for most monitoring efforts should be an initial screening analysis to identify the COPCs. A description of the general process for screening analyses of this type is provided in Chapter 1.

Exhibit 10-10. 33 Urban HAPs (Nationwide Basis)

acetaldehyde	*formaldehyde*
acrolein	hexachlorbenzene
acrylonitrile	hydrazine
arsenic and compounds	*lead and compounds*
benzene	*manganese and compounds*
beryllium and compounds	mercury and compounds
1,3-butadiene	*methylene chloride*
cadmium and compounds	*nickel and compounds*
carbon tetrachloride	polychlorinated biphenyls
chloroform	polycylic organic matter
chromium and compounds	propylene dichloride
coke over emissions	quinolene
1,2-dichloropropane	1,1,2,2-tetrachloroethane
dioxin	*tetrachloroethylene*
ethylene dibromide	*trichloroethylene*
ethylene dichloride	*vinyl chloride*
ethylene oxide	

Compounds monitored in the NATA National Scale Assessment pilot sites are indicated by *italics*.

EPA has not developed methods for many compounds, including some of the 33 urban HAPs. Potential deficiencies in particular monitoring methods include:

- Quantitation limits are not low enough relative to environmental levels and/or health benchmarks;
- Lack of available standards for monitoring protocols (e.g., standards developed by the National Institute of Science and Technology);
- Methods are not practical or easy to implement;
- Compound stability is so poor that the compound degrades significantly between the time it is collected and the time it is analyzed, resulting in poor to no recovery at the time of analysis;
- Recover efficiencies are too low, resulting in poor precision and/or quantitation limits that are not low enough for use relative to health benchmarks;
- Methods have not been sufficiently tested in the laboratory and field;
- Methods are not producing results that are comparable to established methods; and
- Poor reliability.

The deficiencies noted in Exhibit 10-11 are particularly important and have been identified by EPA as needing methodology development.[10] Because they present a similar challenge, EPA has targeted several VOCs for programs to improve monitoring capabilities (Exhibit 10-12). In addition, both diesel exhaust (a complex mixture), acrolein, and arsenic require additional method development to yield accurate, reliable, and field-tested monitoring methods.

10.6.2 Sampling Costs

There is no general guideline for the costs associated with monitoring programs, as they depend on quite an array of factors. Several of the more critical include:

- Whether samples are analyzed "in house" or contracted out.
- Whether monitoring equipment is available or must be purchased or leased.
- The number of monitoring results or samples required (there is some economy of scale, but increased numbers of results also increases cost).
- Whether personnel must be hired and/or trained.
- The potential cost of leases and insurance for monitoring sites.
- Laboratory analytical costs for special analytes. For example, dioxin samples can run as high as $1,000 per sample, making an extensive dioxin sampling scheme generally out of reach for most studies.

10.7 Archiving Air Toxics Monitoring Data

When appropriate, results of a monitoring program should be submitted to the relevant air toxics database, such as EPA's Air Quality System (AQS).[11] The AQS website (www.epa.gov/ttn/airs/airsaqs/sysoverview.htm) provides detailed information on submitting and retrieving such data, including instructions on the file format for the data. Archived data may be accessed at the AQS site.[12]

Exhibit 10-11. Identified Deficiencies in Available Monitoring Methods		
Compound	**Candidate Method**	**Deficiency**
1,3-butadiene 1,2-dibromoethane 1,2-dichloroethane	TO14A/15	sensitivity issue; false highs
acrylonitrile	TO14A/15	NIST standard needed; recovery problems
ethylene oxide	None/NIOSH 1614	poor storage stability
1,1,2,2-tetrachloroethane	TO-15	NIST standard needed
arsenic and compounds	IO-3	sensitivity issues; filter contamination; resource intensive
beryllium and compounds	None	resource intensive; XRF sensitivity issue
mercury and compounds	IO-5	requires special equipment
acrolein	None	TO-11A results in unstable derivative poor recovery
2,3,7,8-tetrachlorodibenzo-p-dioxin	TO-9A	resource intensive

Exhibit 10-12. VOC Compounds Needing Improved Monitoring Methods	
vinyl chloride 1,2-dichloroethene dichloromethane chloroform 1,2-dichloroethane benzene carbon tetrachloride 1,2-dichloropropane trichloroethene	cis- and trans-1,3-dichloropropene 1,1,2-dichoroethane 1,2-dibromoethane tetrachloroethylene 1,1,2,2-tetrachloroethane hexachlorobutadiene acrylonitrile 1,3-butadiene ethylene oxide

10.8 Using Air Monitoring Data to Evaluate Source Contribution

Caution should be used in interpreting the results of a measurement as being uniquely associated with a given source. Most measurements from monitoring data are, depending on the chemical, a combination of background concentrations and the same chemical released from possibly multiple sources. Benzene, for example, is present in background air, is released from mobile sources, and is used and released from multiple types of stationary sources. This is not to say that monitoring data cannot be used to identify releases from a source. Under certain circumstances, analysis of multiple measurements at different locations may indicate a spatial pattern consistent with the known air dispersion pattern accompanying that source (and inconsistent with the patterns from other sources).

Use of Historical Monitoring Data

Historical monitoring data for an assessment area may be of use in developing the analysis plan. They can help with a range of uses, including:

- Identifying the types of chemicals that may be present in the air;
- Selecting locations for monitors;
- Performing preliminary screening level risk estimates; and
- Establishing acceptable monitoring protocols.

The utility of historical data will, of course, be based on an assessment of the quality of the data. For example, data that were not collected with sufficient QA/QC, may not be useful for any of the above purposes.

EPA also has developed "receptor models" which make use of monitoring data, together with emissions inventories, to perform source apportionment analyses, which provide a quantitative estimate of what percent of each pollutant comes from each identified source. EPA's Chemical Mass Balance Model is one such example (available on EPA's SCRAM website at http://www.epa.gov/scram001/tt23.htm). This model uses chemical concentrations measured in samples from sources (emissions) and receptor locations to estimate the contributions of source types to ambient air pollutant concentrations. The model is used primarily in the development of State Implementation Plans for PM_{10}. The model allows the user to select samples, chemical species, and source types for modeling, calculate source contributions and their standard errors, evaluate goodness-of-fit and validate the model results, prepare output documentation, and graph results.

References

1. U.S. Environmental Protection Agency. 2004. *National Air Toxics Monitoring Strategy, Draft.* Office of Air Quality Planning and Standards, Research Triangle Park, NC, January 2004. Available at: http://www.epa.gov/ttn/amtic/files/ambient/airtox/atstrat104.pdf.

2. U.S. Environmental Protection Agency. 2003. *Latest Findings on National Air Quality; 2002 Status and Trends.* Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina. EPA 454/ K-03-001. Available at: http://www.epa.gov/air/airtrends /2002_airtrends_final.pdf

3. U.S. Environmental Protection Agency. 2004. Technology Transfer Network Ambient Monitoring Technology Information Center. Updated March 4, 2004. Available at: http://www.epa.gov/ttn/amtic/airtxfil.html. (Last accessed March 2004.)

4. U.S. Environmental Protection Agency. 2003. *Laboratory Operations and Quality Assurance Manual.* Science and Ecosystem Support Division, Analytical Support Branch, Region 4, Athens, GA. January 2003. Available at: http://www.epa.gov/region4/sesd/asbsop /asbsop.html

   U.S. Environmental Protection Agency. 2003. *Quality Assurance Management Plan for Region 4.* U.S. Environmental Protection Agency, Region 4, Atlanta, GA. May 2003. Available at: www.epa.gov/region4/sesd/oqa/r4qmp598.html.

5. S. Bortnick and S. Stetzer. 2002. *Monitoring Methods and Network Design*. Air Toxics Exposure Assessment Workshop. June 25-27, 2002. San Francisco, California.

6. U.S. Environmental Protection Agency. 1998. *Quality Assurance Handbook for Air Pollution Measurement Systems, Volume II, Part 1: Ambient Air Quality Monitoring Program Quality System Development.* Office of Air Quality Planning and Standards, Research Triangle Park, NC, August 1998. EPA-454/R-98-004. Available at: http://www.epa.gov/ttn/amtic/files/ ambient/qaqc/redbook.pdf

7. U.S. Environmental Protection Agency. 1989. *Risk Assessment Guidance for Superfund, Volume I, Human Health Evaluation Manual (Part A), Interim Final*. Office of Emergency and Remedial Response, Washington, D.C., December 1989. EPA/540/1-89/002. Available at: http://www.epa.gov/oerrpage/superfund/programs/risk/ragsa/index.htm.

   U.S. Environmental Protection Agency. 1989. *Procedures for Dispersion Modeling and Air Monitoring for Superfund Pathway Analysis* (Volume IV of *Procedures for Conducting Air Pathway Analyses for Superfund Applications).* Office of Air Quality Planning and Standards, Research Triangle Park, NC. EPA/450/1-89/004.

8. U.S. Environmental Protection Agency. 1988. *Field Screening Methods Catalog: User's Guide*. Office of Emergency and Remedial Response, Washington, D.C., September 1988. EPA 540-2-88-005 (NTIS / PB89-134159) .

9. U.S. Environmental Protection Agency. 1999. *The EPA Compendium of Methods for the Determination of Toxic Organic Compounds in Ambient Air*. Center for Environmental Research Information, Washington, D.C., January 1999. EPA/625/R-96/10b. Available at: http://www.epa.gov/ttn/amtic/airtox.html.

U.S. Environmental Protection Agency. 1999. *The EPA Compendium of Methods for the Determination of Inorganic Toxics Compounds in Ambient Air*. Office of Research and Development, Washington, D.C., June 1999. EPA/625/R-96/01a. Available at: http://www.epa.gov/ttn/amtic/inorg.html.

10. Whitaker, D. 2002. New Trends in Monitoring Methods. Air Toxics Exposure Assessment Workshop. June 25-27, 2002. San Francisco, California.

11. U.S. Environmental Protection Agency. AirData. *About the AQS Database*. Updated October 3, 2003. Available at: http://www.epa.gov/air/data/aqsdb.html (Last accessed March, 2004.)

12. U.S. Environmental Protection Agency. 2004. Technology Transfer Network Air Quality System. *Download Detailed AQS Data*. Updated March 11, 2004. Available at: http://www.epa.gov/ttn/airs/airsaqs/archived%20data/downloadaqsdata.htm. (Last accessed March 2004.)

U.S. Environmental Protection Agency. 1999. *1997 Urban Air Toxics Monitoring Program (UATMP)*. Office of Air Quality Planning and Standards, Research Triangle Park, NC, January 1999. EPA-454/R-99-036. Available at: www.epa.gov/ttnamti1/files/ambient/airtox/r99-036a.pdf.

Chapter 11 Estimating Inhalation Exposure

Table of Contents

11.1 Introduction 1

11.2 Estimating Inhalation Exposure Concentrations 1
- 11.2.1 General Approaches for Deriving Exposure Concentrations 2
- 11.2.2 Common Ways to Estimate Exposure Concentrations 3

11.3 Exposure Modeling 6
- 11.3.1 Inhalation Exposure Modeling 9
- 11.3.2 Microenvironment Concentration: How is it Developed? 12
- 11.3.3 Sources of Data for Human Activity for Inhalation (and other) Exposure Assessments 13
- 11.3.4 Examples of Inhalation Exposure Models 16
- 11.3.5 Exposure Modeling Examples 19

11.4 Personal Monitoring 20

11.5 Exposure to a Population: Common Descriptors 22

11.6 Evaluating Uncertainty 22

11.7 Presenting the Results of an Exposure Assessment 23

References 23

11.1 Introduction

The previous three chapters discussed how to quantify exposure and release rates and estimate chemical fate and transport. This chapter discusses the final step of estimating exposure. This chapter will discuss inhalation exposure only. Unless persistent bioaccumulative hazardous air pollutants (PB-HAPs) are present in source emissions, most air toxics risk assessments will only estimate inhalation **exposure concentrations**. Limiting the exposure assessment this way is possible because the dose-response values that characterize inhalation risk (e.g., reference concentrations, inhalation cancer unit risk estimates – see Chapter 12) take into consideration the complex physical and pharmacokinetic processes that influence how the chemical reaches the target organ, which may be a region of the respiratory tract or a remote site (see Chapter 12 for a more detailed discussion). Specifically, other than exposure modeling to account for things like time in different microenvironments and microenvironment concentrations, no adjustment for other exposure parameters (e.g., body weight and inhalation rate) are warranted. For multipathway risk assessments, however, where ingestion intake rate is the exposure parameter, it will be necessary to consider parameters such as body weight and contact rate (e.g., amount of soil ingested, fish eaten) for the indirect exposure pathway metrics of exposure (see Chapter 19).

Assessors determine human exposure to an environmental pollutant via inhalation by estimating the concentration of that pollutant in the ambient air and the contact of an individual with that air (along with the characteristics of the contact). Because concentrations in the air vary over space and time, it is important to know where and how long people spend their time in relation to the contaminated air under study. Through air quality modeling and monitoring, the ambient concentrations of pollutants in air can be estimated geographically and temporally. Through the use of exposure modeling, estimates of exposure via the inhalation route can be adjusted from modeling data to take into account the demographics of people in the study area and the time they may spend in various microenvironments.

The remainder of this chapter discusses how to estimate inhalation exposure concentrations for the risk assessment (Section 11.2); exposure modeling (Section 11.3); personal monitoring (Section 11.4); common descriptors (Section 11.5); evaluating uncertainty (Section 11.6); and presenting the results of an exposure assessment (Section 11.7).

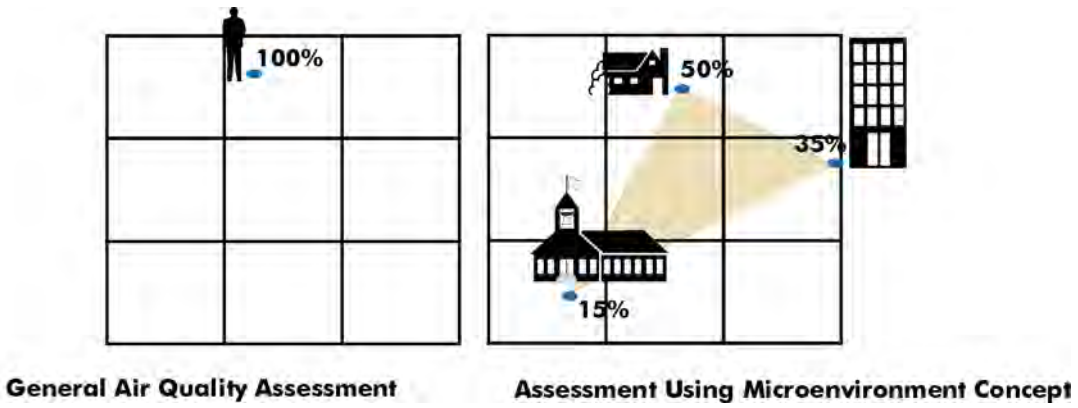
11.2 Estimating Inhalation Exposure Concentrations

The ambient air exposure concentrations (ECs) can be estimated using either (or both of) two general methods: air quality modeling and air quality monitoring. As discussed in Chapter 9, air quality modeling involves defining the pollutant sources and release characteristics and modeling pollutant fate and transport (how the air toxic is transported, dispersed, and transformed over the area of interest). As Chapter 10 discussed, monitoring involves measuring ambient concentrations of chemicals. Because of the time/expense and other limitations associated with monitoring (most notably, questions about representativeness), modeling is the most common approach for estimating ambient air concentrations to be used in the air toxics risk assessment. Monitoring is often used, instead, as a secondary tool to provide input data to the models and validate the model results and to look for important gaps in the emissions inventory used to run the model.

11.2.1 General Approaches for Deriving Exposure Concentrations

There are two general ways to derive the EC for a given risk assessment (see Exhibit 11-1). Both may incorporate the results of air quality modeling and/or monitoring efforts.

Exhibit 11-1. Two General Ways to Estimate Inhalation Exposure Concentration



The left-hand side illustrates the use of ambient air concentrations as a surrogate for the EC. In this example, the analysis assumes that individuals spend 100 percent of their time at a given location, so the estimate of ambient concentration thus represents the EC. The right-hand side illustrates the use of exposure modeling. In this example, the analysis assumes that an individual spends 50 percent of his/her time at home; 15 percent at a school; and 35 percent at an office. The EC is the weighted sum of the product of the ambient concentrations at each location and the amount of time spent there. Both indoor and outdoor concentrations usually are considered at each location.

- **Ambient Air Concentrations as a Surrogate**. For screening-level evaluations, assessors use the concentrations of air toxics generated at each modeling node (or interpolated nodes) or the concentrations determined by a monitor (if modeling is not performed) as surrogates of the inhalation exposure concentrations for the populations in the study locations. The default assumption in such a screening assessment is that the population of interest is breathing outdoor air continuously at the modeled or monitor location. This is believed to be a conservative assumption since indoor air concentrations of air toxics are expected to be the same or lower than the outdoor concentrations (when the indoor concentrations are produced solely by inflow from outside air).

- **Exposure modeling**. More comprehensive inhalation exposure assessments combine estimates of ambient pollutant concentrations (e.g., from air quality models) with information about the population of interest, including the types of people present (e.g., ethnicity, age, sex), time spent in different microenvironments, and microenvironment concentrations. The assessment objective is to identify a representative estimate of the pollutant concentration in the inhaled air in each microenvironment and combine it with an estimate of the time spent in different microenvironments (and the activities within these microenvironments) throughout the daily routine of different groups of people with similar attributes (called **cohorts**).

11.2.2 Common Ways to Estimate Exposure Concentrations

Risk assessors commonly use several different ways to estimate exposure concentrations. Some ways are used primarily for screening-level (Tier 1) assessments; others are used primarily for more refined assessments. Exhibit 11-2 illustrates several different ways to estimate exposure concentrations when ambient air concentrations are used as surrogates.

- **Monitoring locations**. Sites where air monitors are located provide a direct measure of ambient air concentrations at those locations. However, these locations may or may not be representative of ambient air concentrations in other parts of the study area. If monitors are not located where people live, the monitoring results may not be of much value for the risk assessment other than to check the accuracy of modeling. Monitoring results may be used as inputs to exposure modeling.

- **Point of maximum modeled concentration**. This is the modeling node where the maximum modeled ambient air concentration occurs, regardless of whether there is a person there or not. This generally provides a conservative estimate of exposure and could be used as the EC in a screening-level evaluation (for example, using the SCREEN3 model). This point can be used to provide an estimate of "high-end" exposure to the risk manager because, although no one may actually be living there at the present, someone might move their in the future. This point may be referred to as the point of the **"maximum exposed individual (MEI)."**

- **Point of maximum modeled concentration at an actual receptor location**. This is the modeling node where the maximum ambient air concentration occurs to an actual person in the area of impact, usually at an actual residence (or, if the residence falls between modeling nodes, an interpolated value). To identify this point precisely, it is necessary to know detailed information about the location of actual people in the study area. As with the point of maximum modeled concentration above, this point can be used to provide an estimate of "high-end" exposure to the risk manager (in this case, based on current actual exposures). This point may be referred to as the point of the **"maximum individual risk (MIR)."**

- **Census tract/block internal point**. The U.S. Census Bureau provides information about populations in geographic units called census tracts, which are subdivided into block groups/enumeration districts and blocks. In cases where there is only limited information about the census tract (e.g., nothing is known other than the number of people living within the tract), the Census Bureau's "internal point" (sometimes referred to as a centroid) for the tract typically is used as the point of exposure for all the population in the tract. The internal point is a set of geographic coordinates that generally represents the approximate geographic center of a geographic subdivison (see box on next page). The Census Bureau provides an internal point for each of its geographic subdivisions (i.e., tracts, blocks, and block groups). Note that the internal point **<u>is not population weighted</u>** (i.e., it is not located "in the direction of where the people are").

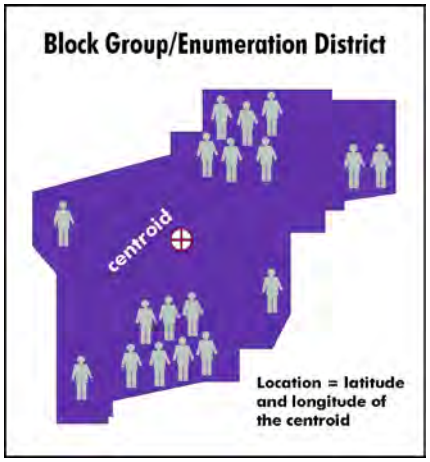
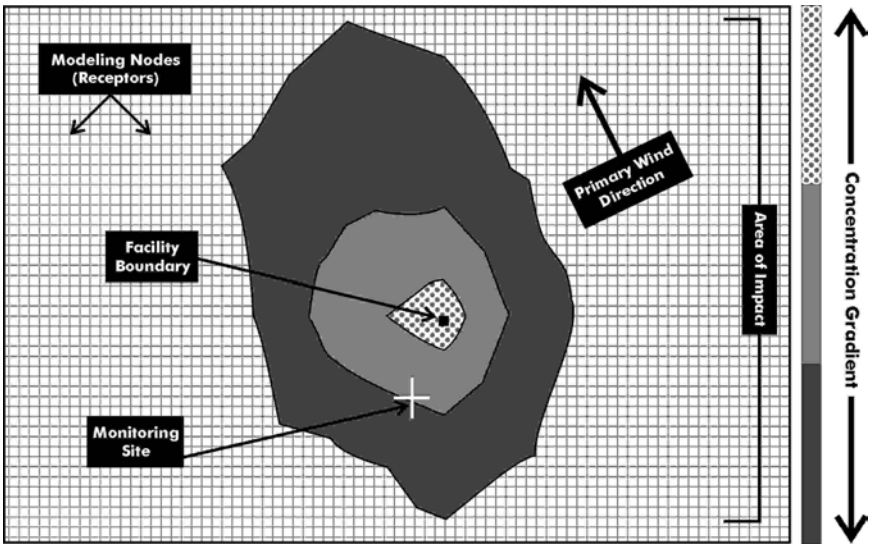


Exhibit 11-2. Illustration of Common Ways to Estimate Exposure Using Ambient Air Concentrations as Surrogates for Exposure Concentration

A



100 meter modeling grid

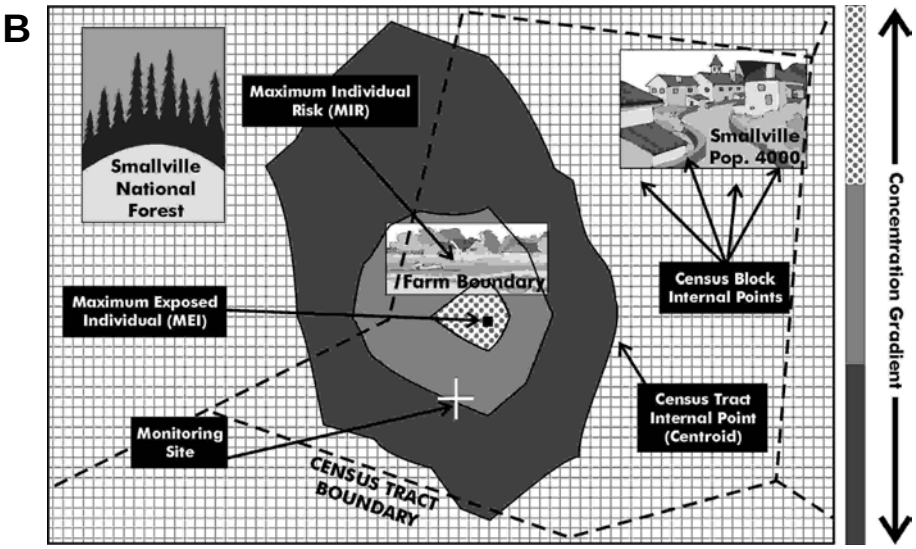
In this hypothetical example, the risk assessors have modeled a release of a volatile organic HAP from a facility using a computerized air quality model, and the ambient air concentration is used as a surrogate for the exposure concentration (EC). The area of impact surrounds the facility and is generally greater in the direction of the primary wind flow (and decreases in concentration with distance from the source). The model was set to make estimates of annual concentration at 100-meter distances from the source in a rectangular grid pattern. The points where the model makes estimates are called "modeling nodes" or "receptors." Note, however, that modeling receptors do not necessarily coincide with actual people (who are also sometimes referred to as receptors) – that is, there may or may not be a person at any given modeling node. There also is one monitoring site.

Knowing only the information displayed in the first version of the map (A), it is difficult to say much about exposure since we do not know where the people are in relation to the facility or the area of impact. To remedy this, our next step is to obtain demographic data (usually from the Census Bureau) and overlay it on the above map. We may also have first-hand knowledge of exactly where people live in the vicinity of the of the facility which we can also include on the map. Performing this analysis and redrawing the map gives picture B (next page).

In the second version of the map (B), we have included the census tract boundaries (dotted lines) and we also know from study area reconnaissance that there is an uninhabited national forest to the west of the facility, a farmer (Mr. MacDonald) directly to the north, and a small town in the northeast. (Note that the town, Smallville, actually can be further subdivided into smaller census blocks; however, they are not shown here to keep the picture simple.) Now that we have a better idea of where people are in relation to the facility (and the area of impact caused by the VOC release), we are in a better position to start making some statements about how people are exposed. Some of the more common ways to characterize the exposures that may be occurring include:

1. **Monitoring Site**. The monitoring site is located in one of the higher parts of the area of impact, but it is southwest of the facility and far from most of the area's populations. This monitoring site would not be appropriate for describing exposure for the people of Smallville, but it could be used for people in the immediate vicinity of the facility and to check the accuracy of the modeling.

Exhibit 11-2 (continued)



2. **Point of Maximum Modeled Concentration**. In this example, this point is located on the facility boundary, where no one currently lives. This point is called the Maximum Exposed Individual or MEI which is defined as the highest estimated risk to a hypothetical exposed individual, regardless of whether people are expected to occupy that area.

3. **Point of Maximum Concentration at a Location Occupied by People**. In this example, this point occurs at Mr. MacDonald's farm. This point is called the Maximum Individual Risk, or MIR, which is defined as the highest estimated risk to an exposed individual in areas that people are believed to occupy. Actually, the concentration used to represent Mr. MacDonald could be described using either an estimate of exposure at a point (e.g., his house) or some other estimate of exposure for the larger farm if there were a good justification for doing so (e.g., an average of all the farm's modeled points, since Mr. MacDonald spends much of his time working around the farm).

4. **Census Tract Internal Point**. In this example, we could simply use the census tract internal point to represent exposure for all people living in the census tract. This is sometimes used, especially when you do not have any first-hand knowledge of the area (i.e., you only have general demographic data from the Census Bureau). However, in this example the census track internal point would not be a very good estimate of exposure concentration because it is higher in concentration than that experienced by most of the population (i.e., the people of Smallville) and it is lower in concentration than that of the highest exposed person (i.e., Mr. MacDonald).

5. **Census Block Internal Points**. So far, this example has focused on characterizing an individual person's exposure living at defined points within the study area (either a real person like Mr. MacDonald, or a hypothetical person like the MIR). What if we wanted to know something more about *how many* people in the study area are living at different levels of exposure? One way to do this is to develop a frequency diagram that displays the exposure concentration at each of the census block internal points and identifies the number of people living in that block (see below). This kind of representation is very helpful to the risk managers because it gives them a sense of the range of exposures and the numbers of people living at different levels of exposure. (In addition, the assessor may also choose to represent the exposure with isopleths of risk (as in the above graphic) and by listing the approximate number people living within each isopleth.)

The internal point with the highest impact in the study area may also be referred to as the point of maximum concentration at a receptor location, although it may not be as precise as the example above where more local knowledge is applied to locate this point.

- **Population-based approaches**. Exposures may be evaluated by tracking individual members of a population and their inhalation through time and space. Such analyses may incorporate a user-specified number of **simulated individuals** or population groups (**cohorts**) to represent the population in the study area. A cohort is defined here as a group of people within a population with the same demographic variables who are assumed to have similar exposures. In this approach, the exposure analysis process consists of relating chemical concentrations in air (outdoor and/or indoor) and tracking the movement of a population cohort through locations where chemical exposure can occur according to a specific activity pattern. Population-based analysis is generally accomplished using exposure models (as described in Section 11.3 below).

- **Personal monitoring**. Exposures may be estimated directly by placing monitors on individuals, which allows collection of more detailed information specific to the exposure pattern for that individual. Such monitors are referred to as **personal monitors** because they provide information on exposure to that individual, rather than to the general area in which an individual might be moving. Personal monitoring is discussed in Section 11.4 below.

Note that the units for the EC estimates are typically expressed in terms of micrograms (or milligrams) of pollutant per cubic meter of air. For pollutants adsorbed to particles, inhalation exposure estimates should be provided as the concentration of these pollutants *on the particles*, not the concentration of the particles themselves.

11.3 Exposure Modeling

This section discusses exposure modeling, which uses the ambient air concentration estimates along with information about the population of interest and information on how the pollutant concentration can vary in different microenvironments to derive estimates of exposure concentration over the period of exposure. Information on human exposure modeling for air toxics can be found on EPA's Fate, Exposure, and Risk Assessment (FERA) website at http://www.epa.gov/ttn/fera/.

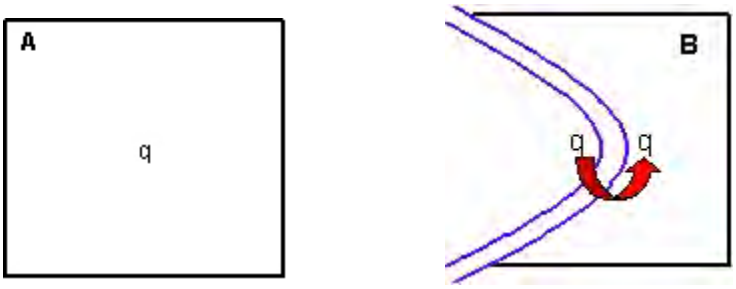
For example, suppose an analyst uses the air quality model, ISCLT3, to estimate the annual average concentration of benzene from a petroleum refinery at each census tract internal point for every census tract within 50 km of the source (for illustration, assume this is 25 census tracts). In a screening level analysis, the analyst may simply use the predicted ambient air concentration as a surrogate for the population chronic exposure concentration of benzene at each of the 25 internal points.

Internal Point or Centroid: Which is Correct?

When evaluating exposure to people in a given place, the modeled air quality at the "internal point" of a geographic entity (such as a census tract or census block) is often used as a starting point to represent exposure for the people in that geographic entity. According to the U.S. Census Bureau:

> *An internal point is a set of geographic coordinates (latitude and longitude) that is located within a specified geographic entity. A single point is identified for each entity; for many entities, this point represents the approximate geographic center of that entity. If the shape of the entity causes this point to be located outside the boundary of the entity or in a water body, it is relocated to land area within the entity. In computer-readable products, internal points are shown to six decimal places; the decimal point is implied. The first character of the latitude or longitude is a plus (+) or a minus (–) sign. A plus sign in the latitude identifies the point as being in the Northern Hemisphere, while a minus sign identifies a location in the Southern Hemisphere. For longitude, a plus sign identifies the point as being in the Eastern Hemisphere, while a minus sign identifies a location in the Western Hemisphere.*

To illustrate how internal points are established, consider the following two examples. In census tract A, the internal point (q) is simply the geographic center of the square. In census tract B, a river flows along the western edge of the tract and makes a sharp bend towards the tract's eastern edge. In this case, the "geographic center" of census tract B is actually outside the tract itself. Since the Census Bureau requires that the internal point be within the physical boundaries of the geographic entity, the Bureau physically moves the point into the tract, as shown (to a point that is no longer the geographic center).



Note that the internal point is generally set to reflect the geographic center of the entity in question, regardless of where people actually live in that entity. In other words, the point is not "population weighted" (the Census Bureau does not provide population weighted internal points for census tracts or block groups). Without population weighting, an exposure concentration estimated at the internal point might not be representative of the concentrations to which persons living in the census entity might be exposed. Analysts routinely modify the Census Bureau internal points for census tracts and census block groups (using census block data) to locate them to a spot more representative of where people are actually located within the geographic entity (e.g., a "population weighted" internal point).

Source: U.S. Department of Commerce, U.S. Census Bureau. 2000. *Geographic Glossary (Census 2000)*. Available at: http://www.census.gov/geo/www/tiger/glossry2.pdf.

However, a limitation of this is that each person in a census tract is not breathing air at the ambient concentration continuously. There are a variety of reasons why this is so. For example:

- People come and go from the census tract for work, play, or travel. They may go to another census tract in the vicinity with either a higher or lower concentration of benzene.

- People do not spend all their time outdoors (which is what our analyst has presumed in our hypothetical example). In fact, most people spend most of their time (with some estimates of about 90 percent) indoors. The chemical concentration of benzene may be higher or lower indoors than outdoors.

- The benzene concentration throughout the census tract, in our example, is probably not always the same as that at the internal point we selected (we have just assumed it was for computational ease).

Exposure modeling was developed to try and help move an analysis into considering these details. Thus, air quality modeling estimates how contaminated the air is in the different locations within a study area. Exposure modeling simulates how different types of people interact differently with that contaminated air to derive integrated (e.g., time weighted) estimates of their exposure for the duration of interest.

This section focuses on exposure models to evaluate inhalation exposures. Exposure models are also available for other routes of exposure as well (e.g., a model may be employed to track patterns of food and drinking water consumption across a population). These indirect pathway exposure models are discussed in Chapter 18.

The estimation of population exposure is a very difficult task because it requires information on the activity patterns of the population as well as information on the air toxics concentrations (indoor and outdoor) to which that population is exposed. Although several databases have been developed to characterize activity patterns (see Section 11.3.3), various sources of variability (e.g., among individuals and geographical regions) introduce uncertainty. Three main factors affect the overall accuracy of exposure modeling:

- Uncertainties associated with indoor air toxics concentrations (note that most people spend the majority of their time indoors);

- How well the subgroups (or cohorts) selected for analysis provide a realistic description of the population composition in a given area; and

- Uncertainty and variability associated with the inputs and parameters of exposure models.

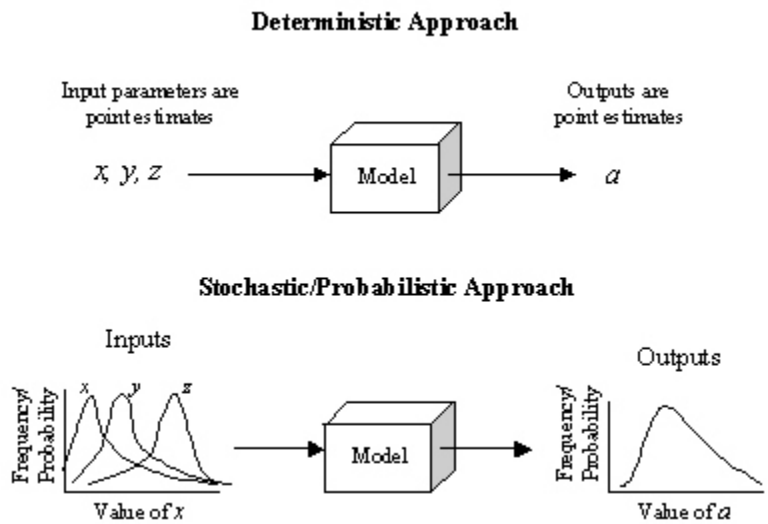
Exposure models can be formulated in a **deterministic** framework, where the value for each input and output variable is characterized by a point estimate (i.e., a single value assumed to apply uniformly). Alternatively, the framework may be **stochastic** or **probabilistic**, with one or more input variables characterized by a frequency or probability distribution[(a)] (see Exhibit 11-3).

---

[a]These terms are introduced and defined in Part VI of this Reference Library.

If the input distributions represent variability[(a)] across the population, the resulting output distribution correspondingly represents the variability of exposures across the population. On the other hand, if the input distributions represent uncertainty[(a)] about input parameters, the output distributions will represent uncertainty about exposure levels. Some of the newer exposure models address both variability and uncertainty separately (see Section 11.3.4).

Exhibit 11-3. Deterministic versus Stochastic/Probabilistic Approaches to Exposure Modeling



In the Deterministic Approach, the assessment assumes that each input to the model is a specific number (and the answer is a number).

In the Stochastic/Probabilistic Approach, the assessment assumes that the inputs to the model may be specified as a distribution (and the answer is a distribution).

11.3.1 Inhalation Exposure Modeling

Inhalation exposure is characterized by the pollutant concentration in the air (i.e., the exposure concentration) reaching an individual's nostrils and/or mouth (in units of $\mu g/m^3$). Estimates of air concentrations from modeling or monitoring can be used in inhalation exposure modeling. When derived from monitoring measurements, exposure concentrations are an **aggregate** of the contributions from all emissions sources impacting the monitor. When derived from modeling studies, the estimated exposure concentrations reflect only the sources that were included in the modeling exercise. Models have an added benefit of allowing the analyst to determine the contribution of a source to the estimated exposure concentration for any of the exposed population groups. (Trying to determine "what source" contributed "how much" to a monitoring result can be a challenging and perhaps impossible task, depending on the chemical and number of sources in the study area).

Lead Exposure Modeling

Lead (Pb) poisoning presents potentially significant risks to the health and welfare of children all over the world today. The Integrated Exposure Uptake Biokinetic Model for Lead in Children (IEUBK) attempts to predict blood-lead concentrations (PbBs) for children exposed to lead in their environment. The model allows the user to input relevant absorption parameters (e.g., the fraction of lead absorbed from water) as well as intake and exposure rates. Using these inputs, the IEUBK model rapidly calculates and recalculates a complex set of equations to estimate the potential concentration of lead in the blood for a hypothetical child or population of children (6 months to 7 years of age). Measured or estimated blood-lead concentration is not only an indication of exposure, but also a widely-used index for discerning future health problems. For additional information see http://www.epa.gov/superfund/programs/lead/ieubk.htm.

Because air pollutant concentrations vary over time and space, inhalation exposure models combine information on human activity patterns and microenvironmental concentrations to estimate exposure concentrations. **Activity patterns** are defined by an individual's or cohort's allocation of time spent in different activities in various microenvironments and various geographic locations. A **microenvironment** is a defined space that can be treated as a well-characterized, relatively homogeneous location with respect to pollutant concentration for a specified time period (e.g., rooms in homes, restaurants, schools, offices; inside vehicles; outdoors).

A common exposure model for inhalation that combines information on microenvironment concentrations and activity patterns calculates a **time-weighted average of all exposures** from the different microenvironments in which a person spends time during the period of interest:

$$EC_A = \frac{1}{T}\left(\sum_j C_j \times t_j\right) \qquad \text{(Equation 11-1)}$$

where:

EC_A = the adjusted average inhalation exposure concentration (µg/m^3),
T = total averaging time ($T = \sum t_j$; years),
C_j = the average concentration for microenvironment j (µg/m^3), and
t_j = time spent in the microenvironment j (years).

Note that the two critical parameters that need to be evaluated in this equation are the concentration of a chemical in a microenvironment and the amount of time spent in that microenvironment. Exhibit 11-4 presents a simple example. General information on how assessors go about obtaining such data is provided below. As a practical matter, most air toxics risk assessments will not actually gather such activity pattern data for study-specific exposure assessments. Rather, available exposure models have already incorporated much of this information for use by the general risk assessment community. However, every model is different and the data input requirements vary from model to model. Usually, assessors carefully review each model's documentation before deciding to use it to determine if it will answer the

question that needs to be answered and what resources would be needed to develop the required inputs.

Exhibit 11-4. Simple Example of How to Estimate Exposure Concentration (EC) for Exposure Modeling

EC. The following exposure profile has been developed for one year (which represents, for example, the 30 years of "work") for a representative individual within the population of interest:

Duration Spent in Each Microenvironment (% year)	Average Concentration of Pollutant A in Each Microenvironment (µg/m^3)
10 = outside	80
50 = at work	20
40 = inside house	10

The EC for that individual is calculated as:

$$EC = (0.1 \times 80) + (0.5 \times 20) + (0.4 \times 10) = 22\ \mu g/m^3$$

Lifetime EC. To derive a lifetime exposure concentration for that individual, annual estimates are combined as follows:

Duration Exposed to Each Annual Concentration (no. years)	Annual Average Concentration of Pollutant A (µg/m^3)
1 = newborn	10
4 = pre-school	40
12 = school	30
4 = college	30
30 = work	22
19 = retirement	40

The Lifetime EC is calculated as:

$$\text{Lifetime EC} = \frac{(1 \times 10) + (4 \times 40) + (12 \times 30) + (4 \times 30) + (30 \times 22) + (19 \times 40)}{70} = 30\ \mu g/m^3$$

Screening exposure estimate. One way to perform a screening level assessment using these data is to set the EC equal to the highest air concentration modeled (e.g., 80 µg/m^3 for annual adjusted or 40 µg/m^3 for lifetime adjusted – see examples above) for all microenvironments. If the hazard and risk, respectively, prove to be below acceptable risk values, the risk manager may conclude that no further evaluation is necessary.

11.3.2 Microenvironment Concentration: How is it Developed?

Microenvironments can be indoors (e.g., school, office, car, bus) or outdoors (e.g., filling station, roadway). Indoor microenvironment concentrations are comprised of contributions from a chemical in outdoor air penetrating the indoor environment and from indoor emission sources of that same chemical (if indoor sources are within the scope of the analysis). They may be derived from direct measurements or estimated from modeling.

There are two common approaches to modeling indoor microenvironment concentrations. One is the **microenvironment factors method**, where the outdoor contribution is estimated from the outdoor concentration and a microenvironment factor that represents the ratio of the microenvironment concentration to the outdoor concentration. Microenvironment factors are typically derived from concurrent measurements of concentrations in the microenvironment (containing no indoor emission sources) and outdoors. The indoor contribution is then added to estimate the overall microenvironment concentration (when indoor sources are included in the scope of the assessment). A general equation for the microenvironment factors method is:

$$C_j = M_j C_o + C_s \qquad \text{(Equation 11-2)}$$

where:

- C_j = concentration in microenvironment *j*
- M_j = microenvironment factor for microenvironment *j*
- C_o = concurrent outdoor concentration
- C_s = concentration contribution to the microenvironment *j* concentration from an indoor emission source

The second approach is the **mass-balance method**. The mass balance method typically assumes that an enclosed microenvironment is a single well-mixed "box," although multi-chamber configurations are possible. The time-varying concentration of an air pollutant in such a microenvironment is estimated from several variables (see Exhibit 11-5). A general formulation for the change in concentration in an enclosed microenvironment over time is:

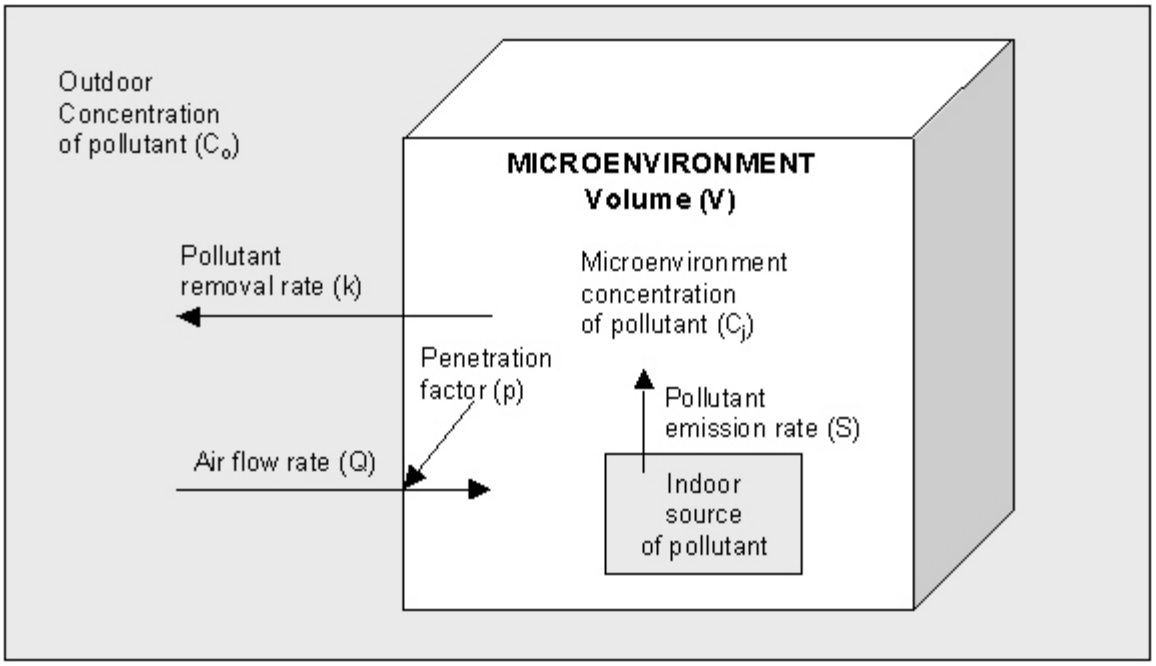
$$V \frac{d}{dt} C_j = pQC_o + S - kC_j - QC_j \qquad \text{(Equation 11-3)}$$

where:

- V = volume of microenvironment enclosure
- C_j = concentration in microenvironment *j*
- p = penetration factor (only applies to incoming air)
- Q = air flow rate
- k = pollutant removal rate (includes all types of removal, including atmospheric decay, surface reactivity, surface adsorption, wall deposition, etc.)
- C_o = concurrent outdoor concentration
- S = indoor source emission rate

The solution to this differential equation can be used to predict a time sequence of microenvironment *j* concentrations.

Exhibit 11-5. Illustration of Mass Balance Method for Modeling Indoor Microenvironments



11.3.3 Sources of Data for Human Activity for Inhalation (and other) Exposure Assessments

Numerous EPA and related databases provide information useful for conducting exposure assessments, including information on activity pattern and demographic information useful for inhalation exposure modeling. Types of information included are human activity surveys, standard values for physiological processes and consumption of food and water, measured exposure data, health status surveys and measurements, nutrition surveys, and data on the spatial distribution of populations. This section provides several of the more notable information sources, some of which are important for inhalation exposure modeling, and some of which are important for modeling exposures through pathways other than inhalation (e.g., ingestion of contaminated fish, soil, and groundwater). Because they are so important for an understanding of exposure, we introduce them here (even though the focus of this Chapter is on inhalation). We will revisit many of these sources in Part III (Multipathway Exposure Assessment).

Indoor vs. Outdoor Concentrations

Indoor air concentrations may be an important consideration in an air toxics risk assessment. Depending on the pollutant and the sources being assessed, concentration levels may be substantially higher outdoors, in one or more indoor microenvironments, or inside vehicles. In general, pollutants that have important indoor emission sources will have higher concentrations indoors than outdoors. Important indoor emission sources include combustion sources, building materials, consumer products, and occupant activities like cigarette smoking. Similarly, pollutants that are primarily emitted by motor vehicles would be expected to have higher in-vehicle concentrations than at outdoor locations distant from roadways.

Information that may be useful to the various methods used to estimate microenvironment concentrations is available from studies involving measurements of indoor and personal exposure concentrations. These include the following EPA studies:

- The Building Assessment, Survey and Evaluation (BASE) study, which was a cross-sectional study of 100 buildings. Information relating to BASE is currently being updated to include basic summary results from the 100 buildings studied. The raw data collected for the 100 buildings is scheduled for release soon.[1]

- The Longitudinal Temporal Indoor Monitoring and Evaluation (TIME) Study in federal buildings.[1]

- The Los Angeles Total Exposure Assessment Methodology (TEAM) study,[2] which collected concurrent indoor and outdoor samples of 18 VOCs for two consecutive 12-hour periods in 1987, around 45 homes in February and 40 homes in July.

- **EPA Consolidated Human Activity Database (CHAD)**. CHAD contains data obtained from human activity studies that were performed at city, state, and national levels. CHAD is intended to provide input data for exposure/intake dose modeling and/or statistical analysis.[3] CHAD is a master database providing access to other human activity databases using a consistent format. This facilitates access and retrieval of activity and questionnaire information from those databases.

  The studies contained in CHAD cover a range of geographic areas. In addition to the National Human Activity Pattern Study (NHAPS) with information about residents from 48 states, there are studies targeting residents of Baltimore, Cincinnati, Denver, Los Angeles, Valdez, Washington DC, and the states of California and Michigan. Because the individual studies differed based on what information was collected, not all fields in the CHAD database are populated for all the records.

  Each CHAD diary record consists of a 24-hour sequence of activities. Specified for each activity is a start time, end time, duration, one of 113 location codes, and one of 145 activity codes. Each diary record is tagged with a CHAD ID, which relates it to a record in the demographic database identifying information about the subject of the diary. Demographic fields include personal characteristics (age, gender, ethnicity, weight), social characteristics (education, occupation, income), residential location (state, county, zipcode) and housing characteristics (heating fuel, cooking fuel). In addition, CHAD has the capability to estimate

the relative metabolic rate for each activity in a record using random sampling from distributions derived from clinical studies.

- **EPA Exposure Factors Handbook**. The Exposure Factors Handbook provides a statistical summary of the available data on various parameters and variables used in assessing human exposure. This Handbook is used by risk assessors who need to obtain data on standard factors to calculate human exposure to toxic chemicals. These factors include human activity factors and residential characteristics. Recommended values are for the general population and also for various segments of the population who may have characteristics different from the general population. Included are full discussions of the issues that assessors may want to consider in deciding how to use these data and exposure parameter recommendations. (The Exposure Factors Handbook is in final form, but as new data become available updates will be posted).[4]

- **EPA Human Exposure Database System (HEDS)**. HEDS is a web-enabled data repository for human exposure studies.[5] Its mission is to provide data sets, documents, and metadata for human exposure studies that can be easily accessed and understood by a diverse set of users. HEDS provides only data and accompanying documentation from research studies; it does not provide interpretations. It allows a user to download documents for review or data sets for analysis on their own computer system. Currently contained in HEDS are various components of the National Human Exposure Assessment Survey (NHEXAS).

- **National Human Exposure Assessment Survey (NHEXAS)**. The National Human Exposure Assessment Survey was developed by US EPA's Office of Research and Development (ORD) in the 1990's to provide information about multimedia and multipathway population exposure to chemicals of various types. Phase I consists of demonstration/scoping studies using probability-based sampling designs. Volunteer participants were randomly selected from several areas of the U.S. These studies included personal exposure, residential concentrations, and biomarker measurements. The Arizona study measured metals, pesticides, and VOCs. The Maryland study measured metals, pesticides, and polycyclic aromatic hydrocarbons (PAHs). The Region 5 study, conducted in Ohio, Michigan, Illinois, Indiana, Wisconsin, and Minnesota, measured metals and VOCs. Researchers worked with the participants to measure the level of chemicals in the air they breathed, in the foods and beverages they consumed (including drinking water), in the soil and dust around their homes, and in their blood and urine. Participants completed questionnaires to help identify possible sources of chemical exposure. Sample collection occurred between 1995 and 1997. The confidentiality of participants is strictly protected. Information about the studies can be found in the related study entries in EIMS and in the *Journal of Exposure Analysis and Environmental Epidemiology*.[6]

- **CDC National Health and Nutrition Examination Survey (NHANES)**. NHANES is a survey conducted by the National Center for Health Statistics (NCHS), Centers for Disease Control and Prevention.[7] This survey has been designed to collect information about the health and diet of people in the United States. NHANES is unique in that it combines a home interview with health tests that are done in a Mobile Examination Center. The current NHANES is eighth in a series of national examination studies conducted since 1960. The results of these surveys are compiled in databases and summarized in a variety of tables and reports. Data from direct examination, testing, and measurement of national samples of the

civilian noninstitutionalized population provide the basis for (1) estimates of medically-defined prevalence in the US and the distribution of the population with respect to physical, physiological, and psychological characteristics, and (2) analysis of relationships among various measurements without reference to an explicit finite universe of persons. Reports also present information about dietary patterns in various segments of the US population.

- **U.S. Census Data**. The U.S. Census provides data on the spatial distribution of population and population subgroups at several geographic levels: national, state, county, tract, block group and block. (For detailed analysis, Summary File 3 is most useful.) Examples of useful spatially-resolved data for exposure assessment include: population by age, gender, and ethnic group; house heating fuel use; estimated travel time to work by various modes of transportation; and levels of employment in various industries. Associated geographic data specifying boundaries of the various geographic entities for mapping are also available in Topologically Integrated Geographic Encoding and Referencing (TIGER) files.[8]

- **LandScan USA**. LandScan is a high resolution population distribution database for the continental U.S. currently under development, following the methodology used to create a similar global database called LandScan1998 (updated in 2000).[9] LandScan uses satellite imagery in population distribution modeling to produce population distribution data at a much finer resolution than previously available. LandScan 1998 and 2000 have a grid cell size of 30 seconds (<1 kilometer) and use census data in combination with many other geospatial data, such as land use/cover, topography, slope, roads, and nighttime lights, in order to improve the estimation and prediction of the spatial distribution of residential populations. Future LandScan updates will use a much smaller grid cell size of 3 seconds (<100 meters). Currently, a pilot study in a 29 county area in southeast Texas (around Houston and Port Neches) is being conducted. LandScan will be very useful for exposure modeling, environmental justice studies, and other types of risk assessments.

11.3.4 Examples of Inhalation Exposure Models

Several exposure models have been or are being developed by EPA and others for a variety of purposes. Some of the important characteristics that vary among the models include:

- Ambient concentrations
  - Modeling or monitoring estimates
  - Time scales (e.g., averaging time)

- Exposure concentration time scale
  - Time increment for calculations (e.g., by minute, hourly, seasonally, annually)
  - Averaging time for reporting (e.g., hourly, annually)

- Spatial scale
  - Geographic resolution of predictions (e.g., Census tracts, Census blocks, grids)
  - Potential size of modeling domain (e.g., neighborhood, county, nation)

- Population activity data
  - Type (e.g., time in microenvironments, commuting locations, food and water ingestion rates)

- Temporal resolution (e.g., by minute, hourly, seasonally, annually)
- Area specific resolution (e.g., national or regional)
- Demographic resolution (e.g., by age, gender, or ethnic group)

- Framework
  - Deterministic: inputs and outputs are characterized as point estimates
  - Stochastic or probabilistic: inputs and outputs are characterized as distributions representing variability and/or uncertainty; Monte Carlo techniques are used to randomly select input values from the distributions for repeated simulations

The remainder of this section provides brief descriptions of some of the most recently developed inhalation exposure models. The features of each model described are summarized in Exhibit 11-6.

Exhibit 11-6. Comparison of Inhalation Exposure Model Features

Model	Population Activity Data	Source of Ambient Concentrations	Spatial Resolution	Framework
HEM-3	none (screening model)	ISCST3	census blocks (additional points can be specified)	deterministic
HAPEM	micro-environment time/sequence, commuting	external model or monitoring data	census tract	stochastic
TRIM.Expo (a.k.a. APEX)	micro-environment time/sequence, commuting	external model or monitoring data	depends on resolution of air quality and demographic inputs	stochastic
CPIEM	micro-environment time/sequence, commuting	external model or monitoring data	user-specified for the selection of activity patterns (e.g., state, region)	stochastic

Human Exposure Model (HEM)

The Human Exposure Model (http://www.epa.gov/ttn/fera/human_hem.html) was designed to screen major stationary sources of air pollutant emissions efficiently, ranking the sources according to the potential cancer risks and noncancer hazard associated with long-term (annual) average exposure concentrations.[10] The current version, Version 3 (HEM-3), is implemented on a Windows platform for ease of use. HEM-3 contains a version of the Gaussian atmospheric dispersion model ISCLT2 (with included meteorological data), and U.S. Census Bureau population data (2000) at the Census block level. A limited amount of source data are required as model inputs (e.g., pollutant emission rates, facility location, height of the emission release, stack gas exit velocity, stack diameter, temperature of the off-gases, pollutant properties, and source location). HEM-3 estimates the magnitude and distribution of ambient air concentrations of pollutant in the vicinity of each source. The model usually estimates these concentrations

within a radial distance of 50 kilometers (30.8 miles) from the source. Exposure concentrations for the residents of each Census block are assumed to be the outdoor concentration at the Census block "internal point." This actually represents a surrogate for exposure, as important exposure variables (e.g., indoor-outdoor concentration differences, human mobility patterns, residential occupancy period, breathing rates) are not explicitly addressed. Multiple facilities (including clusters of facilities, each having multiple emission points) can be addressed by HEM-3. Variability and uncertainty in input data and parameters are not considered.

The Hazardous Air Pollutant Exposure Model (HAPEM5)

The latest version of EPA's Hazardous Air Pollutant Exposure Model (HAPEM5) is a stochastic screening-level inhalation exposure model appropriate for assessing average long-term (annual) exposures of the general population, or a specific sub-population, over spatial scales ranging from urban to national (http://www.epa.gov/ttn/fera/human_hapem.html). This application requires a moderate level of computer modeling skills.

HAPEM5 uses the general approach of tracking representatives of specified demographic groups as they move among 37 indoor, in-vehicle, and outdoor microenvironments and among geographic locations. The estimated pollutant concentrations in each microenvironment visited are combined into a time-weighted average concentration, which is assigned to members of the demographic group (the cohorts). Microenvironment concentrations are estimated from outdoor concentrations with the factors method. HAPEM5 uses five primary sources of information: population data from the U.S. Census; population activity data from CHAD commuting data developed by the Bureau of the Census; user supplied air quality data either from measurements or an air dispersion model; and microenvironmental factors data.

The previous version of HAPEM5, namely HAPEM4, was used in the NATA national scale assessment of the 1996 NEI to develop estimates of risk, by census tract, for each of the 33 HAPs (http://www.epa.gov/ttn/amtic/netamap.html). Specifically, HAPEM4 was used to predict population exposure for each of 10 demographic groups in each tract.

Total Risk Integrated Methodology Exposure Event Model (TRIM.Expo$_{Inhalation}$), also known as Air Pollutants Exposure Model (APEX)

The Air Pollutants Exposure Model (APEX) comprises the inhalation portion of the TRIM exposure module, TRIM.Expo (http://www.epa.gov/ttn/fera/human_apex.html).[(b)]

TRIM.Expo (a.k.a. APEX) uses a personal profile approach rather than a cohort simulation approach. That is, individuals are selected for simulation by selecting combinations of demographic characteristics and finding an activity pattern to match it, rather than directly selecting an activity pattern. If the selection probabilities for the demographic characteristics are the same as within the population to be simulated, this approach will provide a representative sample of that population's activity patterns without the need for post-simulation weighting of results.

---

[b]EPA has developed the Total Risk Integrated Methodology (TRIM) for use in the assessment of air pollutants (both hazardous and criteria). APEX comprises the inhalation exposure component of TRIM.

The current version (APEX3, available on the web) includes a number of useful features including automatic site selection from large (e.g., national) databases, a series of new output tables providing summary statistics, and a thoroughly reorganized method of describing microenvironments and their parameters. The model has the capability to estimate microenvironment concentration from the mass-balance method, but also provides the option of using the factors method. Most of the spatial and temporal constraints were removed or relaxed in APEX3. The model's spatial resolution is flexible enough to allow for the use of finely resolved modeled air quality values, as well as sparser measured values. Averaging times for exposure concentrations are equally flexible. Like HAPEM5, the user must supply the air quality data (from modeling or monitoring) to the model.

California Population Indoor Exposure Model (CPIEM)

The CPIEM[11] is a stochastic inhalation exposure model developed for the California Air Resources Board's (ARB's) Indoor Program to evaluate indoor exposures for the general California population as well as certain sub-populations. CPIEM combines indoor air concentration distributions with Californians' location and activity information to produce exposure and dose distributions for different types of indoor environments.

The temporal resolution and averaging time are user-selected from the options of 1-hour, 8-hour, 12-hour, and 24-hour. The spatial resolution and modeling domain similarly are specified by the user according to county, state region, or the entire state. Although outdoor concentrations may be included in the application, the focus is on indoor exposures and indoor emission sources. The model is implemented on a Windows-based platform for ease of use.

The model uses location/activity profiles that were collected in ARB studies. Microenvironment concentrations are derived from measurement studies for up to nine microenvironments. Concentration distributions from measurement studies for many pollutants and microenvironments are included in the CPIEM database. However, for pollutants and microenvironments not included in the database, the CPIEM presents two alternatives. The first is to estimate indoor air concentration distributions based on distributional information for mass balance parameters with a mass-balance module. The second is for the user to directly specify concentration distributions.

11.3.5 Exposure Modeling Examples

The following applications of air quality modeling and exposure modeling at real-world sites provide useful insights into air toxics modeling. The TRIM.Expo (a.k.a. APEX) inhalation exposure model has also been used with the ISCST3 air quality model to predict human inhalation exposures. A report documenting this aspect of the case study will be available at: http://www.epa.gov/ttn/fera/human_apex.html.

National-scale Air Toxics Assessment (NATA). EPA's NATA is designed to provide a comprehensive evaluation of air toxics exposure and risk across the U.S. Activities include expansion of air toxics monitoring, improving and periodically updating emission inventories, improving national- and local-scale modeling, continued research on health effects and exposures to both ambient and indoor air, and improvement of assessment tools. As noted previously, one component of NATA is a National Scale Assessment conducted with the ASPEN and the

HAPEM4 to estimate annual average exposure concentrations of the 33 urban air toxic pollutants in every US Census tract. Specific examples of the results of the National Scale Assessment and additional information on NATA activities can be found on-line.[12]

Houston Case Study. This study was carried out by EPA's Office of Air Quality Planning & Standards (OAQPS) and the Office of Transportation and Air Quality (OTAQ) as a component of the Integrated Urban Strategy.[13] For the Houston metropolitan area, ISCST3 modeling was applied, using emissions data for point, non-point, and mobile sources from EPA's 1996 National Toxics Inventory. Ambient air concentrations for numerous air toxics were predicted at the census tract level with ISCST3 and HAPEM, which were then employed to obtain estimates of population exposures. Modeling results were compared to the results obtained through studies of this area carried out as part of the NATA National Scale Assessment. The study demonstrated that modeling using ISCST3 and an improved emissions inventory provides more realistic patterns and better agreement with monitoring data. In addition, elevated concentrations (hot spots) were found that were not detected in the national scale analysis.

11.4 Personal Monitoring

Thus far, we have focused on monitoring devices that generally are located in a secure compound (and sometimes on roof tops) that measure air quality that is representative of some specific geographic scale. An alternative to such an approach is to place monitors directly on individuals, which allows collection of more detailed information specific to the exposure pattern for that individual. Such monitors are referred to as **personal monitors** because they provide information on exposure to that individual, rather than to the general area in which an individual might be moving. An advantage is that personal monitors reflect the time-varying concentrations (unless they are integrating monitors) an individual experiences as he or she moves about through various activities. Personal monitors have seen increasing use in recent years due to two factors: they are more readily available, reliable, and cheaper than in the past, and there is growing evidence that personal exposures may at times be correlated poorly with average values derived for larger geographic areas (see Exhibit 11-7).

Two modes of personal monitoring have been developed. One relies on direct measurements of air concentration for toxics in the breathing zone or otherwise on/near the body of an individual (these are called direct measurement methods). The other relies on changes in biological properties such as blood level of an air toxic (or metabolite). The latter is not considered here because it does not strictly measure ambient air concentrations or estimate exposure. Personal monitors, as with area or fixed monitors described previously in this chapter, are available in two types:

- **Active monitors** use a small air pump to draw air through a filter, packed tube, or similar device. They can be both continuous and integrated. Such a personal exposure monitor is available to measure PM_{10} and $PM_{2.5}$ in air using a 37 mm Teflon filter and a 4 L/min flow rate. The pump and battery pack are worn in a bag, while the filter can be located essentially anywhere on the body. In addition, cyclone personal samplers are available for measuring particulates in air (the term "cyclone" refers to the fact that the sampler measures the particulates by "spinning" the particles in an air stream, which then collect on the sides of the device for collection and analysis). Combinations of impactor and denuder filter packs are

available to sample both aerosols and gases such as SO_2, NH_3, and HNO_3. Different coating materials on the diffuser tube can be used to collect different gases.

Exhibit 11-7. Examples of the Use of Personal Monitoring
• **Relationship of Indoor, Outdoor and Personal Air (RIOPA) study**.[14] Indoor and outdoor concentrations of 30 polycyclic aromatic hydrocarbons (PAHs) were measured in 55 homes in Los Angeles, CA, Houston, TX, and Elizabeth, NJ. The study focused on areas in each city characterized by worst-case conditions in the outdoor air, generally located close to major sources. Integrating MSP samplers, polyurethane foam cartridges, and quartz fiber filters were used for the field sampling, and the samples were analyzed subsequently in the lab. Among many results, the study showed that indoor air was dominated by outdoor sources for these compounds, with reasonably strong correlations between the indoor and outdoor air concentrations.
• **National Human Exposure Assessment Survey (NHEXAS)**.[15] The NHEXAS program was designed to "describe the distribution of human exposure to multiple chemicals from multiple routes on a community and regional scale, and its association with environmental concentrations and personal activities." It is being conducted in three stages: (1) design, field evaluation and demonstration projects; (2) exposure field studies; and (3) special studies to examine issues such as highly exposed populations and long-term exposures. Extensive statistical analyses of the data have been performed, including characterizations of background levels of exposure to selected chemicals, as well as correlations among environmental concentrations, individual exposures, biomarkers, and survey data on personal activities.
• **EPA's Total Exposure Assessment Methodology (TEAM)** studies[16] estimated exposures of about 800 persons to 25 VOCs; about 300 persons to 32 pesticides; and 1,200 persons to carbon monoxide. The general approach in all four of the main TEAM studies was the same: a probability-based selection of respondents, so that they would represent a much larger population (e.g., the 800 persons in the TEAM VOC studies actually represented about 800,000 persons in 8 cities); the use of personal monitors as well as outdoor monitors to estimate actual personal exposure; and the use of an Office of Management and Budget (OMB)-approved questionnaire and activity diary to try to pinpoint local sources. In two of the TEAM Studies for VOCs and carbon monoxide, an effort was made to measure body burden, by collecting a breath sample from each of the 2,000 persons involved. This was important in identifying active smoking as the main source of exposure to benzene and styrene, for example. Also, the breath measurements identified a "dirty dozen" pollutants that were prevalent in almost every person. The Centers for Disease Control later collected blood samples from 800 different persons and found essentially the same dozen pollutants prevalent in blood.

- **Passive monitors** rely on sorption, entrapment, etc., driven largely by diffusion. They are primarily integrated sampling devices, giving a estimate of average exposure over the sampling period. Examples include diffusion tubes, badges, and detector tubes. Diffusion badges currently are available for measurement of NO_2, O_3, SO_2, CO and formaldehyde. Organic vapors can be measured in passive devices using activated charcoal badges, although the range of compounds, aside from organics, that can be sampled in this way is small.

Reviews of such methods of personal sampling can be found in Bower et al. (1997).[17] However, many of the same limitations as ambient methods exist, and in some cases additional quantitation limit and precision problems are present.

In general, air toxics risk assessments that rely on monitoring to characterize exposure will generally not rely on personal monitoring because of the highly complex and resource intensive nature of this technique, and because personal monitoring and its findings are currently more geared toward basic research.

11.5 Exposure to a Population: Common Descriptors

There are a wide variety of ways to describe exposure to a population, some of which may be legally required, others which may be chosen based on the requirements of the risk manager. No matter what specific measure is chosen, the risk assessment needs a clear and scientifically supportable rationale for the approach taken; risk assessors generally describe that approach clearly and thoroughly in the exposure assessment portion of the risk assessment documentation. Risk assessors aim for there to be no ambiguity about what was done in the exposure assessment.

EPA policy and guidance recommend that exposure to a population be described using several different ways to give the risk manager a sense of the range and magnitude of the exposures. For example, a "high end" exposure estimate might describe the exposure experienced by actual people in the most highly concentrated part of the area of impact, while a "central tendency" exposure estimate might describe the exposure experienced by people in the study area who experience more modest concentrations.

A variety of statistical values are used to describe high-end and central tendency exposures, including 95th percentile exposures (for high-end) and 50th percentile values for central tendency. Risk assessors will want to obtain and become familiar with EPA's *Risk Characterization Handbook* to better understand various ways exposure and risk can be adequately characterized.[18] EPA's *Guidelines for Exposure Assessment*[19] is also invaluable in this regard. Some of the alternative approaches for characterizing air toxics exposures are illustrated in Exhibit 11-2 above.

11.6 Evaluating Uncertainty

Uncertainty includes the assumptions and unknown factors inherent in the exposure assessment. Discussing uncertainty places the risk estimates in proper perspective. Specific uncertainties associated with the chemical monitoring data, fate and transport models, and the input data (especially emissions inventory data) that assessors use to estimate exposure concentrations usually account for the bulk of uncertainty within the assessment. Exposure models also contribute to the overall uncertainty in exposure assessment. The assessor needs to understand the extent to which variability and uncertainty are considered in all the fate and transport and exposure models that are used. HAPEM and other exposure models can accept input data on the distributions of time spent in different micro-environments and produce time-average exposure estimates for defined populations.

The assessor should be familiar with the extent to which the various components of the exposure assessment can and do accommodate uncertainty and variability analyses. In addition, it is important to consider the compatibility of models in the various steps in the exposure assessment (emissions, transport, etc.) with regard to addressing important sources of uncertainty. Once the capabilities and data requirements of the various models are known, the assessor should consider

the appropriate level of detail for addressing uncertainty in specific variables, and approaches for integrating uncertainty analyses across the models.

11.7 Presenting the Results of an Exposure Assessment

The summary of exposure assessment for air toxics consists of presenting the ECs for each chemical of potential concern (COPC) with the duration of exposure for the populations of interest, as well as characterizing salient features of the study population(s), particularly those that may be influencing their exposure and resultant risk (e.g., size and proximity to sources and /or locations of highest ambient concentrations). The assumptions used to develop these estimates should also be presented and discussed. In addition to the summary tables, it is useful to show sample calculations for each pathway to aid in the review of the calculations. (If exposure modeling is used, a thorough discussion with sample calculations is usually also provided.)

References

1. U.S. Environmental Protection Agency. 2003. Building Assessment, Survey and Evaluation Study (BASE) Study, updated June 17, 2003, and Longitudinal Temporal Indoor Monitoring and Evaluation (TIME) study in federal buildings. Available at: http://www.epa.gov/iaq/largebldgs/base_page.htm. (Last accessed March 2004).

2. Wallace L., W. Nelson, R. Ziengenfus, E. Pellizzari, L. Michael, R. Whitmore, H. Zelon, T. Hartwell, R. Perritt and D. Westerdahl. 1991. The Los Angeles TEAM Study: Personal Exposures, Indoor-Outdoor Air Concentrations, and Breath Concentrations of 25 Volatile Organic Compounds. *J. Expos. Anal. Environ. Epidem.*, 1(2):157-192.

3. U.S. Environmental Protection Agency. 2003. Consolidated Human Activity Database (CHAD). Updated November 20, 2003. Available at: http://www.epa.gov/chadnet1/. (Last accessed March 2004).

4. U.S. Environmental Protection Agency. 2002. The Exposure Factors Handbook. Updated December 30, 2002. Available at: http://cfpub.epa.gov/ncea/cfm/exposfac.cfm?ActType=default. (Last accessed March 2004).

5. U.S. Environmental Protection Agency. 2003. Human Exposure Database System (HEDS). Updated February 11, 2003. Available at: http://www.epa.gov/heds/. (Last accessed March 2004).

6. *Journal of Exposure Analysis and Environmental Epidemiology*, Vol. 5, No. 3, 1995.

7. Centers for Disease Control. 2003. National Health and Nutrition Examination Survey. Updated October 30, 2003. Available at http://www.cdc.gov/nchs/nhanes.htm. (Last accessed March 2004).

8. U.S. Census Bureau. 2004. *Index of /census_2000/datasets/Summary_File_3*. Available at: http://www2.census.gov/census_2000/datasets/Summary_File_3/ (Last accessed March 2004).

U.S. Census Bureau. Topologically Integrated Geographic Encoding and Referencing system (TIGER). Updated March 5, 2004. Available at: http://www.census.gov/geo/www/tiger/index.html. (Last accessed March 2004).

9. U.S. Environmental Protection Agency. 2003. Port Neches Environmental Justice Study and Demonstration Project for the Development of Landscan USA. Updated December 30, 2003. Available at: http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=22406. (Last accessed March 2004).

10. U.S. Environmental Protection Agency. 2003. Human Exposure Modeling - Human Exposure Model (HEM). Updated April 23, 2003. Available at: http://www.epa.gov/ttn/fera/human_hem.html. (Last accessed March 2004).

11. ICF Consulting. 2002. *California Population Indoor Exposure Model, Version 2: User's Guide*. Prepared for the California Environmental Protection Agency, California Air Resources Board.

12. U.S. Environmental Protection Agency. 2002. The National-Scale Air Toxics Assessment. Updated September 18, 2002. Available at: http://www.epa.gov/ttn/atw/nata/. (Last accessed March 2004).

13. U.S. Environmental Protection Agency. 2004. Technology Transfer Network. Air Toxics Strategy: Overview. Updated February 10, 2004. Available at: http://www.epa.gov/ttn/atw/urban/urbanpg.html. (Last accessed March 2004).

14. Naumova, Y., Eisenreich, S., Turpin, B., Weisel, C., Morandi, M., Colome, S., Totten, L., Stock, T., Winer, A., Alimokhtari, S., Kwon, J., Shendell, D., Jones, J., Maberti, S. and Wall, S. 2002. Polycyclic Aromatic Hydrocarbons in the Indoor and Outdoor Air of Three Cities in the U.S. *Environmental Science and Technology*, 36: 2552-2559.

15. U.S. Environmental Protection Agency. 2003. Human Exposure Measurements: National Human Exposure Assessment Survey (NHEXAS). Updated July 21, 2003. Available at: http://www.epa.gov/heasd/edrb/nhexas.htm. (Last accessed March 2004).

16. Ozkaynak H., J. Xue, R. Weker, D. Butler and J. Spengler. 1995. *The Particle TEAM (PTEAM) Study: Analysis of the Data, Volume III, Draft Final Report*. U.S. Environmental Protection Agency, Research Triangle Park, NC.

    Ozkaynak H., J. Xue, J. Spengler, L. Wallace, E. Pellizzari, and P. Jenkins. 1996. Personal exposure to airborne particles and metals: results from the Particle TEAM Study in Riverside, *California. Journal of Exposure Analysis and Environmental Epidemiology*, 6(1):57-78.

    Pellizzari, E. D., K. W. Thomas, C. A. Clayton, et al. 1992. *Particle Total Exposure Assessment Methodology (PTEAM): Riverside, CA Pilot Study Vol. I.* U.S. Environmental Protection Agency, Research Triangle Park, NC. (EPA 600/R-93-050).

Sheldon, L., A. Clayton, J. Keever, et al. 1992. PTEAM: *Monitoring of Phthalates and PAHs in Indoor and Outdoor Air Samples in Riverside, California. Final Report*. California Air Resources Board. Sacramento, California.

Wallace, L. 1987. *The Total Exposure Assessment Methodology (TEAM) Study: Summary and Analysis: Volume 1*. U.S. Environmental Protection Agency, Office of Research and Development, Washington, D.C. EPA/600/6-87/002a.

Wallace, L. 1989. Major sources of benzene exposure. *Environ. Health. Perspect*., 82:165-169.

Wallace L., W. Nelson, R. Ziengenfus, E. Pellizzari, L. Michael, R.Whitmore, H. Zelon, T. Hartwell, R. Perritt and D. Westerdahl. 1991. The Los Angeles TEAM Study: Personal Exposures, Indoor-Outdoor Air Concentrations, and Breath Concentrations of 25 Volatile Organic Compounds. *J. Expos. Anal. Environ. Epidem*., 1(2):157-19.

17. Bower et al. 1997. *A Practical Guide to Air Quality Monitoring*. National Environmental Technology Centre, England.

18. U.S. Environmental Protection Agency. 1992. *Guidance on Risk Characterization for Risk Managers and Risk Assessors.* Risk Assessment Council, Washington, D.C., February 26, 1992.

19. U.S. Environmental Protection Agency. 1992. *Guidelines for Exposure Assessment. Federal Register* 57:22888-22938, May 29, 1992. Available at http://cfpub.epa.gov/ncea/raf/recordisplay.cfm?deid=15263

Chapter 12 Inhalation Toxicity Assessment

Table of Contents

12.1 Introduction 1
12.1.1 Hazard Identification and Dose-Response Information 1
12.1.2 Dose-Response Assessment Methods 5

12.2 Hazard Identification 8
12.2.1 Weight of Evidence – Human Carcinogenicity 9
12.2.2 Identification of Critical Effect(s) – Non-Cancer Endpoints 11

12.3 Dose-Response Assessment for Cancer Effects 12
12.3.1 Determination of the Point of Departure (POD) 13
12.3.2 Derivation of the Human Equivalent Concentration 14
12.3.3 Extrapolation from POD to Derive Carcinogenic Potency Estimates 17

12.4 Dose-Response Assessment for Derivation of a Reference Concentration 19
12.4.1 Determination of the Point of Departure and Human Equivalent Concentration 20
12.4.2 Application of Uncertainty Factors 22

12.5 Sources of Chronic Dose-Response Values 24

12.6 Acute Exposure Reference Values 26

12.7 Evaluating Chemicals Lacking Health Reference Values 31
12.7.1 Use of Available Data Sources 31
12.7.2 Route-to-Route Extrapolation 31

12.8 Dose-Response Assessment for Mixtures 32

References 35

12.1 Introduction

The purpose of the toxicity assessment is to weigh available evidence regarding the potential for toxicity in exposed individuals (**hazard identification**) and to quantify the toxicity by deriving an appropriate dose-response value (**dose-response assessment**). Toxicity assessment is the second part of the general risk equation. Although the toxicity assessment is an integral and important part of the overall air toxics risk assessment, it is usually accomplished prior to the risk assessment. EPA has completed this toxicity assessment for many HAPs and has made available the resulting toxicity information and dose-response values, which have undergone extensive peer review (see Appendix C).[1]

Risk = *f* (metric of exposure, metric of toxicity)

Toxicity Assessment is a 2-Step Process:

1. **Hazard Identification** – What types of effects does the chemical cause? Under what circumstances?
2. **Dose-response Assessment** – How potent is the chemical as a carcinogen and/or for noncancer effects?

In most air toxics risk assessments, little new toxicological evaluation of primary data will be required. However, it is important to understand how the available data were analyzed to produce the dose-responses values used in a risk assessment. In the risk characterization step, the risk assessor will need to describe the nature of the available toxicological evidence and the uncertainties inherent in the development of the dose-response values used in the inhalation risk assessment (see Chapter 13).

Additionally, in the event that there are significant data analysis and interpretation issues, or if a dose-response value does not exist and needs to be developed for a particular air toxic of interest, this chapter provides information about how to locate toxicity assessments, accompanying dose-response values, and relevant guidance documents. However, development and interpretation of toxicity information and dose-response values requires toxicological expertise and should not be undertaken by those without appropriate training and experience.

12.1.1 Hazard Identification and Dose-Response Information

As part of the hazard identification step, evidence is gathered from a variety of sources regarding the potential for an air toxic to cause adverse health effects in humans. These sources may include human data, experimental animal studies, and supporting information such as *in vitro* laboratory tests. The source of data affects the overall uncertainties in the resulting human dose-response values, as discussed below.

- **Human data**. Human toxicity data associated with exposures to air toxics may be located in epidemiological studies, controlled exposure studies, or studies of accidental exposures. Well-conducted epidemiological studies that show a positive association between exposure to a chemical and adverse health effects often provide evidence about human health effects associated with chronic exposures. Such data, however, are available only for a limited number of air toxics. Epidemiological data also are very difficult to interpret, because the number of exposed individuals may be small, the incidence of effects may be low, doses are usually not well-characterized, and there may be complicating factors such as simultaneous exposure to multiple chemicals and heterogeneity among the exposed group in terms of age, sex, diet, and other factors. Controlled exposure studies provide stronger evidence, since both the exposure duration and exposure concentrations are more accurately known. However, such studies with humans are generally limited to acute exposure durations. Studies reporting health effects associated with accidental exposures may be helpful, although exposure concentrations to air toxics may be high, and effects may be acute rather than chronic. Also note that small sample size is often a significant limitation to interpreting controlled and accidental exposure studies.

> **Epidemiology** is the study of the distribution and determinants of disease or health status in a population.

- **Animal data**. The toxicity database for most air toxics is drawn from experiments conducted on non-human mammals such as rats, mice, rabbits, guinea pigs, hamsters, dogs, or monkeys. The underlying assumption is that the susceptibility of humans and these animals to the effects of the chemicals is broadly similar because we share many common biological attributes (e.g., similar organs, similar and, in some cases, identical metabolic processes). However, some observations in animals may be of uncertain relevance to humans (e.g., if tumors are observed in an animal experiment, but the organ in which the tumor is formed does not exist in humans). Also, it is necessary to adjust the results from animal studies to humans due to differences in body mass, anatomy, metabolic rate, and other species-specific factors (see, for example, Section 12.3.3). This is why derivation of dose-response values from animal studies requires considerable expertise.

- **Supporting data**. Metabolic, pharmacokinetic, and genotoxicity studies are sometimes used to infer the likelihood of adverse effects in humans. Metabolic studies on absorption, distribution, metabolism, and elimination can provide information about the mechanisms of toxicity associated with a particular chemical in humans. In physiologically based pharmacokinetic (PBPK) models,[a] the body is subdivided into a series of anatomical or physiological "compartments" that represent specific organs or lumped tissue and organ groups, and the behavior of the chemical is modeled in each compartment. Data on a chemical's pharmacokinetics, genotoxicity, and possible mode of action can be used to refine a toxicity assessment. In some cases, computer models using structure-activity relationships (i.e., predictions of toxicological activity based on analysis of chemical structure) also may be used as supporting evidence. EPA considers these types of data to be supportive, not definitive, evidence of a chemical's toxicity.

---

[a] A PBPK model estimates the dose to a target tissue or organ by taking into account the rate of absorption into the body, distribution among target organs and tissues, metabolism, and excretion.

Information from these sources is considered in the hazard and dose-response assessment steps in characterizing a chemical with regard to the type(s) of effect a chemical produces (the hazard) and the circumstances in which this occurs, as well as the level of exposure required to produce that effect. The output of the dose-response assessment is the relationship between **dose** (the level of exposure) and the resulting **response** (the increased incidence and/or severity of adverse effects). A dose-response assessment is the process of quantitatively evaluating toxicity information, characterizing the relationship between the dose of the contaminant received (or the inhalation exposure concentration, for inhalation assessments) and the incidence of adverse health effects in the exposed subjects (which may be animal or human) and then, as appropriate, extrapolating these results to human populations. Depending on the type of effect and the chemical, there are two types of dose-response values that traditionally may be derived: predictive cancer risk estimates, such as the **inhalation unit risk estimate (IUR)**, and predictive non-cancer estimates, such as the **reference concentration (RfC)**.[b] Both types of dose-response values may be developed for the same chemical, as appropriate.

Inhalation Dose-Response Values[a]

Inhalation Unit Risk (IUR): The upper-bound excess lifetime cancer risk estimated to result from continuous exposure to an agent via inhalation per $\mu g/m^3$ over a lifetime. The interpretation of the IUR would be as follows: if IUR = 2×10^{-6} $\mu g/m^3$, not more than 2 excess tumors are expected to develop per 1,000,000 people if exposed continuously for a lifetime to 1 µg of the chemical per cubic meter of inhaled air. The number of expected tumors is likely to be less; it may even be none.

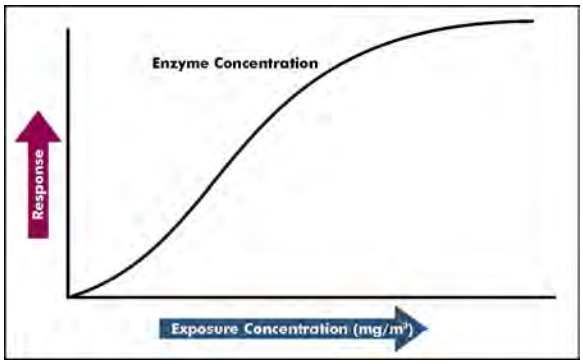
Reference Concentration (RfC): An estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive sub-populations) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Generally used in EPA's noncancer health assessments.

[a]The phrase "dose-response" is used generally here and elsewhere in the document. EPA's values for inhalation, however, are derived for exposure concentration, although with consideration of dose. Consideration of the relationship between exposure concentration, dose, and dosimetry (how the body handles a chemical once it is inhaled) is inherent in the derivation of these exposure concentration-response values.

The relationship of dose to response can be illustrated as a graph called a **dose-response curve**. There are two general types of response data that may be considered and graphed. One is termed "continuous" and refers to responses such as the severity in changes to a physiological parameter in a given individual as dose increases (see Exhibit 12-1, A) . The second describes the incidence of a particular response in a population (see Exhibit 12-1, B). By convention, dose or exposure is represented on the x-axis; response on the y-axis (Exhibit 12-1).

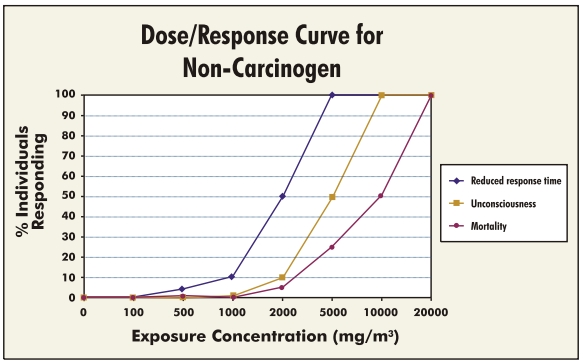
[b]While the majority of RfCs are derived for effects other than cancer, RfCs may be derived for all effects, including cancer, when a non-linear mode of action has been demonstrated for carcinogenicity.

Exhibit 12-1. Examples of Dose-Response Curves



A. Continuous Response Data

Simple example of a dose-response curve for graded responses of a specific physiological parameter to increasing exposure.



B. Different Responses in a Population

Simple example of the incidence of three different effects in an exposed population in response to different exposure concentrations (over the same duration).

While the primary focus of this chapter is on description of dose-response values relevant to chronic (long-term) exposures, the information reviewed for developing those values may include effects associated with acute (short-term) exposures. Additionally, information on acute exposures is essential to the development of acute exposure reference values (see Section 12.6).

- **Acute exposures** are usually relatively short in duration, but relatively high in concentration and may result in immediate respiratory and sensory irritation, chemical burns, narcosis, eye damage, and various other effects. Acute exposures also may result in longer-term health effects.

- **Chronic exposures** are usually relatively long in duration, but relatively low in concentration and may result in health effects that do not show up immediately and that persist over the long term, such as cardiovascular disease, respiratory disease, liver and kidney disease, reproductive effects, neurological damage, and cancer.

Generally, chronic reference values are derived for exposure periods between seven years and a lifetime. Acute reference values (see section 12.6) are generally developed for very short exposures (e.g., hours to days; Exhibit 12-2). For intermediate exposures, subchronic reference values are available from some sources (e.g., ATSDR). Most air toxics risk assessments will focus on chronic and acute evaluations; however, under more limited circumstances, subchronic evaluations may be performed.

Exhibit 12-2. Reference Values of Different Durations
In the Agency's *Review of the Reference Dose and Reference Concentration Processes*,[2] it was recommended that in addition to the traditional chronic reference value (i.e., RfC or RfD) included in the IRIS database, values of several shorter durations also be developed, where possible. As a first step in this direction, the *Review* proposed the following definitions. EPA currently is considering these and other recommendations made in the *Review*. These definitions are based on exposure durations for humans, and were not intended to be rigid specifications, but simply general descriptions of the relevant exposure time period. • **Acute**: Exposure by the oral, dermal, or inhalation route for 24 hours or less. • **Short-term**: Repeated exposure[(a)] by the oral, dermal, or inhalation route for more than 24 hours, up to 30 days. • **Longer-term**: Repeated exposure by the oral, dermal, or inhalation route for more than 30 days, up to approximately 10 percent of the life span in humans[(b)] (more than 30 days up to 90 days in typically used laboratory animal species[(c)]). • **Chronic**: Repeated exposure by the oral, dermal, or inhalation route for more than approximately 10 percent of the life span in humans (more than approximately 90 days to 2 years in typically used laboratory animal species).
[(a)]A repeated exposure may be either continuous, periodic, or intermittent. A continuous exposure is a daily exposure for the total duration of interest. A periodic exposure is one occurring at regular intervals (e.g., inhalation exposure 6 hours/day, 5 days/week; or oral exposure 5 days/week). An intermittent exposure is one in which there is no effect of one exposure on the effect of the next; this definition implies sufficient time for the chemical and its metabolites to clear the biological system before the subsequent (i.e., noncumulative pharmacokinetics). A periodic exposure may or may not be intermittent. [(b)]An average of 70 years is typical default used for chronic exposures. [(c)]Examples of typically used laboratory species include rats, mice, and rabbits.

12.1.2 Dose-Response Assessment Methods

Depending on whether a substance causes cancer and whether its dose-response curve is thought to have a threshold, EPA may use either of two approaches in a dose-response assessment. One approach produces a predictive estimate (e.g., inhalation cancer risk estimate), and the other produces a reference value (e.g., RfC). Historically, the use of a predictive estimate has been limited to cancer assessment. That is, dose-response assessments for cancer have been expressed as predictive cancer risk estimates based on an assumption that any amount of exposure poses some risk. Assessments of effects other than cancer usually have been expressed as reference values at or below which no harm is expected. Many substances have been assessed both ways: the first for cancer and the second for adverse effects other than cancer. While this use of predictive estimates for cancer and reference values for other effects is still the practice for the vast majority of chemicals, EPA now recognizes that there are chemicals for which the data support an alternate approach.

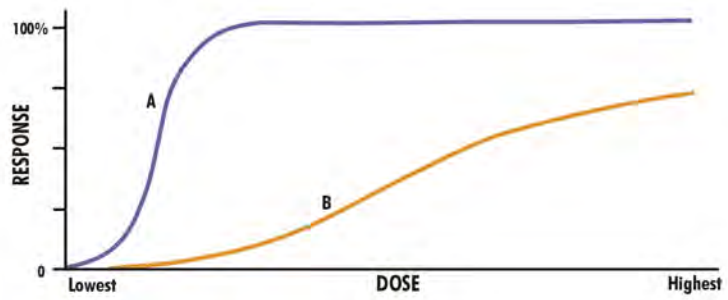
An important aspect of dose-response relationships is whether the available evidence suggests the existence of a threshold. For many types of toxic responses, there is a **threshold dose** or dose rate below which there are thought to be no adverse effects from exposure to the chemical. The human body has defenses against many toxic agents. Cells in human organs, especially in the liver and kidneys, break down many chemicals into less toxic substances that can be eliminated from the body in urine and feces. In this way, the human body can withstand some chemical exposure (at doses below the threshold) and still remain healthy. For example, many air toxics are naturally occurring substances to which people routinely receive trace exposures at non-toxic levels.

Identification of a threshold dose depends on the type of response and the way in which the toxic chemical produces it. EPA has developed guidelines [3] for assessing the dose-response for various types of adverse effects, which provide more information about evaluating evidence to determine if a threshold exists.

> *All substances are poisons: there is none which is not a poison. The right dose differentiates a poison and a remedy.*
> – Paracelsus

Both the point at which the dose-response curve begins to ascend (its threshold, which may be zero) and the slope of the curve (its steepness) provide information about the toxicity of a chemical (Exhibit 12-3). The potency of a chemical is a measure of its strength as a toxicant compared with other chemicals. Therefore, the lower the threshold dose, the more potent (or toxic) the chemical. The slope of the curve is a measure of the range of doses from the threshold dose (at which the adverse effect is first measured) to the dose at which the effect is complete (i.e., higher doses produce no additional incidence of that effect, although other adverse effects may begin to appear). The steeper the dose-response curve, the smaller the range between the first appearance of an effect and a substantial response.

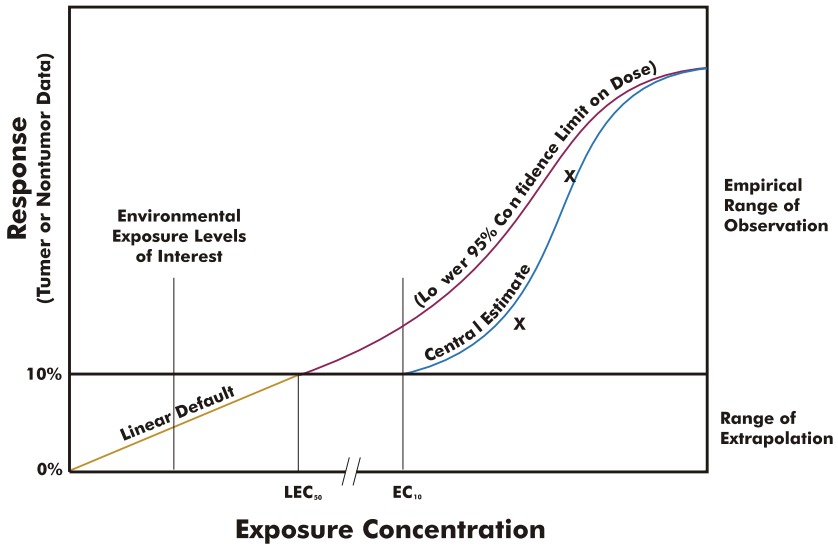
Different Responses Exhibit Different Dose-Response Curves



Line A – A sharp increase in response with increasing dose
Line B – A more gradual increase in response with increasing dose

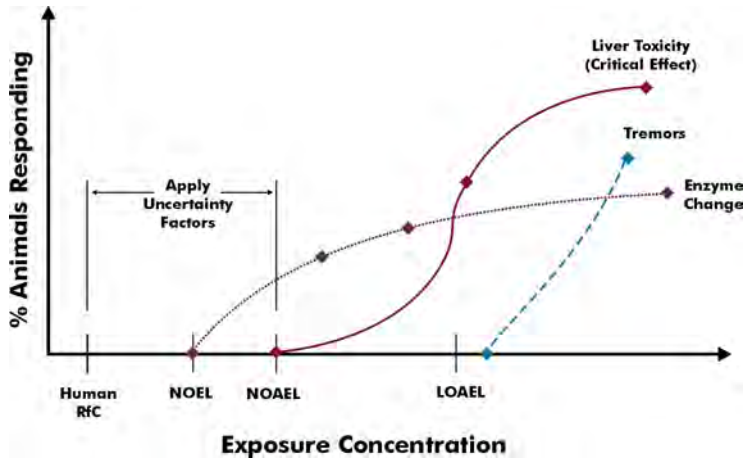
Exhibit 12-3. Dose-Response Relationships for Carcinogens and Noncarcinogens

A. Example Linear Carcinogen



In the absence of clear evidence to the contrary, EPA assumes as a matter of science policy that even a very low exposure to a cancer-causing pollutant can increase the risk of cancer (albeit a small amount). Experimental data are used to construct a dose-response relationship and identify the point of departure – the dose that can be considered to be near or in the range of observed responses and, thus, no significant extrapolation is needed. To estimate the dose-response relationship at doses below the point of departure, the dose-response relationship between the point of departure and zero is assumed to be linear. Thus, at doses below the point of departure, with each unit of increase in exposure (dose), there is an increase in cancer response. Where evidence supports the acceptance of a non-linear mode of action, a reference concentration approach may be employed, as shown in "B" below. LEC_{50}= lethal effective concentration for 50 percent of the population; EC_{10}= effective concentration that causes an observable adverse effect in 10 percent of the population.

B. Example Non-linear Approach



A dose may exist below the minimum health effect level for which no adverse effects occur. EPA typically assumes that at low doses the body's natural protective mechanisms prevent or repair any damage caused by the pollutant, so there is no ill effect at low doses. Even long-term (chronic) exposures below the threshold are not expected to have adverse effects. The dose-response relationship (the response occurring with increasing dose) varies with pollutant, individual sensitivity, and type of health effect. NOEL = no-observed-effect-level; NOAEL = no-observed-adverse-effect-level; LOAEL = lowest-observed-adverse-effect-level.

Epidemiologic and toxicologic data on air toxics typically result from exposure levels that are high relative to environmental levels. Therefore, **low-dose extrapolation** (prediction) is necessary to derive an appropriate dose-response value. For a few air toxics (e.g., the criteria air pollutants ozone and carbon monoxide), data are sufficient to characterize dose-response relationships at environmental levels. In such cases, there is no need for extrapolation of toxicity data to lower doses. Such is not the case for most air toxics. Low-dose extrapolation requires either information or assumptions about the type of dose-response curve likely under low dose situations. EPA risk assessment guidelines provide more detailed information on how EPA performs low-dose extrapolation for chemicals with various toxic effects, such as developmental effects or neurotoxic effects.[3]

12.2 Hazard Identification

The hazard identification, which is usually part of an existing dose-response assessment for each chemical, provides a summary of the available toxicity information for the air toxics being studied, and includes the weight of evidence determination and identification of critical effects. This step should answer the following questions:

Items to Include in the Hazard Identification of an Air Toxics Risk Assessment

- List of chemicals detected
- Summaries of toxic effects and quality of the toxicological evidence
- Discussion that focuses the risk assessment on chemicals most likely to cause adverse effects

- Can exposure to a chemical be linked causally to particular health effects?
- Could these effects occur at environmentally relevant concentrations?
- What is the nature and strength of the evidence of causation?

By definition, all HAPs and many other air toxics have the potential to cause adverse effects in the exposed population. Exhibit 12-4 provides examples of cancer and non-cancer effects, and Appendix C identifies which HAPs have been associated with carcinogenic (cancer) effects or non-cancer effects, along with the strength and ratings of the toxicity evidence that has been evaluated by EPA or other international environmental agencies.

Exhibit 12-4. Examples of Adverse Health Effects
• Birth defects • Tremors • Infertility • Skin rash • Melanoma

An air toxics risk assessment should include in its hazard identification a summary of the quality of the toxicological evidence (i.e., the nature and strength of the evidence of causation) for the chemicals of concern. Study factors such as the route of exposure used, the type and quality of health effects, the biological plausibility of findings, and the consistency of findings across studies all contribute to the strength of the hazard identification statement.

12.2.1 Weight of Evidence – Human Carcinogenicity

A major determination made during the hazard identification step concerns the potential of a chemical to cause cancer in humans. This determination, which involves considering (or weighing) all the available evidence, is called the weight of evidence determination. This determination is complicated by possible inadequacies of the published studies, as well as differences in body processes between people and laboratory animals. EPA's *Guidelines for Carcinogen Risk Assessment* guide scientists in interpreting available studies to assess the potential human carcinogenicity of environmental pollutants. (EPA's carcinogen risk assessment guidelines were first published in 1986. Revisions were proposed in 1996 and 2001 and the July 1999 draft of the revisions was adopted as interim guidance. A subsequent 2003 draft of the Guidelines has been released for public and scientific review prior to adoption as final. The guidelines are available on the web.)[4] When compared with EPA's original 1986 guidelines, the 1999 interim Guidelines recommend a more comprehensive evaluation of the evidence with regard to a chemical's potential mode of action, and a more complete description of the context of a chemical's carcinogenic potential (e.g., "likely carcinogenic by inhalation and not likely carcinogenic by oral exposure"). The weight of evidence determination now includes one of five descriptors, and is accompanied by additional text that more completely summarizes EPA's interpretation of the evidence. The narrative statements consider the quality and adequacy of data and the consistency of responses induced by the agent in question (see Exhibit 12-5).

Exhibit 12-5. Information Regularly Included in a Narrative Statement Describing the Characterization of Weight of Evidence for Carcinogenicity (1999 Interim Guidelines)

- Name of agent and Chemical Abstracts Services number, if available
- Conclusions (by route of exposure) about human carcinogenicity, using one of five standard descriptors: "Carcinogenic to Humans" "Likely to be Carcinogenic to Humans" "Suggestive Evidence of Carcinogenicity, but Not Sufficient to Assess Human Carcinogenic Potential" "Data are Inadequate for An Assessment of Human Carcinogenic Potential" "Non Likely to be Carcinogenic to Humans".
- Summary of human and animal tumor data on the agent or its structural analogues, their relevance, and biological plausibility
- Other key data (e.g., structure-activity data, toxicokinetics and metabolism, short-term studies, other relevant toxicity or clinical data)
- Discussion of possible mode(s) of action and appropriate dose-response approach(es)
- Conditions of expression of carcinogenicity, including route, duration, and magnitude of exposure

Source: EPA (1999) *Guidelines for Carcinogen Risk Assessment. Review Draft*[(4)]

Many existing carcinogen assessments were developed pursuant to EPA's 1986 *Guidelines for Carcinogen Risk Assessment*, which used a simpler but less informative weight of evidence system (see Exhibit 12-6).

Information bearing on the qualitative assessment of carcinogenic potential may be gained from human epidemiological data, animal studies, comparative pharmacokinetic and metabolism studies, genetic toxicity studies, structure-activity relationship (SAR) analysis, and other studies of an agent's properties. Information from these studies helps to elucidate potential modes of action and biological fate and disposition.

Exhibit 12-6. EPA's Weight of Evidence Classification for Carcinogens (1986 Guidelines)	
Group A:	Human Carcinogen (sufficient evidence of carcinogenicity in humans)
Group B:	Probable Human Carcinogen (B1 - limited evidence of carcinogenicity in humans; B2 - sufficient evidence of carcinogenicity in animals with inadequate or lack of evidence in humans)
Group C:	Possible Human Carcinogen (limited evidence of carcinogenicity in animals with inadequate or lack of human data)
Group D:	Not Classifiable as to Human Carcinogenicity (inadequate or no evidence)
Group E:	Evidence of Noncarcinogenicity for Humans (no evidence of carcinogenicity in adequate studies)
Source: EPA (1986). *Guidelines for Carcinogen Risk Assessment*[4]	

Upon such consideration, both EPA systems assign a consensus interpretation to the weight of evidence, evaluating the likelihood that the agent is a human carcinogen. Toxicological evidence is characterized separately for human studies and animal studies as: sufficient, limited, inadequate, no data, or evidence of no effect. The characterizations of these two types of data are combined, and based on the extent to which the agent has been shown to be a carcinogen in experimental animals or humans, or both, the chemical is given a weight of evidence classification.

Generally, no single factor is determinative. For example, strength of association is one of the criteria for causality. A strong association between exposure and cancer in animals is more likely to indicate causality than a weak association. However, finding of a large cancer incidence in a single study must be balanced against the lack of consistency as reflected by null results from other equally well-designed and well-conducted studies. In this situation, the positive association of a single study may either suggest the presence of chance, bias, confounding factors, or different exposure conditions. On the other hand, evidence of weak but consistent associations across several studies suggests either causality or that the same confounder may be operating in all of these studies.

If information is available to consider the mode of action for carcinogenicity, the carcinogenicity assessment will evaluate that information and draw conclusions that influence the dose-response method for the substance. If the evidence is sufficient to support a conclusion of nonlinear dose-response, then the information on carcinogenicity may be considered in combination with the information on other effects in deriving a reference value such as an RfC (see section 12.4). Otherwise, a linear dose-response approach leading to a predictive risk estimate, such as an IUR, will usually be pursued. If the information supports it, the guidelines also accommodate the development of a non-linear predictive risk estimate.

Biological Effects of Carcinogens

Carcinogens are chemicals that induce cancers. Examples include:

- *4-Aminobyphenol*, which targets the bladder;
- *Benzene*, which targets the tissue that make white blood cells;
- *Asbestos*, which targets the lung's tissue;
- *Benzidene*, which targets the bladder;
- *Beryllium*, which targets the lungs;
- *Chromium*, which targets the respiratory tract;
- *Radionucleotides*, which targets bone marrow and the lungs; and
- *Vinyl chloride*, which targets the liver.

There are various types of carcinogens, including:

- **Primary Carcinogens**: A primary carcinogen is a substance that is carcinogenic as it occurs in the environment.
- **Procarcinogen**: A procarcinogen is a substance that becomes carcinogenic only after conversion from some benign form. Most environmental carcinogens are of this type.
- **Cocarcinogen**: A cocarcinogen is a substance that is not carcinogenic by itself, but potentiates the carcinogenic effect of other chemicals.

Chemicals also can serve as **mutagens**, causing changes in genetic material that can disrupt cell function and lead to cancer or other health problems.

12.2.2 Identification of Critical Effect(s) – Non-Cancer Endpoints

As part of the characterization of the available information on non-cancer health effects (or including cancer, if a threshold mode of action has been established), the targets of chemical toxicity within the body are identified, along with what have been termed "critical effects" associated with the toxicity. A **critical effect** is described as "either the adverse effect that first appears in the dose scale as dose is increased, or as a known precursor to the first adverse effect." Underlying this designation is the assumption that if the critical effects are prevented, then all other adverse effects observed at higher exposure concentrations or doses are also prevented.[(c)] Note that not all observed effects in toxicity studies are considered adverse effects. The identification of the critical effect(s) depends on a comprehensive review of the available data with careful consideration of the exposure conditions associated with each observed effect, so that comparisons of effect levels or potential reference values are made on a common basis (see Section 12.4). A more comprehensive discussion of hazard identification and the evaluation of the underlying database for non-cancer effects is included in the EPA documents *Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry* (1994) and *A Review of the Reference Dose and Reference Concentration Process* (2002).[5]

---

[c]A similar, more recent term, "key event," is defined as "an empirically observed precursor" to an adverse effect (e.g., liver cancer or other liver toxicity) consistent with a particular mode of action. The phrase "mode of action" refers to the way a given chemical may act in the body to initiate one or more adverse effects.

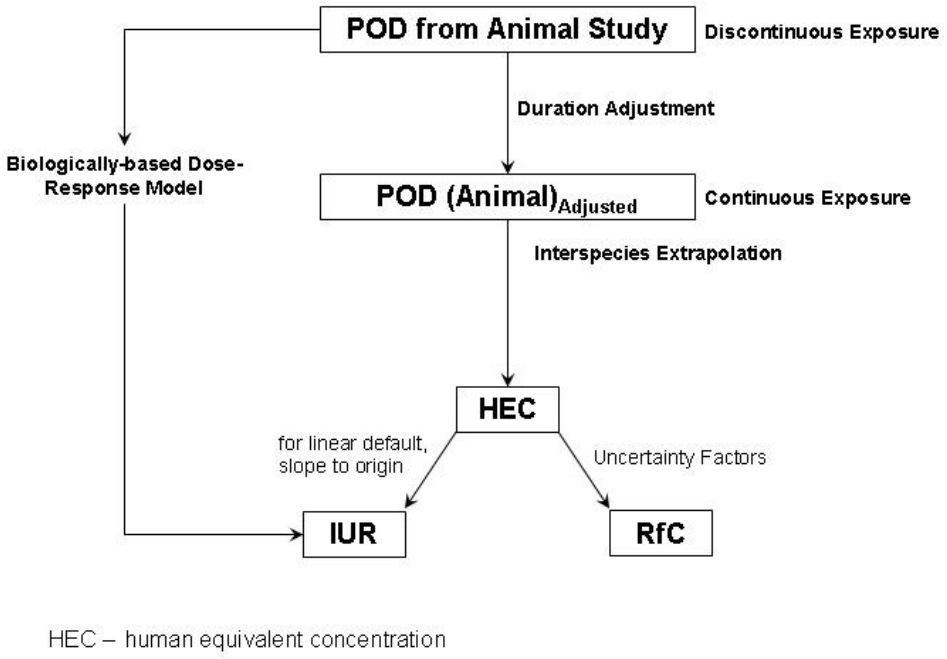
12.3 Dose-Response Assessment for Cancer Effects

The process for deriving a quantitative dose-response estimate for cancer (e.g., a cancer slope factor) involves the following three steps:

1. Determination of the concentration associated with the point of departure or POD (Section 12.3.1);
2. Derivation of the human equivalent concentration corresponding to the POD (Section 12.3.2); and
3. Extrapolation from the POD (expressed as human equivalent concentration) to derive carcinogenic potency estimates (Section 12.3.3).

The first two steps are also performed in the derivation of reference values such as the RfC (Exhibit 12-7); in that case, these steps are followed by the application of uncertainty factors (see Section 12.4).

Exhibit 12-7. Steps involved in deriving an RfC or IUR From an Animal Study



12.3.1 Determination of the Point of Departure (POD)

Dose-response assessment for cancer and other effects begins with identification of the point of departure (an exposure concentration or intake) from the experimental data. This point (in terms of its human equivalent), while within the range of observation, is the point from which extrapolation begins, either for the purposes of deriving a cancer risk estimate (the IUR) or a RfC for non-cancer health effects.

Example POD for Benzene

EPA's characterization of the carcinogenic effects of benzene was updated in 1998. The IUR for benzene is based on epidemiologic studies showing clear evidence of a causal association between exposure to benzene and leukemia. The specific mechanisms by which benzene and its metabolites lead to cancer remain uncertain.

EPA selected the Rinsky et al. 1981 epidemiologic study of 1,165 Pliofilm rubber male workers at three facilities in Ohio as the data set for the dose-response relationship for determining the IUR. The workers had been employed between 1940 and 1965 and were followed through 1981. Rinsky et. al. expanded the study to include additional workers and published it in 1987. The Rinsky data suffers - as many epidemiologic studies do - from uncertainties about exposure levels in the early years. There are no measurements of benzene in the facilities' air prior to 1946, so exposures for these years must be estimated.

Using one set of exposure estimates with the Rinsky et al. study, EPA concluded that exposure to benzene increases the risk of leukemia at a level of 40 ppm-years of occupational exposure (8 hours/day, 5 days/week, 50 weeks/year). Below this number, the shape of the dose-response curve cannot be determined. Converting the occupational exposure of 40 ppm-years to an equivalent lifetime of environmental exposure yields 120 ppb, as a POD, below which the shape of the dose-response curve is uncertain.

EPA decided there is not sufficient evidence to demonstrate that the dose-response relationship below the POD is non-linear. As a science policy default, EPA assumed low-dose linearity for extrapolation from the POD to zero. Given a range of plausible exposure estimates for the Rinsky et al. study, the Agency determined that the benzene inhalation unit risk at 1 $\mu g/m^3$ ranges from 7.1×10^{-3} to 2.5×10^{-2} depending on the exposure estimates and modeling approach used to derive the POD.

Source: U.S. EPA. 1998. *Carcinogenic Effects of Benzene: An Update.* Office of Research and Development, National Center for Environmental Assessment, Washington, D.C. EPA/600/P-97/001F.; Rinsky, R.A., Young, R.J., and Smith, A.B. 1981. Leukemia in benzene workers. *American Journal of Industrial Medicine.* 2(3) 217:245.

The POD may be the traditional no observed adverse effect level (NOAEL), lowest observed adverse effect level (LOAEL), or **benchmark concentration (BMC)**.[(d)] EPA has recommended the use of the BMC approach, where possible, because the traditional use of the LOAEL or NOAEL in determining the POD has long been recognized as having several limitations (and

[d]Note that the corresponding value for ingestion exposures is the benchmark dose (BMD). This often is used as the general term for the BMC/BMD process.

generally is not used in dose-response for cancer effects. In particular, the LOAEL-NOAEL approach:

- Is limited to one of the doses in the study and thus is dependent on study design;
- Does not account for variability and uncertainty in the estimate of the dose-response relationship;
- Does not account for the slope of the dose-response curve; and
- Cannot be applied, where there is no NOAEL, except through the application of an uncertainty factor.

If the dose-response data are of high quality, a mathematical dose-response model may be fitted to the data to determine a more precise POD than the NOAEL or LOAEL. When a model is used, the POD is calculated as the statistical lower confidence limit of the dose at which there is a low toxic response (usually 5 or 10 percent incidence in populations with an effect or a change in a physiological measurement indicating adversity).[(6)] The selection of the response percentage is intended to coincide with the sensitivity limit of the experimental design or professional judgment. This calculated POD is called the BMC.

The BMC approach is an alternate way of determining the point of departure for low-dose extrapolation. It can be used in cancer and noncancer risk assessment as the starting point for linear low-dose extrapolation, calculation of a margin of exposure, or application of uncertainty factors for calculating RfCs or other dose-response values. BMC methods involve fitting various mathematical models for dose-response to reported data and using the different results to select a BMC that is associated with a predetermined benchmark response, such as a 10 percent increase in the incidence of a particular lesion or a 10 percent decrease in body weight gain (Exhibit 12-8). EPA has developed the Benchmark Dose Software (BMDS) to facilitate these operations. BMDS currently offers 16 different mathematical models that can be fit to the laboratory data. EPA plans to continually improve and expand the BMDS system.[6]

It is likely that there will continue to be situations that are not amenable to BMC modeling and for which a NOAEL or LOAEL approach should be used. In some cases, there may be a combination of benchmark doses and NOAELs to be considered in the assessment of a particular agent.

12.3.2 Derivation of the Human Equivalent Concentration

Because inhalation toxicity studies typically involve discontinuous exposures (e.g., animal studies routinely involve inhalation exposures of 6 hours per day, 5 days per week), the POD will usually need to be extrapolated to a continuous exposure scenario (as appropriate for the RfC and IUR). This duration adjustment step is essential in interpreting inhalation studies, but is not routinely necessary for the interpretation of oral exposures. Operationally, this is accomplished

by applying a concentration-duration product, or **C × t product**[e] for both the number of hours ina daily exposure period and the number of days per week that the exposures are performed. For example, for a POD of 100 mg/m^3 derived from an animal study in which animals are exposed by inhalation for 6 hours per day, 5 days per week, the adjustment to a continuous

$$100\,mg/m^3 \times \frac{6}{24} \times \frac{5}{7} = 18\,mg/m^3 \qquad \text{(Equation 12-1)}$$

exposure concentration would consider both hours per day and days per week:

Thus, 18 mg/m^3 is the POD concentration adjusted for continuous exposure versus 100 mg/m^3 unadjusted. This approach assumes there is no dose-rate effect (i.e., that the same total inhaled material produces the same effect regardless of the time over which this material was inhaled).

Exhibit 12-8. Example Derivation of Benchmark Dose Level

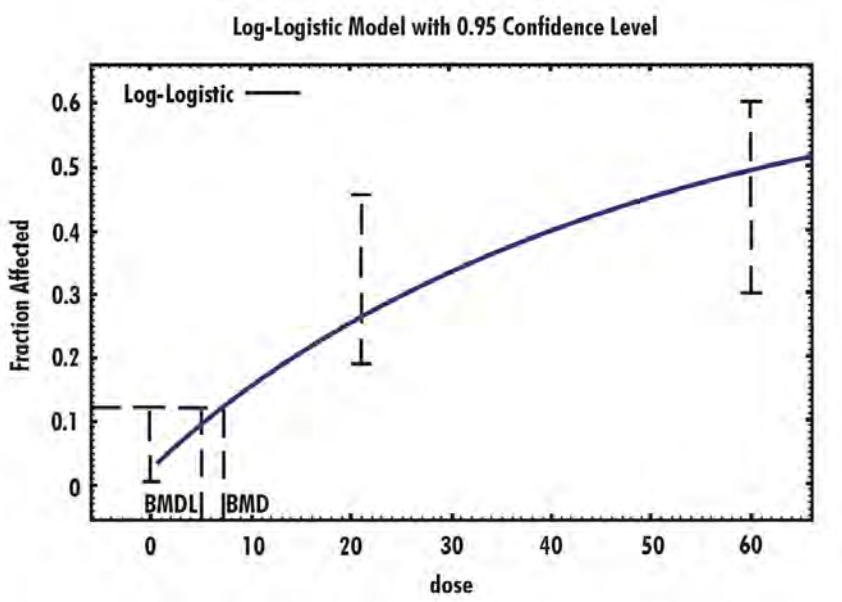


Illustration of the computation of a benchmark dose (BMD) and BMDL (a lower one-sided confidence limit on the BMD) for an extra risk of 0.10 (as suggested by the BMDS guidance document), using a one-sided 95 percent confidence interval. A number of models were fit to the data, and the log-logistic model illustrated provides the best fit to the example data. The predicted curve comes well within the confidence limits for each data point. Other data and models are illustrated in examples provided in the BMDS guidance document.[6]

[e] "C × t" is a component of Haber's Law that refers to the default assumption (in lieu of information to the contrary) that effects observed are related to the cumulative exposure or "area under the curve" (quantified by concentration, C, multiplied by duration, t). It is noted that when going from a discontinuous inhalation exposure regiment to a continuous exposure, the result will always be a lower value for concentration, thus providing an automatic margin of protectiveness for chemicals for which C alone (vs. C × t) may be appropriate, while providing the appropriate conversion for substances for which cumulative exposure is the appropriate measure.[4]

Exposures documented from human occupational epidemiological studies are most often reported as 8-hr time-weighted averages (TWAs) and therefore, also are discontinuous. Adjustment of these exposures is usually done as part of the dosimetric adjustment to derive a human equivalent concentration (HEC), rather than as a discrete step, and is explained below in Section 12.3.3. The duration adjustment step also is explicitly incorporated into physiologically based pharmacokinetic (PBPK) models used to extrapolate an animal or occupational study-derived POD into an HEC.

After duration adjustment, the POD is converted into a **human equivalent concentration (HEC)** from the experimental animal dose. This conversion may be done using default methods specific to the particular chemical class of concern or more refined methods such as PBPK modeling.

The Agency's inhalation dosimetry methodology[5] provides a recommended hierarchy, as well as default generalized procedures for deriving **dosimetric adjustment factors** (**DAFs**) for this extrapolation. Application of DAFs to an animal exposure value yields an estimate of the corresponding concentration relevant to humans (i.e., the HEC) given differences in physiology and in the form of the pollutant that influence how the chemical exerts its effect. The DAF depends on the chemical category (i.e., gas or particle) and whether the adverse effect occurs in the respiratory tract or outside of the respiratory tract. HECs are derived using DAFs for both RfC development (noncancer effects) and IUR development (cancer).

Choice of a Default DAF for Extrapolation from Animal Data Depends on the Physical and Chemical Properties of the Pollutant

Gases

- Category 1 (effect in respiratory system) – default DAF based on inhalation rate, and surface area of target portion of respiratory tract
- Category 2 (some characteristics intermediate or common to category 1&3) – default DAF is the more restrictive of the defaults for category 1 & 3
- Category 3 (systemic effect[s]) – default DAF based on blood:air partition coefficient

Particles

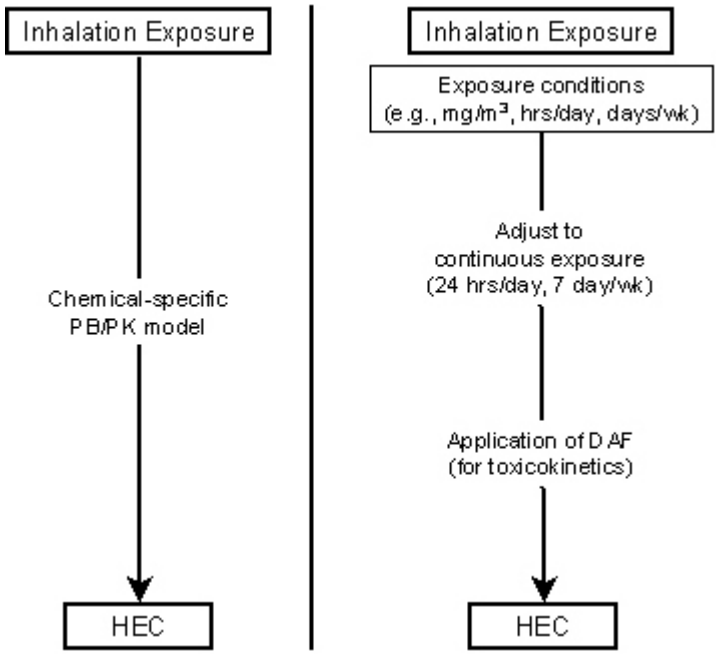
- Respiratory toxicant – default DAF based on fractional deposition, inhalation rate, and surface area of target portion of respiratory tract
- Systemic toxicant – default DAF based on inhalation rate, body weight, and fractional deposition

Source: U.S. EPA. 1994. *Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry*.[5]

When data are adequate to support it, the preferred EPA approach for calculating a HEC is to use a chemical-specific PBPK model parameterized for the animal species and regions (e.g., of the respiratory tract) involved in the toxicity (Exhibit 12-9).

In PBPK models, the body is subdivided into a series of anatomical or physiological "compartments" that represent specific organs or tissue and organ groups. The transfer of chemicals between compartments is described by a set of differential equations. The parameters of the model are of three types: physiological parameters (such as tissue perfusions or tissue volumes), physicochemical parameters (such as partition coefficients that describe the degree of partitioning of a given chemical to a given tissue), and biochemical parameters describing metabolic processes. The structure of a PBPK model is determined by the intended use of the model, the biochemical properties of the chemical studied, and the effect site of concern.

Exhibit 12-9. Extrapolation of Inhalation Exposure to Calculate the HEC



EPA employs a hierarchy of approaches for deriving the human equivalent concentration. Preference is given to the use of a physiologically-based pharmacokinetic model, followed by intrermediate, less detailed approaches, which are followed by the default approach, which utilizes a DAF specific to the type of chemical and how it exerts its effect.

With sufficient data, a PBPK model is capable of calculating internal doses to a target organ in an animal from any exposure scenario and then estimating what human exposure would result in this same internal dose (i.e., the HEC). A formal DAF is not calculated in this process; rather, the model itself serves as a DAF in estimating HECs. However, constructing a PBPK model is an information-intensive process, requiring much chemical-specific data. Consequently, these models are usually available for only a subset of chemicals. For example, EPA's IRIS toxicity assessment for vinyl chloride relies on a PBPK model.

12.3.3 Extrapolation from POD to Derive Carcinogenic Potency Estimates

Observable cancer rates in laboratory or human occupational epidemiologic studies tend to be several orders of magnitude higher than cancer risk levels that society is willing to tolerate from involuntary chemical exposures. To obtain observable results, laboratory studies need to be

conducted at exposures usually well above environmentally relevant concentrations. Thus, extrapolation from the POD-HEC to lower doses is usually necessary. This extrapolation is performed consistent with the mode of action, if adequately supported. Where the mode of action supports a biologically-based model and the data set is not rich enough to support a biologically based model, a non-linear reference concentration approach is employed (see Section 12.4.2). When the data are insufficient to support a mode of action decision, or where the data support a linear mode of action, a linear extrapolation is employed.

For linear extrapolation, a straight line is drawn from the point of departure expressed as a human equivalent dose to the origin (i.e., zero incremental dose, zero incremental response) to give an incremental probability dose unit. That is, the slope of the line expresses extra risk per dose unit (e.g., the IUR, expressed as extra risk per $\mu g/m^3$ of lifetime exposure). EPA's 1999 proposed guidelines[4] for carcinogen risk assessment recommend the use of the lowest effective dose using a 10 percent response level (LED_{10}) (as estimated by the lower one-sided confidence limit on the benchmark concentration [or $BMCL_{10}$]) as the POD for linear extrapolation. This approach is to draw a straight line between the estimated point of departure, generally, as a default, the LED_{10}. The LED_{10} is the lower 95 percent limit on a dose that is estimated to cause a 10 percent response. The linear extrapolation approach to assessing risk is considered generally conservative of public health, including sensitive subpopulations, in the absence of specific information about the extent of human variability in sensitivity to effects.

The inhalation cancer dose-response value derived by linear extrapolation is the IUR. It is presented as an upper-bound estimate of the excess cancer risk resulting from a lifetime (assumed 70-year) of continuous exposure to an agent at a concentration of 1 $\mu g/m^3$ in air. As illustrated previously in Exhibit 12-2(A), risk is the product of the slope and the estimated exposure. The IUR is a plausible upper-bound estimate of the risk (i.e., the risk is not likely to be higher but may be lower and may be zero). When adequate human epidemiology data are available, maximum likelihood estimates may be used instead of upper bounds to generate the IUR. When only animal data are available and linear extrapolation is used, the IUR is derived from the largest linear slope that is consistent with the data (within the upper 95 percent confidence limit). In other words, the true risk to humans, while not identifiable, is not likely to exceed the upper-bound estimate (the IUR), and is likely to be lower. This means that any estimate of risk for air toxics using an IUR is likely to be protective of all potentially exposed populations. In addition, this means that air toxics risk estimates are likely to be conservative, that is, protective of public health.

Risk = EC × IUR, where

EC	=	lifetime estimate of continuous inhalation exposure to an individual air toxic
IUR	=	the corresponding inhalation unit risk estimate for that air toxic

The evidence for the carcinogenic mode of action may lead to a conclusion that the dose-response relationship is nonlinear, with response falling much more quickly than linearly with dose, or may be most influenced by individual differences in sensitivity. In some cases this may be due to the mode of carcinogenic action being a secondary effect of toxicity or of an induced physiological change that is itself a threshold phenomenon. EPA does not generally try to distinguish between modes of action that might imply a "true threshold" from those with a nonlinear dose-response relationship. Except in unusual cases where extensive information is available, it is not possible to distinguish between

these empirically. Therefore, as a matter of science policy, nonlinear probability functions are only fitted to the response data to extrapolate quantitative low-dose risk estimates when the carcinogenic mechanism of the toxicant is very well-understood. When the evidence indicates a non-linear dose response function containing a significant change in slope, and alternate nonlinear approach may be considered. For example, when carcinogenesis can be shown to be a secondary effect of threshold toxicity, the EPA draft carcinogen guidelines recommend derivation of a reference concentration.

12.4 Dose-Response Assessment for Derivation of a Reference Concentration

The reference concentration is defined as an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive sub-populations) that is likely to be without an appreciable risk of deleterious effects during a lifetime. The RfC is expressed as a chronic exposure level to the chemical in ambient air (in units of milligrams of the substance per cubic meter of air, or mg/m^3). This value is usually derived for use with effects other than cancer. But when a chemical's carcinogenicity has been shown to be associated with a nonlinear mode of action (see Agency's Cancer Guidelines),[4] a reference concentration may be derived for use with all effects, including cancer.

Inherent in the derivation of a reference concentration is the recognition of an exposure level likely to be without an appreciable risk of adverse effects (e.g., a sub-threshold level for adverse effects). The objective of this type of dose-response assessment, then, is to estimate that exposure level for humans. The RfC is derived after a thorough review of the health effects database for an individual chemical and identification of the most sensitive and relevant endpoint (the "critical effect") along with the principal study(ies) demonstrating that endpoint. In addition to an analysis of the study data available for the chemical, risk assessors also use uncertainty factors to account for differences in sensitivity between humans and laboratory animals, the possibility of heightened sensitivity of some population groups (e.g., people with respiratory disease, very young children, the aged), and any limitations of the database. The methodology for derivation of an inhalation reference concentration is described in detail in EPA's *Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry*.[5]

The first part of this type of assessment, which involves a careful qualitative and quantitative analysis of the study data, parallels that performed for linear cancer dose-response assessment (i.e., derivation of the point of departure in terms of a human equivalent concentration [POD_{HEC}]). The qualitative analysis is described in Section 12.2.2, while the quantitative analysis is described in Sections 12.3.1 and 12.3.2. The latter part of this type of assessment involves the application of uncertainty factors to address limitations of the data used (e.g., the factors raised above).

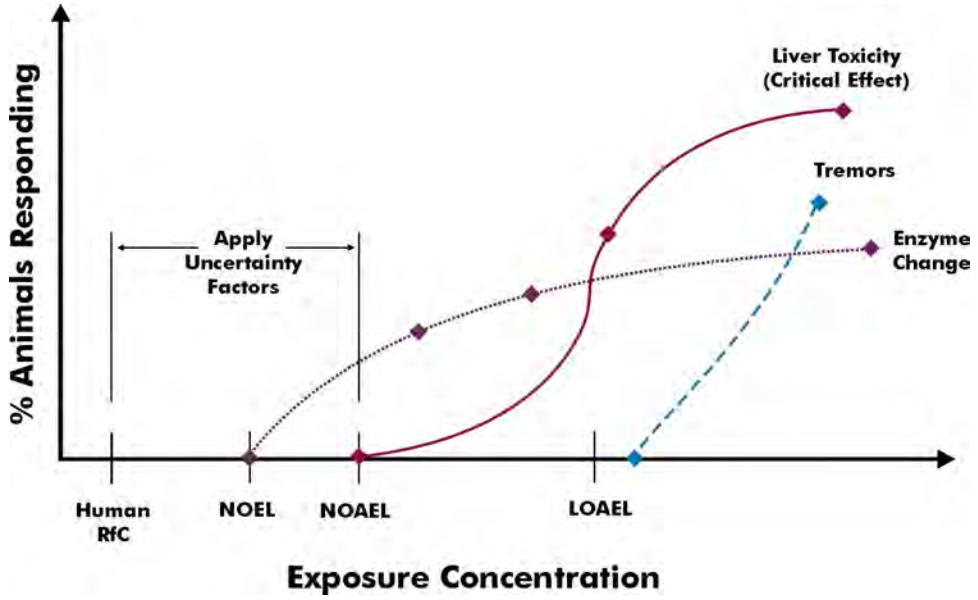
In IRIS, EPA includes with each RfC a statement of high, medium, or low confidence based on the completeness of the database for that substance. High confidence RfCs are considered less likely to change substantially with the collection of additional information, while low confidence RfCs may be especially vulnerable to change.[5]

12.4.1 Determination of the Point of Departure and Human Equivalent Concentration

In earlier sections (Section 12.2.2, 12.3.1 and 12.3.2) the analysis of the database and identification of the critical effect, as well as the derivation of the POD in terms of human equivalent concentrations are discussed.

In developing a dose-response assessment, toxicologists evaluate the available data for a substance. Studies of high quality are selected, and the assessment is focused on the most appropriate studies. As the RfC is a chronic value, preference is given to long-term studies over short-term ones, to studies using animals that exhibit effects similar to those experienced by humans, to studies using an appropriate exposure route (e.g., inhalation exposure for developing an RfC), and to studies showing a clear pattern of increasing frequency or severity of response with increasing dose. Toxicologists use the information to identify the **critical effect** (i.e., the adverse effect that appears at the lowest dose). Afterwards, appropriate human data are chosen as the basis for the RfC or, if human data are not adequate, data from the most appropriate species are identified. If this is not known, the data from the most sensitive species is usually chosen. This analysis is described in Section 12.2.2. The objective in identifying the critical effect or effects is to identify the effect(s) - among all those associated with exposure to the chemical of interest - that occur at the lowest exposure and would lead to derivation of the lowest RfC (Exhibit 12-10).

Exhibit 12-10. Overview to Develop a Reference Concentration



The LOAEL (HEC) and NOAEL (HEC) are illustrated with low-dose extrapolation with the application of uncertainty or modifying factors to derive the human health-protective RfC. Note that this figure represents data from appropriate animal species.

Using the dose-response relationship for the critical effect, toxicologists identify the POD from the experimental data. This exposure concentration (in terms of its human equivalent) which marks the boundary between the range of observation and that of extrapolation, is the point from

which extrapolation begins for derivation of a RfC. The POD may be derived from benchmark modeling (see Section 12.3.1 regarding the derivation of a BMCL). If the data do not meet requirements for benchmark modeling, the POD is derived by the use of a statistical analysis to identify the **no-observed-adverse-effect-level**, or **NOAEL**, defined as the highest dose level administered to laboratory animals that did not cause statistically or biologically significant observable adverse effects after chronic (usually lifetime) exposure in the studied population. In some cases, a LOAEL is used in the absence of a NOAEL. In either case, the POD is transformed into a continuous inhalation exposure (e.g., from an intermittent animal exposure, 6 hours/day, 5 days/week) and then into a human equivalent concentration (as described in Section 12.3.2). In order for the appropriate critical effect to be identified, a comparison of PODs across different endpoints is done in terms of human equivalent concentrations (or potential RfC values, which incorporate the application of UFs, need to be compared).[5]

Derivation of RfC Using BMC Methodology – 1,3-dichloropropene

A review of the available animal studies indicated changes to the surface cells of the nasal portion of the respiratory tract as the critical effect for 1,3-dichloropropene. Benchmark modeling was performed on the data demonstrating this effect. The seven statistical models for dichotomous data from the Agency's benchmark dose modeling software (BMDS Version.1b) were applied to the incidence data for the adjusted administered doses. The best model fit was determined by eliminating all models that did not have a statistically significant goodness-of-fit ($p<0.05$). The remaining models were then ranked by best visual fit of the data, especially for the lower doses, as observed in the graphical output of the Benchmark Dose Software. The model with statistically significant goodness-of-fit and best visual and statistical fit was used to estimate the BMC at 10 percent risk and the 95 percent lower confidence limit of the BMC (the BMCL). The gamma, logistic, multistage, Weibull, and quantal-quadratic models provided statistically significant fits. The gamma model was the best fit overall because it provided the best visual fit. This model yielded a BMC_{10} of 5.9 mg/m^3 and a $BMCL_{10}$ of 3.7 mg/m^3.

The $BMCL_{10}$ was identified as the POD and was adjusted from experimental conditions to a continuous inhalation exposure value (POD_{adj}). Because the critical target was the nasal mucosa, algorithms for extrathoracic effects for Category 1 gases were used to adjust continuous animal exposure concentration to HEC. The POD_{HEC} for a Category 1 gas was derived by multiplying the animal $BMCL_{10}$ by an interspecies dosimetric adjustment for gas:respiratory effects in the extrathoracic area of the respiratory tract. Using default values, the adjustment factor was equal to 0.2. For example, for 1,3-dichloropropene:

$$POD_{HEC} = BMCL_{10}(HEC) = BMCL_{10}\ (adj) \times 0.2 = 3.7 \times 0.2 = 0.7\ mg/m^3$$

The POD_{HEC} was divided by uncertainty factors for interspecies extrapolation (UF of 3) and intraspecies variation (UF of 10) and rounded to one significant figure to yield the RfC for 1,3-dichloropropene:

$$RfC = POD_{HEC} / 30 = 0.02\ mg/m^3$$

12.4.2 Application of Uncertainty Factors

The RfC is an estimate derived from the POD_{HEC} for the critical effect (based on either a $BMCL_{HEC}$, $NOAEL_{HEC}$ or $LOAEL_{HEC}$) by consistent application of UFs. The UFs are applied to account for recognized uncertainties in the use of the available data to estimate an exposure concentration appropriate to the assumed human scenario. The general formula for deriving an RfC from a POD_{HEC} is:

$$RfC\,(mg/m^3) = \frac{POD_{HEC}(mg/m^3)}{UF} \qquad \text{(Equation 12-2)}$$

A UF of 10, 3, or 1 is applied for each of the following extrapolations used to derive the RfC (see Exhibit 12-11):

- Animal to human;
- Human to sensitive human populations;
- Subchronic to chronic;
- LOAEL to NOAEL; and
- Incomplete to complete database.

The UFs are generally an order of magnitude (10), although incorporation of dosimetry adjustments or other information may result in the use of reduced UFs for RfCs (3 or 1). The composite UF applied to an RfC will vary in magnitude depending on the number of uncertainties involved; however, an RfC will not be derived when use of the data involves more than four areas of extrapolation. The composite UF when four factors are used generally is reduced from 10,000 to 3,000 in recognition of the lack of independence and the conservatism of these factors.

The 2002 Agency review of the reference dose (RfD)/reference concentration process[(2)] encouraged the development of guidance in the area of chemical-specific adjustment factors (CSAFs). These factors utilize specific data to replace the default UFs for interspecies or inter-individual variation. The review panel noted, however, that the CSAF approach for any single substance is determined principally by the availability of relevant data. For many substances there are relatively few data available to serve as an adequate basis to replace defaults for interspecies differences and human variability with more informative CSAFs.

Because of this procedure to address the lack of information on the translation from experimental data to a human scenario, the resulting RfC for many HAPs is on the order of 100 to 300 times lower than the NOAEL actually observed in the animal testing (see Exhibit 12-12). This reflects the lowering of the RfC to address the uncertainties in the extrapolations mentioned above. For those HAPs that have had their effects well documented in human studies, the RfC may be much closer to the highest concentration at which an adverse effect was not observed (e.g., within a factor of 3 to 10).

Exhibit 12-11. Uncertainty Factors Used in the Derivation of an Inhalation RfC	
Standard Uncertainty Factors	**Processes Considered in UF Purview**
A = Animal to human Extrapolation from valid results of long-term studies on laboratory animals when results of studies of human exposure are not available or are inadequate. Intended to account for the uncertainty in extrapolating laboratory animal data to the case of average healthy humans.	• Pharmacokinetics/Pharmacodynamics • Relevance of laboratory animal model • Species sensitivity
H = Human to sensitive human Extrapolation of valid experimental results for studies using prolonged exposure to average healthy humans. Intended to account for the variation in sensitivity among the members of the human population.	• Pharmacokinetics/Pharmacodynamics • Sensitivity • Differences in mass (children, obese) • Concomitant exposures • Activity Pattern • Does not account for idiosyncrasies
S = Subchronic to chronic Extrapolation from less than chronic exposure results on laboratory animals or humans when there are no useful long-term human data. Intended to account for the uncertainty in extrapolating from less than chronic NOAELs to chronic NOAELs.	• Accumulation/Cumulative damage • Pharmacokinetics/Pharmacodynamics • Severity of effect • Recovery • Duration of study • Consistency of effect with duration
L = LOAEL to NOAEL Derivation from a LOAEL instead of a NOAEL. Intended to account for the uncertainty in extrapolating from LOAELs to NOAELs.	• Severity • Pharmacokinetics/Pharmacodynamics • Slope of dose-response curve • Trend, consistency of effect • Relationship of endpoints • Functional vs histopathological evidence • Exposure uncertainties
D = Incomplete to complete data Extrapolation from valid results in laboratory animals when the data are "incomplete". Intended to account for the inability of any single laboratory animal study to adequately address all possible adverse outcomes in humans.	• Quality of critical study • Data gaps • Power of critical study/supporting studies • Exposure uncertainties
Source: U.S. EPA. 1994. *Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry*.[5]	

Exhibit 12-12. Examples of the Use of Uncertainty Factors in Deriving RfCs	
RfC from NOAEL **Example: Diesel Engine Emissions**	**RfC from LOAEL** **Example: Toluene**
Toxicity data: 144 µg chemical/m^3 air ($NOAEL_{HEC}$ from chronic rodent study) *Uncertainty factors*: 3 x 10 = 30 3 = animal-to-human extrapolation 10 = human to sensitive human subpopulations RfC = 144/30 = 4.8 µg/m^3 = 0.005 mg/m^3	*Toxicity data:* 119 mg chemical/m^3 air ($LOAEL_{HEC}$ from chronic occupational study) *Uncertainty factors*: 10 x 10 x 3 = 300 10 = human to sensitive human subpopulations 10 = LOAEL-to-NOAEL extrapolation 3 = database deficiencies RfC = 119/300 mg/m^3 = 0.4 mg/m^3
$NOAEL_{HEC}$ = No-Observed-Adverse-Effect Level (Human Equivalent Concentration) $LOAEL_{HEC}$ = Lowest-Observed-Adverse-Effect Level (Human Equivalent Concentration) *Source*: EPA's IRIS database http://www.epa.gov/IRIS/.	

In some of the older IRIS assessments a "modifying factor" may have been applied in addition to the traditional uncertainty factors. It had been used with professional judgement when it was determined that another uncertainty factor was needed; its magnitude depended upon the professional assessment of scientific uncertainties of the study and database not explicitly treated via the other uncertainty factors.[5] The 2002 Agency review of the RfD/RfC process, however, recommended against continued use of the modifying factor. It was felt that the traditional factors could account for any remaining uncertainties.[2]

12.5 Sources of Chronic Dose-Response Values

Appendix C provides a current listing of appropriate chronic dose-response values (i.e., RfCs or comparable values and IURs) for HAPs.[f] References for acute exposure levels are provided below in Exhibit 12-13. Hazard identification and dose-response assessment information for chronic exposure, presented in Appendix C, was obtained from various sources and prioritized according to (1) conceptual consistency with EPA risk assessment guidelines, and (2) level of review received. The prioritization process was aimed at incorporating into our assessments the best available science with respect to dose-response information. The sources listed below were used, and provide this information for chemicals beyond the 188 Clean Air Act hazardous air pollutants listed in Appendix C.

- **U.S. Environmental Protection Agency (EPA)**. EPA has developed dose-response assessments for chronic exposure to many pollutants. These assessments typically specify an RfC (to protect against effects other than cancer) and/or IUR (to estimate the probability of

[f] As noted earlier, see http://www.epa.gov/ttn/atw/toxsource/summary.html for a current listing of this information.

contracting cancer). Background documents, particularly for the more recent files, also contain information on physical and chemical properties, toxicokinetics, and hazard characterization. EPA disseminates dose-response assessment information in several forms, based on the level of review. Dose-response assessments that have achieved full intra-agency consensus are incorporated in the **Integrated Risk Information System (IRIS)**, which is regularly updated and available on-line (www.epa.gov/iris). All IRIS assessments since 1996 also have undergone independent external peer review. In the past, dose-response assessments for some substances were prepared by the EPA Office of Research and Development, but were never submitted for EPA consensus. EPA has assembled the results of many such assessments in the **Health Effects Assessment Summary Tables (HEAST)**. Although the values in HEAST have undergone some review and have the concurrence of individual Agency program offices, they have not had enough review to be recognized as Agency-wide consensus information. In addition, since HEAST has not been updated since 1997, other sources described here are, for many chemicals, more reliable.

- **Agency for Toxic Substances and Disease Registry (ATSDR)**. ATSDR, which is part of the US Department of Health and Human Services, develops and publishes Minimum Risk Levels (MRLs) for many toxic substances. The MRL is defined as an estimate of daily human exposure to a substance that is likely to be without an appreciable risk of adverse effects (other than cancer) over a specified duration of exposure. MRLs are derived for acute (1-14 days), intermediate (>14-364 days), and chronic (365 days and longer) exposures by inhalation and oral routes. ATSDR describes MRLs as substance-specific estimates to be used by health assessors to select environmental contaminants for further evaluation. MRLs are presented with only one significant figure and are considered to be levels below which contaminants are unlikely to pose a health threat. Exposures above an MRL do not necessarily represent a threat, and MRLs are therefore not intended for use as predictors of adverse health effects or for setting cleanup levels. The MRL data undergo a rigorous review process, including internal ATSDR review, peer reviews, and public comment periods. Appendix C shows the ATSDR chronic MRL where no IRIS value is available, because the MRL's concept, definition, and derivation are philosophically consistent (though not identical) with EPA's guidelines for assessing noncancer effects. ATSDR publishes MRLs as part of pollutant-specific toxicological profile documents, and also in regularly-updated on-line tables.[7]

- **California Environmental Protection Agency (CalEPA)**. The CalEPA Office of Environmental Health Hazard Assessment (OEHHA) has developed dose-response assessments for many substances, based both on carcinogenicity and health effects other than cancer. The process for developing these assessments is similar to that used by EPA to develop IRIS values and includes significant external scientific peer review. The non-cancer information includes inhalation health risk guidance values expressed as chronic inhalation reference exposure levels (RELs). CalEPA defines the REL as a concentration level at (or below) which no health effects are anticipated, a concept that is substantially similar to EPA’s approach to non-cancer dose-response assessment. Appendix C shows the chronic REL (including both final and proposed values) where no IRIS RfC/RfD or ATSDR MRL exists. CalEPA’s quantitative dose-response information on carcinogenicity by inhalation exposure is expressed in terms of the IUR, defined similarly to EPA’s IUR. Appendix C shows specific CalEPA UREs where no IRIS values exist. CalEPA’s dose response assessments for carcinogens and noncarcinogens are available on-line.[8]

- **International Agency for Research on Cancer** (**IARC**). The IARC, a branch of the World Health Organization, coordinates and conducts research on the causes of human cancer and develops scientific strategies for cancer control. The IARC sponsors both epidemiological and laboratory research, and disseminates scientific information through meetings, publications, courses and fellowships. As part of its mission, the IARC assembles evidence that substances cause cancer in humans and issues judgments on the strength of evidence. IARC's categories are Group 1 (carcinogenic in humans), Group 2A (probably carcinogenic), Group 2B (possibly carcinogenic), Group 3 (not classifiable), and Group 4 (probably not carcinogenic). The categorization scheme may be applied to either single chemicals or mixtures; however, IARC does not develop quantitative dose-response metrics such as UREs,. IARC's categories for substances are included in Appendix C to support or augment EPA's weight-of evidence (WOE) determinations, which do not cover all substances and in some cases may be out-of-date. The list of IARC evaluations to date is available on-line (http://193.51.164.11/monoeval/grlist.html).

Additionally, the EPA has compiled fact sheets for the 188 CAA hazardous air pollutants and makes them available on the Air Toxics website (http://www.epa.gov/ttn/atw/hapindex.html). This collection is called the **Health Effects Notebook for Hazardous Air Pollutants**, and provides for each HAP a summary of available information in the following categories: hazard summary, physical properties, uses, sources and potential exposure, and health hazard information. These fact sheets are useful for describing hazards associated with the 188 HAPs.

12.6 Acute Exposure Reference Values

Many air pollutants can cause adverse health effects after acute or short-term exposures lasting from a few minutes to several days. For some pollutants, acute exposures may be of greater concern than chronic exposures. The severity of effects from acute exposures may vary widely. Agency-wide guidance on how to assess toxic effects from short-term exposures is currently being developed. This guidance for Acute Reference Exposure (ARE) levels is intended to assist acute risk assessment activities. A variety of other short-term, acute exposure limits are also described in Exhibit 12-12.[9] Appendix C provides a current listing of acute dose-response values for HAPs.

Methods for dose-response assessment of acute exposures are usually similar to the approach for chronic exposure, with their derivation involving the identification of a "critical effect," determination of a NOAEL or comparable value for that effect, and application of uncertainty factors (e.g., animal to human population). However, the process by which most acute inhalation dose-response assessment values are derived differs from the chronic RfC methodology in two important ways. First, "acute" may connote exposure times varying from a few minutes to two weeks. The time frame for the value is critical, because the safe dose (or the dose that produces some defined effect) may vary substantially with the length of exposure. Second, some acute dose-response assessments include more than one level of severity. A typical assessment may have values for level 1 (at which only mild, transient effects may occur), level 2 (above which irreversible or other serious effects may occur), and level 3 (above which life-threatening effects may occur). Therefore, many acute assessments present dose-response assessment values as a matrix, with one dimension being length of exposure and the other a severity-of-effect category.

Exhibit 12-13. Examples of Available Short-Term, Acute Exposure Levels				
Acronym	**Full Name**	**Group or Agency**	**Purpose/Definition**	**Source/Website**
AEGL	Acute Exposure Guideline Level	National Research Council (NRC) National Advisory Committee (NAC)	The AEGLs represent short-term threshold or ceiling exposure values intended for the protection of the general public, including susceptible or sensitive individuals, but not hypersusceptible or hypersensitive individuals. The AEGLs represent biological reference values for this defined human population and consist of three biological endpoints for four different single emergency (accidental) exposure periods (30 minutes, l hour, 4 hours, and 8 hours). In some instances, AEGLs also are developed for 5 or 10 minutes. The biological endpoints are defined as follows: • AEGL-1 is the airborne concentration (expressed as parts per millions [ppm] or milligrams [mg]/meters [m]^3) of a substance at or above which it is predicted that the general population, including "susceptible" but excluding "hypersusceptible" individuals, could experience notable discomfort. Airborne concentrations below AEGL-1 represent exposure levels that could produce mild odor, taste, or other sensory irritations. • AEGL-2 is the airborne concentration (expressed as ppm or mg/m^3) of a substance at or above which it is predicted that the general population, including "susceptible" but excluding "hypersusceptible" individuals, could experience irreversible or other serious, long-lasting effects or impaired ability to escape. Airborne concentrations below the AEGL-2 but at or above AEGL-1 represent exposure levels that may cause notable discomfort. • AEGL-3 is the airborne concentration (expressed as ppm or mg/m^3) of a substance at or above which it is predicted that the general population, including "susceptible" but excluding "hypersusceptible" individuals, could experience life-threatening effects or death. Airborne concentrations below AEGL-3 but at or above AEGL-2 represent exposure levels that may cause irreversible or other serious, long-lasting effects or impaired ability to escape.	http://search.nap.edu/books/0309072948/html/
ARE	Acute Reference Exposure	U.S. Environmental Protection Agency	The ARE is an informed estimate of the highest inhalation exposure (concentration and duration) that is not likely to cause adverse effects in a human population, including sensitive subgroups, exposed to that scenario, even on an intermittent basis.[10] For these purposes, acute exposures are single continuous exposures lasting 24 hours or less; AREs may be derived for any duration of interest within that period. "Intermittent" implies sufficient time between exposures such that one exposure has no effect on the health outcome produced by the next exposure. EPA is in the process of finalizing the methodology for development of AREs.	

Exhibit 12-13. Examples of Available Short-Term, Acute Exposure Levels				
Acronym	**Full Name**	**Group or Agency**	**Purpose/Definition**	**Source/Website**
BEI	Biological Exposure Indices	American Conference of Governmental Industrial Hygenists	BEIs® are health-based values for use by industrial hygienists in making decisions regarding safe levels of exposure to various chemical and physical agents found in the workplace.	http://www.acgih.org/TLV/
CEEL	Community Emergency Exposure Level	National Research Council (NRC) National Advisory Committee (NAC)	CEELs are ceiling exposure values for the public applicable to emergency exposures of foreseeable magnitude and duration, usually not exceeding 1 hour. Three CEELs were established: • CEEL-1: Concentration above which discomfort, for example eye and nose irritation or headaches, becomes increasingly common; • CEEL-2: Concentration above which disability, for example, severe eye or respiratory irritation, becomes increasingly common; • CEEL-3: Concentration above which death or life-threatening effects, for example, pulmonary edema, cardiac failure, or cancer, become increasingly common.	Guidelines for Developing Community Emergency Exposure Levels for Hazardous Substances (NRC, 1993)
EEGL	Emergency Exposure Guidance Level	NAS Committee on Toxicology	Exposure levels judged to be acceptable for military personnel performing tasks during emergency situations. Not considered safe exposure level for routine or normal operations.	
ERPG	Emergency Response Planning Guideline	American Industrial Hygiene Association's (AIHA) Emergency Response Planning Committee	These guidelines are intended for application by persons trained in emergency response planning. ERG-1: The maximum concentration in air below which it is believed nearly all individuals could be exposed for up to one hour without experiencing other than mild transient adverse health effects or perceiving a clearly defined objectionable odor. ERG-2: The maximum concentration in air below which it is believed nearly all individuals could be exposed for up to one hour without experiencing or developing irreversible or other serious health effects or symptoms that could impair their abilities to take protective action. ERG-3: The maximum concentration in air below which it is believed nearly all individuals could be exposed for up to one hour without experiencing or developing life-threatening health effects.	http://www.bnl.gov/scapa/erpgpref.htm http://www.bnl.gov/scapa/scapawl.htm
IDLH	Immediately Dangerous to Life or Health Concentration	National Institute for Occupational Safety and Health (NIOSH)	An immediately dangerous to life or health condition is one "that poses a threat of exposure to airborne contaminants when that exposure is likely to cause death or immediate or delayed permanent adverse health effects or prevent escape from such an environment. The purpose of establishing an IDLH is to ensure that the worker can escape from a given contaminated environment in the event of failure of the respiratory protection equipment.	NIOSH Respirator Decision Logic [NIOSH 1987], http://www.cdc.gov/niosh/idlh/intridl4.html

Exhibit 12-13. Examples of Available Short-Term, Acute Exposure Levels				
Acronym	**Full Name**	**Group or Agency**	**Purpose/Definition**	**Source/Website**
LOC	Level of Concern	U.S. Environmental Protection Agency, Federal Emergency Management Agency, U.S. Department of Transportation	Defined by the Technical Guidance for Hazards Analysis (a guide developed to assist in planning for accidental chemical releases). As the concentration of an extremely hazardous substances in air above which there may be serious irreversible health effects or death as a result of a single exposure for a relatively short period of time. In the 1987 Technical Guidance for Hazards Analysis document, an LOC was estimated by using one-tenth of the IDLH level published by the National Institute for Occupational Safety and Health. For the purposes of offsite consequence analysis performed as part of accidental release requirements under Section 112(r) of the CAA, this value is superceded by ERPG-2 values as available, and the Agency intends to supercede those values with AEGL-2 values as they are developed and adopted.	Technical Guidance for Hazards Analysis. Emergency Planning for Extremely Hazardous Substances. (USEPA, FEMA, USDOT, 1987). 61 FR 31672; June 20, 1996
MRL	Acute Minimum Risk Levels	U.S. Agency for Toxic Substances and Disease Registry (ATSDR)	The MRL is an estimate of human exposure to a substance that is likely to be without an appreciable risk of adverse effects (other than cancer) over a specified duration of exposure, and can be derived for acute exposures by the inhalation and oral routes. Unlike the one-hour focus of most of the other values listed here, acute MRLs are derived for exposures of 1 to 14 days duration.	http://www.atsdr.cdc.gov/mrls.html
REL	Reference Exposure Level	California EPA Office of Environmental Health Hazard Assessment (OEHHA)	The acute REL is an exposure that is not likely to cause adverse effects in a human population, including sensitive sub-populations, exposed to that concentration for one hour on an intermittent basis. RELs are based on the most sensitive, relevant, adverse health effect reported in the medical and toxicological literature. RELs are designed to protect the most sensitive individuals in the population by the inclusion of margins of safety. Since margins of safety are incorporated to address data gaps and uncertainties, exceeding the REL does not automatically indicate an adverse health impact OEHHA has defined the lowest available acute severity level as the REL.	http://www.oehha.ca.gov/air/pdf/acuterel.pdf
SPEGL	Short-term Public Emergency Exposure Guidance Level	National Academy of Sciences (NAS) Committee on Toxicology	The NAS develops short-term public emergency exposure guidance levels (SPEGLs) to apply to the exposures of the general public to contaminants during airborne chemical releases; SPEGLs are generally set at a level of 0.1 to 0.5 times the EEGL and are measured as 60 minute or 8 hour exposure time frames.	*Criteria and Methods for Preparing Emergency Exposure Guidance Level (EEGL), Short-Term Public Emergency Guidance Level (SPEGL), and Continuous Exposure Guidance Level (CEGL) Documents.* 1986. National Academy Press, National Academy of Sciences ,Washington, D.C.

Exhibit 12-13. Examples of Available Short-Term, Acute Exposure Levels				
Acronym	**Full Name**	**Group or Agency**	**Purpose/Definition**	**Source/Website**
STEL	Short-Term Exposure Limit	American Conference of Governmental Industrial Hygenists (ACGIH)	STELs are time weighted average (TWA) guidelines for the control of short term exposure in the workplace. These are important supplements to the eight-hour TWA exposure standards which are more concerned with the total intake over long periods of time. Generally, STELs are established to minimize the risk of the occurrence in nearly all workers of: intolerable irritation; chronic or irreversible tissue change; and narcosis to an extent that could precipitate industrial accidents, provided the eight hour TWA exposure standards are not exceeded. STELs are recommended for those substances only when there is evidence either from human or animal studies that adverse health effects can be caused by high short term exposure. STELs are expressed as airborne concentrations of substances, averaged over a period of 15 minutes.	

12.7 Evaluating Chemicals Lacking Health Reference Values

12.7.1 Use of Available Data Sources

If EPA-derived IRIS assessments are available for the chemicals being examined, these values should generally be used in the risk assessment. Use of IRIS or other EPA-derived dose-response values prevents duplication of effort in toxicity assessment and ensures consistency in the dose-response values among risk assessments. If EPA-derived dose-response values are not available, the other sources described in Section 12.6 should be given next priority. Use of these sources in a hierarchical manner has been implemented in tables developed for the 188 hazardous air pollutants (see Appendix C and http://www.epa.gov/ttn/atw/toxsource/table1.pdf). The Toxicology Excellence for Risk Assessment (TERA) maintains a database of international dose-response values (see www.TERA.org/iter).

If those sources also lack inhalation dose-response values, then route-to-route extrapolation (discussed below) may be considered. This approach, however, may be quite detailed, and requires assistance from a professional toxicologist. If all sources and approaches have been researched, and no dose-response value is available, the assessor should describe the effects of the chemical qualitatively and discuss the implications of the absence of the chemical from the risk estimate in the uncertainty section of the risk assessment.

12.7.2 Route-to-Route Extrapolation

For cases in which appropriate dose-response values are not available for the route of exposure being considered, but are available for another route, it may be possible to use route-to-route extrapolation. Route-to-route extrapolation is recommended only from oral to inhaled exposure and only for carcinogens. The ability to perform quantitative route-to-route extrapolation is critically dependent on the amount and type of data available. Regardless of the toxic endpoint being considered, a minimum of information is required to construct plausible dosimetry for the routes of interest. This information includes both the nature of the toxic effect and a description of the relationship between exposure and the toxic effect.

Route to route extrapolations should only be done by qualified toxicologists.

Data from other routes of exposure may be useful to derive an RfC (for carcinogens only; discussed below) only when respiratory tract effects and/or "first pass" effects can be ruled out. First pass effects are cases where metabolism takes place in the portal-of-entry tissues, prior to entry into the systemic circulation. The respiratory tract can exhibit a first-pass effect after inhalation. Unless the first-pass effect and dosimetry are adequately understood, there can be substantial error introduced in route-to-route extrapolation that does not account for these considerations.

Oral toxicity data should *not* be used for route-to-route extrapolation in the following cases (unless these effects can be accounted for in a PBPK model):

- When groups of chemicals have different toxicity by the two different routes (e.g., metals, irritants, and sensitizers);
- When a first-pass effect by the respiratory tract is expected;

- When a first-pass effect by the liver is expected;

- When a respiratory tract effect is established, but dosimetry comparison cannot be clearly established between the two routes;

- When the respiratory tract is not adequately studied in the oral studies; and

- When short-term inhalation studies, dermal irritation, in vitro studies, or characteristics of the chemical indicate potential for portal-of-entry effects at the respiratory tract, but studies themselves are not adequate for an RfC development.

The actual impact of exposure by different routes can only be estimated by taking account of factors that influence absorption at the portal of entry, such as (1) physicochemical characteristics of the chemical; (2) exposure factors; and (3) physiologic parameters. The preferred method for performing route-to-route extrapolation involves the development of a PBPK model that describes the disposition of the chemical for the routes of interest. As previously discussed, PBPK models account for fundamental physiologic and biochemical parameters and processes such as blood flow, ventilatory parameters, metabolic capacities, and renal clearance, tailored by the physicochemical and biochemical properties.

If appropriate toxicity information is not available, a qualitative rather than quantitative evaluation of the chemical is recommended. The implications of the absence of the chemical from the risk estimate should be discussed in the uncertainty section.

12.8 Dose-Response Assessment for Mixtures

The recommended approach for assessing risks from exposure to a mixture of pollutants (e.g., coke oven emissions, diesel exhaust, etc.) is to utilize a dose-response assessment developed for that mixture or a mixture judged similar.[11 12] Where such an assessment is not available, a component-by-component approach may be employed. There are several commonly used approaches. Selection among the approaches involves consideration of the similarity of the mixture components with regard to their toxicological activity. There are a few groups of toxicologically similar chemicals for which the Agency recommends the use of relative potency factors (RPFs) or toxicity equivalence factors (TEFs). These factors have been developed by EPA and other organizations for two classes of compounds: PAHs and dioxins/furans. The World Health Organization (WHO) has developed TEFs for polychlorinated biphenyls (PCBs) as an extension of the factors for dioxins/furans (see Exhibit 12-14).

- **Polycyclic Aromatic Hydrocarbons (PAHs)**. EPA has not developed IURs or CSFs for carcinogenic PAHs other than benzo(a)pyrene. EPA recommends use of a RPF based on the potency of each compound relative to that of benzo(a)pyrene.[13] Although several references may be found in the literature with proposed RPFs for PAHs, EPA recommends the following RPF values for seven PAHs, which are classified as B2, probable human carcinogens:[(g)]

[g]CalEPA has developed IURs based on RPFs for several additional PAHs that have been classified as probably or possibly human carcinogens (e.g., IARC).

PAH	RPF
Benzo(a)pyrene	1.0
Benzo(a)anthracene	0.1
Benzo(b)fluoranthene	0.1
Benzo(k)fluoranthene	0.01
Chrysene	0.001
Dibenz(a,h)anthracene	1.0
Indeno(1,2,3-c,d)pyrene	0.1

Thus, for these seven PAHs, the IUR for benzo(a)pyrene is multiplied by the applicable RPF to derive the IUR.

- **Dioxins, Furans, and PCBs**. For carcinogenic dioxins and furans, the TEF approach has an underlying assumption of additivity across mixture components. EPA currently recommends TEFs for specific congeners, rather than isomeric groups (see Exhibit 12-13). TEFs were determined by inspection of the available congener-specific data and an assignment of an "order of magnitude" estimate of relative toxicity when compared to 2,3,7,8-TCDD. The cancer potency of certain dioxin and furan congeners is estimated relative to 2,3,7,8-TCDD based on other toxicity information that is available for the congeners. Scientific judgment and expert opinion formed the basis for these TEF values. External review of the toxicity and pharmacokinetic data utilized in setting these TEF values supported the basic approach as a "reasonable estimate" of the relative toxicity of polychlorinated dibenzo-dioxins (PCDDs) and polychlorinated dibenzo-furans (PCDFs).[14] TEF values developed by scientific groups over the past 15 years are provided in Exhibit 12-13. The most recent consensus of the scientific community (including representation by EPA scientists) is represented by the WHO 1997 values.

  TEFs based on the relative cancer potencies are used to adjust the exposure concentrations of mixture components, which are subsequently summed into a single exposure concentration for the mixture. That exposure concentration based on TEFs is then used, along with the 2,3,7,8-TCDD IUR or noncancer reference value, to estimate cancer risks or other health hazards for the mixture.

Exhibit 12-14. Toxicity Equivalence Factors for Dioxins, Furans and PCBs				
Congener	**EPA (1987)**[15]	**NATO (1989)**[16]	**WHO (1994)**[17]	**WHO (1997)**[18]
TCDDs				
2,3,7,8-TCDD	1	1		1
1,2,3,7,8-PeCDD	0.5	0.5		1
1,2,3,4,5,8-HxCDD	0.04	0.1		0.1
1,2,3,7,8,9-HxCDD	0.04	0.1		0.1
1,2,3,6,7,8-HxCDD	0.04	0.1		0.1
1,2,3,4,6,7,8-HpCDD	0.001	0.1		0.01
1,2,3,4,6,7,8,9-OCDD	0	0.001		0.0001
TCDFs				
2,3,7,8-TCDF	0.1	0.1		0.1
1,2,3,7,8-PeCDF	0.1	0.05		0.05
2,3,4,7,8-HxCDF	0.1	0.5		0.5
1,2,3,4,7,8-HxCDF	0.01	0.1		0.1
1,2,3,7,8,9-HxCDF	0.01	0.1		0.1
1,2,3,6,7,8-HxCDF	0.01	0.1		0.1
2,3,4,6,7,8-HxCDF	0.01	0.1		0.1
1,2,3,4,6,7,8-HpCDF	0.001	0.01		0.01
1,2,3,4,7,8,9-HpCDF	0.001	0.01		0.01
1,2,3,4,6,7,8,9-OCDF	0	0.001		0.0001
PCBs **IUPAC # Structure**				
77 3,3',4,4'-TCB			0.0005	0.0001
81 3,4,4',5-TCB			–	0.0001
105 2,3,3',4,4'-PeCB			0.0001	0.0001
114 2,3,4,4',5-PeCB			0.0005	0.0005
118 2,3',4,4',5-PeCB			0.0001	0.0001
123 2',3,4,4',5-PeCB			0.0001	0.0001
126 3,3',4,4',5-PeCB			0.1	0.1
156 2,3,3'4,4',5-HxCB			0.0005	0.0005
157 2,3,3',4,4',5'-HxCB			0.0005	0.0005
167 2,3',4,4',5,5'-HxCB			0.00001	0.00001
169 3,3'4,4'5,5'-HxCB			0.01	0.01
170 2,2',3,3',4,4',5-HpCB			0.0001	–
180 2,2',3,4,4',5,5'-HpCB			0.00001	–
189 2,3,3',4,4',5,5'-HpCB			0.0001	0.0001
Source: EPA's dioxin reassessment activities[19]				

References

1. U.S. Environmental Protection Agency. Air Toxics Website: Summary. Updated November 28, 2003. Available at: http://www.epa.gov/ttn/atw/toxsource/summary.html. (Last accessed March 2004).

2. U.S. Environmental Protection Agency. 2002. *A Review of the Reference Dose and Reference Concentration Process.* Risk Assessment Forum, Washington, D.C., 2002. EPA/630/P-02/002F. Available at: http://cfpub.epa.gov/ncea/raf/recordisplay.cfm?deid=55365

3. U.S. Environmental Protection Agency. 2002. Office of Research and Development, National Center for Environmental Assessment. *Risk Assessment Guidelines.* Updated June 30, 2002. Available at: http://cfpub.epa.gov/ncea/raf/rafguid.htm. (Last accessed March 2004).

4. U.S. Environmental Protection Agency. 1999. *Guidelines for Carcinogen Risk Assessment. Review Draft.* Risk Assessment Forum, Washington, D.C., July 1999. NCEA-F-0644. Available at: http://cfpub.epa.gov/ncea/raf/rafguid.htm

   U.S. Environmental Protection Agency. 1986. *Guidelines for Carcinogen Risk Assessment. Federal Register* 51(185):33992-43003, September 24, 1986. Available at: http://cfpub.epa.gov/ncea/raf/rafguid.htm

   U.S. Environmental Protection Agency. 2003. *Draft Final Guidelines for Carcinogen Risk Assessment (External Review Draft),* Risk Assessment Forum, Washington, D.C. NCEA-F-0644A. Available at http://cfpub.epa.gov/ncea/raf/rafguid.htm

5. U.S. Environmental Protection Agency. 1994. *Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry.* Washington, D.C.

   U.S. Environmental Protection Agency. 2002. *A Review of the Reference Dose and Reference Concentration Process.* Risk Assessment Forum, Washington, D.C. December 2002. EPA/630/P-02/002F. Available at: http://www.epa.gov/iris/RFD_FINAL%5B1%5D.pdf (Last accessed April 2004).

6. U.S. Environmental Protection Agency. 2002. Benchmark Dose Software (BMDS). Office of Research and Development. Updated June 30, 2002. Available at http://cfpub.epa.gov/ncea/cfm/bmds.cfm. (Last accessed March 2004).

   U.S. Environmental Protection Agency. 1996. *Benchmark Dose Technical Guidance Document.* Risk Assessment Forum, Washington, D.C., August 1996. EPA/600/P-96/002A. Available at: http://www.epa.gov/cgi-bin/claritgw?op-Display&document=clserv:ORD:0603;&rank=4&template=epa.

7. Agency for Toxic Substances and Disease Registry (ATSDR). 2004. Toxicological Profile Information Sheet. Updated March 19, 2004. Available at: http://www.atsdr.cdc.gov/toxpro2.html. (Last accessed March 2004).

Agency for Toxic Substances and Disease Registry (ATSDR). 2004. Minimal Risk Levels (MRLs) for Hazardous Substances. Updated March 22, 2004. Available at: http://www.atsdr.cdc.gov/mrls.html. (Last accessed March 2004).

8. State of California Office of Environmental Health Hazard Assessment. 2003. Air - Hot Spots Guidelines. Available at: http://www.oehha.ca.gov/air/hot_spots/index.html. (Last accessed March 2004).

9. Office of Response and Restoration, National Ocean Service, National Oceanic and Atmospheric Administration. 2002. Occupational Exposure Limits. Updated March 26, 2002. Available at: http://response.restoration.noaa.gov/cameo/locs/worklims.html. (Last accessed March 2004).

10. The ARE is described in http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=60983.

    U.S. Environmental Protection Agency. 1998. *An SAB Report: Development of the Acute Reference Exposure*. Science Advisory Board, Washington, D.C., EPA-SAB-EHC-99-005. November 1998. Available at: http://www.epa.gov/sab/pdf/ehc9905.pdf.

11. U.S. Environmental Protection Agency. 1986. *Guidelines for the Health Risk Assessment of Chemical Mixtures*. Risk Assessment Forum, Washington, D.C. EPA/630/R-98/002. Federal Register 51(185):34014-34025, September 24, 1986.

12. U.S. Environmental Protection Agency. 2000. *Supplementary Guidance for Conducting Health Risk Assessment of Chemical Mixtures*. Risk Assessment Forum, Washington, DC, EPA/630/R-00/002. Available at: http://www.epa.gov/ncea/raf/pdfs/chem_mix/chem_mix_08_2001.pdf.

13. U.S. Environmental Protection Agency. 1993. *Provisional Guidance for Quantitative Risk Assessment of Polycyclic Aromatic Hydrocarbons.* EPA/600/R-93/089.

14. Olson, J.R., Bellin, J.S., Barnes, D.G., et al. 1989. Reexamination of data used for establishing toxicity equivalency factors (TEFs) for chlorinated dibenzo-p-dioxins and dibenzofurans (CDDs 45 and CDFs). *Chemosphere* 18(1-6):371-381.

15. U.S. Environmental Protection Agency. 1987. *Interim Procedures for Estimating Risks Associated with Exposures to Mixtures of Chlorinated dibenzo-p-dioxins and -dibenzofurans (CDDs and CDFs).* EPA/625/3-87/012.

16. NATO/CCMS. 1989. *Scientific Basis for the Development of International Toxicity Equivalent Factor (I-TEF) Method of Risk Assessment for Complex Mixtures of Dioxins and Related Compounds*. Report No. 178, December, 1998.

17. Ahlborg, U., Becking, G.C., Birnbaum, L.S., et al. 1994. Toxicity equivalence factors for dioxin-like PCBs: Report on a WHO-ECEH and IPCS consultation, December 1993. *Chemosphere* 28(6):1049-1067.

18. van Leeuwen, F.X.R. 1997. Derivation of toxic equivalency factors (TEFs) for dioxin-like compounds in humans and wildlife. *Organohalogen Compounds* 34:237.

19. For more information on EPA's dioxin reassessment activities, see: http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=55264&CFID=12120688&CFTOKEN=95507561,

Chapter 13 Inhalation Risk Characterization

Table of Contents

13.1 Introduction 1

13.2 Quantification of Cancer Risk and Noncancer Hazard 4
- 13.2.1 Cancer Risk Estimates 5
  - 13.2.1.1 Characterization of Individual Pollutant Risk 5
  - 13.2.1.2 Characterization of Cancer Risk from Exposure to Multiple Pollutants 6
- 13.2.2 Noncancer Hazard Estimates 8
  - 13.2.2.1 Characterizing Individual Pollutant Hazard for Chronic Exposures . . . 8
  - 13.2.2.2 Characterizing Multiple Pollutant Hazard for Chronic Exposures 9
  - 13.2.2.3 Characterizing Hazard for Acute Exposures 11
- 13.2.3 Quantifying Risk From Background Sources 12

13.3 Interpretation and Presentation of Inhalation Cancer Risks and Noncancer Hazards 13
- 13.3.1 Presenting Risk and Hazard Estimates 17
- 13.3.2 Exposure Estimates and Assumptions 17
- 13.3.3 Toxicity Estimates and Assumptions 18
- 13.3.4 Assessment and Presentation of Uncertainty in Risk Characterization 19
  - 13.3.4.1 Practical Approaches to Uncertainty Assessment 20
  - 13.3.4.2 Presentation of Uncertainty Assessment 23
- 13.3.5 Additional Information 23

References 25

13.1 Introduction

The last component of risk assessment, risk characterization, integrates the information from the exposure assessment (Chapter 11) and toxicity assessment (Chapter 12), using a combination of qualitative and quantitative information and including a discussion of uncertainty and variability.[1] The risk characterization and its components should be presented so that the details of the analysis are transparent, clear, consistent with EPA guidance and policy, and will generally support the conclusion that the analysis is reasonable for its intended purpose. Risk assessors aim for the risk summary and risk conclusions to be complete, informative, and useful for decision-makers. One way of accomplishing this is to make sure that major uncertainties associated with determining the nature and extent of the risk are identified and discussed.

Risk = *f* (metric of exposure, metric of toxicity)

Risk characterization combines the information from the exposure assessment and the toxicity assessment to provide a quantitative estimate of potential cancer risk and/or hazard for other adverse effects, along with a statement of confidence about the data and methods used

EPA has developed several key policies about how to characterize and present risk assessment information. EPA's *Policy for Risk Characterization*[2] specifies that a risk characterization "be prepared in a manner that is clear, transparent, reasonable, and consistent with other risk characterizations of similar scope prepared across programs in the Agency." The purpose of the memorandum was to ensure that risk management decisions are well-supported and well-understood, both inside the EPA and outside the Agency. The confidence in the data, science policy judgments, and the uncertainties in the database should be clearly communicated. The 1995 *Guidance for Risk Characterization* has been updated by the *Handbook for Risk Characterization*, which provides more background and approaches to presenting the risk characterization results.[3] Risk assessors may want to become familiar with the information provided in both the policy and handbook before beginning a risk assessment.

A 1992 memorandum from EPA's Office of the Administrator provides guidance on describing risk assessment results.[4] This memorandum focuses on communicating the full range of information used in developing the assessment, rather than providing only point estimates of risk to the public. The risk characterization guidance and handbook[3] recommends presenting a full and complete picture of risk that includes: a statement of confidence about data and methods used to develop the assessment; greater consistency and comparability in risk assessment across EPA programs; and statement of the level of scientific judgment inherent in risk management decisions. Information should be presented on the range of exposures derived from exposure scenarios using multiple risk descriptors (e.g., central-tendency, high-end of individual risk, population risk, important sub-populations, if known). For risk management decisions, the risk estimates are compared to legally mandated or other risk objectives (see Part V of this Reference Manual).

Information should be presented on the range of exposures derived from exposure scenarios and on the use of multiple risk descriptors (e.g., central tendency, high end of individual risk, population risk, important sub-populations, if known) consistent with terminology in the *Guidance on Risk Characterization*, Agency risk assessment guidelines, and program-specific guidance.

EPA *Policy for Risk Characterization*[2]

Risks are often evaluated initially for **individuals** within the potentially exposed population. **Population risks** for the exposed population may also be estimated, which may be useful in estimating potential economic costs and benefits from risk reduction. Sensitive subpopulations should also be considered, when possible. Estimates of **incidence** also are possible (see Exhibit 13-1).

Incidence is defined by the National Cancer Institute as "The number of new cases of a disease diagnosed each year." For example, a State's cancer registry might report that the statewide 5-year average incidence of lung cancer (i.e., the average number of actual people that were diagnosed by a doctor over the 5 year period) is 700 new cases per 100,000 people (5-year averages are often used to provide an estimate that is more stable over time). In comparison, air toxics risk assessments provide only a theoretical estimate of the likelihood that an individual in the exposed population will contract cancer as a result of exposure over a period of time (e.g., 50 or 100 years of a facility lifetime).

The potential risks calculated for specific inhalation exposures are typically **incremental risks**; that is, they are potential risks that are *in addition to* those risks already faced by the population under study for reasons other than exposure to air toxics (e.g., hereditary, lifestyle risks such as smoking). The risk estimates are used to answer questions concerning the general risks posed to the exposed population, the risk levels of various groups within the population, and the potential range of risks across the population (e.g., central-tendency (e.g., average) or high-end (e.g., maximum) risk for individuals within the populations of interest).

Steps in an Inhalation Risk Characterization

1. Organize outputs of inhalation exposure and toxicity assessments.
2. Derive inhalation cancer risk estimates and noncancer hazard quotients for each pollutant in each pathway for each type of receptor being studied.
3. Derive cumulative inhalation cancer risk estimates and noncancer hazards for each receptor for all chemicals in a pathway and then across pathways.
4. Identify key features and assumptions of exposure and toxicity assessments.
5. Assess and characterize key uncertainties and variability associated with the assessment.
6. Consider additional relevant information (e.g., related studies).

Risk characterization should include a risk summary and risk conclusions that are complete, informative, and useful for decision-makers, and which clearly identify and discuss the major uncertainties associated with determining the nature and extent of risk. See references 2 and 3 at the end of this chapter for more information.

Estimated cancer risks and noncancer hazards are generally developed for each chemical to which people are exposed in the study area and each exposure pathway through which exposure can occur. The results are then summed in a specific way to provide total estimates of risk and hazard. The general steps involved in risk characterization are:

- Quantify risks and hazards for each chemical through each pathway for each receptor;
- Review exposure estimates and assumptions;
- Review toxicity estimates and assumptions;
- Assess uncertainties and variability; and
- Consider additional relevant information (e.g., related studies).

Exhibit 13-1. Estimates of Risk
Individual risk. Estimates of cancer risk are usually expressed as a statistical probability represented in scientific notation as a negative exponent of 10. For example, an additional risk of contracting cancer of one chance in 10,000 (or one additional person in 10,000) is written as 1×10^{-4} (or 1E-04). This means that for every 10,000 people that are exposed, ***in the way that we have presumed***, one of those people may develop cancer over their lifetime. Likewise, a risk of one person in one million is written 1×10^{-6} (or 1E-06) and a risk or one in one hundred thousand is written 1×10^{-5} (or 1E-05).
Population Risk. Estimates of cancer risk can be expressed as the number of people in the population who may have the same risk level (e.g., 1,000,000 people in the exposed population under study may have a risk of 1×10^{-6}, 2,495 may have a risk of 1×10^{-5}, and 300 may have a risk of 1×10^{-4}).
Incidence. Estimates of cancer risk can be expressed as the incidence of cancer cases in a population. For example, the estimated incidence of cancer in a population of 500,000 individuals where the individual risk is 1×10^{-5} (based on a 100 year exposure scenario) is simply: $$Population\ Size \times \frac{Individual\ Risk}{Averaging\ Time} \times Exposure\ Duration;\ or$$ $$500{,}000\ Individuals \times \frac{1\times 10^{-5}}{70\ Years} \times 100\ Years = up\ to\ 7\ New\ cancer\ cases$$ Note that since the individual cancer risk value is a lifetime value, it is divided by 70 years (average lifetime length) prior to multiplying by the exposure period duration (100 years). It is also important to note the assumptions in this example calculation (e.g., average population size of 500,000 individuals and individual lifetime risk value of 1×10^{-5} for the 100 year period). Given these assumptions, these possible seven new cases are the expected number of cases over the total exposure duration of 100 years. If one wanted to estimate the number of new cases per year, simply use an exposure duration of one year. In our example, $$500{,}000\ Individuals \times \frac{1\times 10^{-5}}{70\ Years} \times 1\ Year = up\ to\ 0.07\ new\ cancer\ cases$$ This points out two problems with using risk estimates to derive incidence estimates. First, a fraction of a cancer case (which often results from this exercise) is not a very helpful statistic when assessing a potential air toxics problem. Second, people living in different areas with the same individual risks, but with very different exposed population sizes can end up with very different incidence rates. For example, if our population above only had 10,000 people, the incidence rate would have been predicted to be no more than 0.1 (versus seven). While the first situation indicates a higher potential population impact, the second situation nevertheless indicates identical individual risk predictions for members of the population. Both metrics are informative to the risk manager, and reflect different considerations which may have different weights in different decisions. Other ways of describing risk to an exposed population are also possible.

Risk estimates in screening-level (Tier 1) analyses typically are deterministic estimates based on point estimates of exposure and toxicity. Deterministic estimates are useful screening tools in a tiered analysis, but need to be qualified by transparent discussions of the nature and extent of uncertainties in the input variables and the subsequent likely impact on the ultimate risk characterization. Deterministic analyses with appropriate uncertainty characterization can be used to identify situations of low incremental risk and to focus on areas where additional analysis might improve the basis for selection of a risk management action. At higher tiers of analyses, risk assessors commonly describe exposure (and less frequently, toxicity) by probability distributions rather than by point values and propagate these distributions through the exposure assessment and risk characterization process. This type of probabilistic analysis, which may address uncertainty and variability as distinct issues, will result in an estimate of risk that is a probability distribution rather than a point value. A more detailed discussion of the assessment and presentation of uncertainty in the risk characterization process is provided in Section 13.3.4. Probabilistic uncertainty analysis is discussed in Chapter 31.

13.2 Quantification of Cancer Risk and Noncancer Hazard

Quantification of risk and hazard is the step where exposure concentrations in air are combined with applicable inhalation dose-response values. Predictive cancer risk estimates are presented separately from noncancer hazard quotients. Risks are quantified for the pathways, receptors, and exposure scenarios outlined in the conceptual site model.

Risk versus Hazard...What's the Difference?

Risk assessors purposefully use the term *risk* to mean the statistical probability of developing cancer over a lifetime (even if exposure only occurs over a portion of that lifetime). Noncancer "risks," on the other hand, are not expressed as a statistical probability of developing a disease. Rather they are expressed as a simple comparison of the exposure concentration to a reference concentration associated with the observable adverse health effects. To help make this distinction, the potential harm from exposure to carcinogens is called "risk" and the potential harm from noncarcinogens is called "hazard."

Information about the distribution of exposure and risk for the population is an important component of risk characterization. Distributions are often more useful than point estimates. However, since developing fully distributional estimates of risk is usually out of the scope of most risk assessments, assessors can provide a sense of the range of risks by developing both central tendency and high-end estimates.[5]

- **Central tendency** estimates are intended to give a characterization of risk for the typical individual in the population. This is usually either based on the arithmetic mean risk (average estimate) or the median risk (median estimate).

- **High-end** estimates are intended to estimate the risk that is expected to occur in the upper range of the distribution (e.g., risk above about the 90th percentile of the population distribution). For example, the maximum exposed individual (MEI) risk or maximum individual risk (MIR) might be used to estimate high-end risks.

An evaluation of the uncertainty in the risk descriptors is an important component of the uncertainty discussion in the assessment. Both quantitative and qualitative evaluations of uncertainty can be useful to users of the assessment (see Section 13.3.4 and Chapter 31).

13.2.1 Cancer Risk Estimates

Estimated individual cancer risk is expressed as the upper bound probability that a person may develop cancer over the course of their lifetime as a result of the exposures under study. This predicted risk is the **incremental risk** of cancer from the exposure being analyzed that is above the risk that the individuals in the population have already (i.e., due to non-air toxics related issues). Due to the nature of the assumptions in their derivation, inhalation unit risks (IURs) are generally considered to be "plausible upper-bound" estimates of potency. As such, the calculated risks are usually a conservative estimate (i.e., the true risk may be lower).

As described above, risks may be estimated for both the central tendency (average exposure) case and for the high-end (exposure that is expected to occur in the upper range of the distribution) case. However, for both types of estimates, the same estimate of toxicity (i.e., an IUR or reference concentration [RfC]) is generally used to calculate the risk. In other words, while the estimate of exposure may be allowed to vary to derive a sense of the range of exposures in a population, *the same estimate of toxicity* is used to calculate risk for both average and high-end risks. With few exceptions, toxicity values are not currently presented as a range.

Cancer risk characterization typically is performed first for individual air toxics, then is summed over all of the air toxics to which a person may be exposed at the same time. These steps are described in separate subsections below.

13.2.1.1 Characterization of Individual Pollutant Risk

For inhalation exposures, chronic cancer risks for individual air toxics are typically estimated by multiplying the estimate of long-term exposure concentration (EC) by the corresponding IUR for each pollutant to estimate the potential incremental cancer risk for an individual:

$$\text{Risk} = EC_L \times IUR \qquad \text{(Equation 13-1)}$$

where:

Risk	=	Cancer risk to an individual (expressed as an upper-bound risk of contracting cancer over a lifetime);
EC_L	=	Estimate of long-term inhalation exposure concentration for a specific air toxic; and
IUR	=	the corresponding inhalation unit risk estimate for that air toxic.

Performing the estimate in this way provides an estimate of the probability of developing cancer over a lifetime due to the exposure in question. Because of the way this equation is written, the underlying presumption is that a person is exposed continuously to the EC_L for their full lifetime (usually assumed to be 70 years).[(a)] The EC_L is an estimate of this long-term exposure even

---

[a]EPA is currently reviewing methods for assessing cancer risk for less than lifetime exposures occurring in childhood. EPA's Draft Document *Supplemental Guidance for Assessing Cancer Susceptibility from Early-Life Exposure to Carcinogens* (http://www.epa.gov/sab/panels/sgacsrp.html) recommends a change to the current method for strong mutagens. This document is undergoing public and Science Advisory Board review and will be completed sometime in the future with consideration of that review. EPA's methods for air toxics assessments will be consistent with the final document.

though it is probably based on only one year's worth of monitoring data or a modeling run that covers only one year's worth of time. (As noted in Chapter 11, exposure modeling can be used, in some cases, to derive a better estimate of the amount of time people interact with contaminated air. Nevertheless, the probability of developing cancer is still averaged out over the full lifetime of the individual.)

Estimates of cancer risk are usually expressed as a statistical probability represented in scientific notation as a negative exponent of 10. For example, an additional risk of contracting cancer of one chance in 10,000 (or one additional person in 10,000) is written as 1×10^{-4} (or 1E-04). This means that for every 10,000 people that are exposed, ***in the way that we have presumed***, one of those people may develop cancer over their lifetime. Likewise, a risk of one person in one million is written 1×10^{-6} (or 1E-06) and a risk or one in one hundred thousand is written 1×10^{-5} (or 1E-05).

Because IURs are typically upper-bound estimates, actual risks may be lower than predicted (see Chapter 12), and the true value of the risk is unknown and may be as low as zero.[5] These statistical projections of hypothetical risk are intended as screening tools for risk managers and cannot make realistic predictions of biological effects. Such risk estimates also cannot be used to determine whether someone who already has cancer is ill because of a past exposure. Part VI of this volume provides an overview of the Public Health Assessment process used to evaluate whether past exposures resulted in current illness.

Risks for cancer are generally expressed as individual risks (i.e., the risk borne by an individual in a larger exposed population). The number of people in the population who have the same risk level may also be provided (e.g., 1,000,000 people in the exposed population under study have a risk of 1×10^{-6}, 2,495 have a risk of 1×10^{-5}, and 300 have a risk of 1×10^{-4}). It is also possible to calculate the number of expected cases of cancer expected over a 70-year period by multiplying the cancer risk to an individual by the number of individuals; however, even though the calculation might yield an estimate of incidence, low predicted cancer incidence rates (even vanishingly small) do not mean that individuals within the population will not get cancer because of air toxics exposures.

13.2.1.2 Characterization of Cancer Risk from Exposure to Multiple Pollutants

People may receive exposure to multiple chemicals, rather than a single chemical, at the same time. The concurrent exposure to multiple carcinogens may occur through the same pathway or across several pathways. With a few exceptions (e.g., coke oven emissions), cancer dose-response values (e.g., IURs) are usually available only for individual compounds within a mixture.

The following equation estimates the predicted cumulative incremental individual cancer risk from multiple substances, and assumes an additive effect from simultaneous exposures to several carcinogens:

$$\mathrm{Risk}_T = \mathrm{Risk}_1 + \mathrm{Risk}_2 + \ldots + \mathrm{Risk}_i \qquad \text{(Equation 13-2)}$$

where:

$Risk_T$ = total cumulative individual pathway-specific cancer risk (expressed as an upper-bound risk of contracting cancer over a lifetime); and
$Risk_i$ = individual risk estimate for the ith substance in the inhalation pathway.

In screening-level assessments of carcinogens for which there is an assumption of a linear dose-response, the cancer risks predicted for individual chemicals may be added to estimate cumulative cancer risk. This approach assumes that the risks associated with individual chemicals in the mixture are additive. In more refined assessments, the chemicals under assessment may be evaluated to determine whether effects from multiple chemicals are synergistic (greater than additive) or antagonistic (less than additive), although sufficient data for this evaluation are usually lacking. In those cases where IURs are available for a chemical mixture of concern, risk characterization can be conducted on the mixture using the same procedures used for a single compound. When more than one pathway is involved, the pathway specific risks are generally summed first, and then summed across pathways. This process is described in Part III of this reference manual. Note that for carcinogens being assessed based on the assumption of nonlinear dose-response, for which an RfC considering cancer as well as other effects has been derived, the hazard quotient approach will be appropriate (see Section 13.2.2).

Example Calculation to Estimate Cancer Risk (Hypothetical)

A Tier 1 modeling analysis was performed to estimate risk to the maximum exposed individual, assumed to reside at the point of maximum concentration for ABC Factory. Four HAPs were potentially of concern: benzene, dichloroethyl ether, formaldehyde, and cadmium compounds. Cancer risk estimates were obtained for each HAP by multiplying the estimated *annual average* EC by the IUR for each HAP. The resulting upper bound cancer risk estimates ranged from 2×10^{-6} (benzene, formaldehyde) to 8×10^{-4} (dichloroethyl ether). The cancer risk estimates for each HAP were summed to obtain an estimate of total inhalation cancer risk (9×10^{-4}). Note that 97 percent of the estimated total risk results from dichloroethyl ether, and that more than 99 percent results from dichloroethyl ether and cadmium compounds. In this hypothetical example, the risk assessor would need to decide which HAPs to carry to higher tiers by weighing the small proportion of risk posed by benzene and formaldehyde against the fact that these risks nevertheless exceeded one in one million.

HAP	EC $\mu g/m^3$	IUR $1/(\mu g/m^3)$	Cancer Risk Estimate[(a)]	Percent of Total Risk
Benzene	0.3	7.8×10^{-6}	2×10^{-6}	< 1%
Dichloroethyl ether	2.5	3.3×10^{-4}	8×10^{-4}	97 %
Formaldehyde	0.2	1.3×10^{-4}	2×10^{-6}	< 1 %
Cadmium compounds	0.01	1.8×10^{-3}	1×10^{-5}	2 %
Total			9×10^{-4}	

[(a)] Standard rules for rounding apply which will commonly lead to an answer of one significant figure in both risk and hazard estimates. For presentation purposes, hazard quotients (and hazard indices) and cancer risk estimates are usually reported as one significant figure.

13.2.2 Noncancer Hazard Estimates

For noncancer effects (as well as carcinogens being assessed based on the assumption of nonlinear dose-response), exposure concentrations are compared to RfCs, which are estimates (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive sub-populations) that is likely to be without an appreciable risk of deleterious noncancer effects during a lifetime (see Chapter 12).

13.2.2.1 Characterizing Individual Pollutant Hazard for Chronic Exposures

For inhalation exposures, noncancer hazards are estimated by dividing the estimate of the chronic inhalation EC by the RfC to yield a hazard quotient (HQ) for individual chemicals:

$$HQ = EC_C \div RfC \qquad \text{(Equation 13-3)}$$

where:

HQ	=	the hazard quotient for an individual air toxic;
EC_C	=	estimate of chronic inhalation exposure to that air toxic; and
RfC	=	the corresponding reference concentration for that air toxic.

In screening inhalation risk assessments, which are routinely built around a particular year's estimate of emissions, the exposure estimate is usually based on an assumption of continuous long-term exposure using an annual average as the estimate of exposure concentration. A more refined assessment (e.g., by use of an exposure model) may generate an estimate of a more realistic exposure (e.g., by the application of an exposure model or refined emissions estimates over the longer time period).

Based on the definition of the RfC, an HQ less than or equal to one indicates that adverse noncancer effects are **not likely to occur**, and thus can be considered to have negligible hazard. Unlike cancer risks, however, HQs greater than one are not statistical probabilities of harm occurring. Instead, they are a simple statement of whether (and by how much) an exposure concentration exceeds the RfC. Moreover, the level of concern does not increase linearly or to the same extent as HQs increase above one for different chemicals because RfCs do not generally have equal accuracy or precision and are generally not based on the same severity of effect. Thus, we can only say that with exposures increasingly greater than the RfC, (i.e., HQs increasingly greater than 1), the **potential for adverse effects increases**, but we do not know by how much. An HQ of 100 does not mean that the hazard is 10 times greater than an HQ of 10. Also an HQ of 10 for one substance may not have the same meaning (in terms of hazard) as another substance resulting in the same HQ.

Example Calculation to Estimate Chronic Noncancer Hazard (Hypothetical)

A Tier 1 modeling analysis was performed to estimate chronic noncancer hazard to the maximum exposed individual, assumed to reside at the point of maximum concentration for ABC Factory. Four HAPs were potentially of concern: benzene, dichloroethyl ether, formaldehyde, and cadmium compounds. Noncancer hazard estimates were obtained for each HAP by dividing the estimated Exposure Concentration (EC) by the Inhalation Reference Concentration (RfC) for each HAP (note that the EC is expressed in units of mg/m^3 for this analysis). The resulting Hazard quotient (HQ) estimates ranged from 1×10^{-3} (formaldehyde) to 1 (cadmium compounds). Note that no RfC was available for dichloroethyl ether. The HQs for each HAP were summed to obtain an estimate of the Hazard Index (HI) of 1. Note that cadmium compounds account for 95 percent of the HI, suggesting that the other HAPs may not need further consideration (although this determination should be made in consideration of all relevant information, including uncertainties such as confidence in the exposure concentration and uncertainty factors used to derive each RfC).

HAP	EC mg/m^3	RfC (mg/m^3)	HQ[(b)]	Percent of HI
Benzene	6×10^{-4}	6×10^{-2}	1×10^{-2}	1 %
Dichloroethyl ether[(a)]	5×10^{-3}	---	---	---
Formaldehyde	4×10^{-4}	1×10^{-2}	1×10^{-3}	4 %
Cadmium compounds	2×10^{-5}	2×10^{-5}	1	95 %
Hazard Index (HI)			1	

[(a)] note that the absence of an RfC value means that we cannot quantitatively assess a HAP.
[(b)] Standard rules for rounding apply which will commonly lead to an answer of one significant figure in both risk and hazard estimates. For presentation purposes, hazard quotients (and hazard indices) and cancer risk estimates are usually reported as one significant figure.

13.2.2.2 Characterizing Multiple Pollutant Hazard for Chronic Exposures

Noncancer health effects data are usually available only for individual compounds within a mixture. In these cases, the individual HQs can be summed together to calculate a multiple-pollutant hazard index (HI):

$$HI = HQ_1 + HQ_2 + ...+ HQ_i \qquad \text{(Equation 13-4)}$$

where

HI = hazard index; and
HQ = hazard quotient for the ith air toxic.

For screening-level assessments, a simple HI may first be calculated for all chemicals of concern within the inhalation pathway (adding hazards across pathways is discussed in Part III). If the HI is less than your decision criterion, a more refined analysis is usually not performed. Adding HQs in this fashion is based on the assumption that even when individual pollutant levels are

lower than the corresponding reference levels, some pollutants may work together such that their potential for harm is additive and the combined exposure to the group of chemicals poses greater likelihood of harm. Some groups of chemicals can also behave antagonistically, such that combined exposure poses less likelihood of harm, or synergistically, such that combined exposure poses harm in greater than additive manner. Where this type of HI exceeds the criterion of interest, a more refined analysis is warranted.

Although the HI approach encompassing all chemicals in a mixture is commonly used for a screening-level study, it is important to note that application of the HI equation to compounds that may produce different effects, or that act by different toxicological mechanisms, could overestimate the potential for effects. Consequently, it is more appropriate to calculate a separate HI for each endpoint of concern for which mechanisms of action are known to be similar.

Because the assumption of dose additivity is most appropriate for compounds that induce the same effect by similar modes of action, EPA's *Guidance for Conducting Health Risk Assessment of Chemical Mixtures* and *Supplementary Guidance*[6] suggest subgrouping pollutant-specific HQs by toxicological similarity of the pollutants for subsequent calculations; that is, to calculate a **target-organ-specific-hazard index (TOSHI)** for each subgrouping of pollutants. This calculation allows for a more appropriate estimate of overall hazard.

Segregation of hazard indices by effect and mechanism of action can be complex and time-consuming because it is necessary to identify all the major effects and target organism for each chemical and then to classify the chemicals according to target organ(s) or mechanism of action. This analysis is not simple and a toxicologist with familiarity in developing TOSHIs is best suited to perform this function. If the segregation is not carefully done, an underestimate of true hazard could result.

Procedure for Segregation of HIs by Effect

Segregation of HIs requires identification of the major effects of each chemical, including those seen at higher doses than the critical effect (e.g., the chemical may cause liver damage at an EC of 20 $\mu g/m^3$ and neurotoxicity at an EC of 50 $\mu g/m^3$). Major effect categories include neurotoxicity, developmental toxicity, reproductive toxicity, immunotoxicity, and adverse effects by target organ (i.e., hepatic, renal, respiratory, cardiovascular, gastrointestinal, hematological, musculoskeletal, and dermal/ocular effects).

Acute HQs are developed in the same manner as chronic HQs, with the caveat that the exposure duration associated with the exposure concentration should match the exposure duration embodied in the acute toxicity value. Whereas summing chronic HQs to a total hazard index is a relatively straightforward exercise, the issues related to developing acute HI are more subtle and complex. A toxicologist familiar with acute exposure and risk analysis should be consulted to perform this process.

13.2.2.3 Characterizing Hazard for Acute Exposures

Risk assessors can derive estimates of acute noncancer hazard for each HAP by combining the applicable short-term exposure concentration (EC) and acute dose-response value (AV) for the HAP to obtain the acute Hazard Quotient (HQ) for the HAP using the following equation:

$$HQ_A = EC_{ST} \div AV$$

where:

HQ_A = the acute hazard quotient for an individual HAP;
EC_{ST} = estimate of short-term inhalation exposure to that HAP; and
AV = the corresponding acute dose-response value for that HAP.

Note that ambient air concentrations are calculated for an exposure duration compatible with the acute dose-response value used.

Available acute dose-response values are more diverse than chronic values, because they were developed for different purposes and considering different exposure durations. The most effective characterization of acute risk often is to compare the maximum estimated hourly concentrations with a range of acute dose-response values from sources described in Chapter 12. If the ambient concentration is lower than all the acute benchmarks, it is generally reasonable to conclude that the potential for significant acute hazard is negligible. If the concentration exceeds some benchmarks but not others, the assessment should include a discussion of the implications for the chemical of interest, with attention to the details of both the exposure scenario and the benchmarks included in the analysis.

Acute noncancer health effects data are usually available only for individual HAPs within a mixture. In these cases, it may be possible to combine the individual acute HQs to calculate a multi-pollutant acute hazard index (HI) using the following formula:

$$HI_A = HQ_{A1} + HQ_{A2} + ...+ HQ_{Ai}$$

where

HI_A = acute hazard index; and
HQ_{Ai} = acute hazard quotient for the ith HAP.

Although this appears similar to the process for combining chronic HQs, the summing of acute HQs is complicated by several issues that do not pertain to chronic HQs. First, acute dose-response values have been developed for purposes that vary more widely than chronic values. Some sources of acute values define exposures at which adverse effects actually occur, while other sources develop only no-effect acute values. Second, some acute values are expressed as concentration-time matrices, while others are expressed as single concentrations for a set exposure duration. Third, some acute values may specifically consider multiple exposures, whereas others consider exposure as a one-time event. Fourth, some sources of acute values are intended to regulate workplace exposures, assuming a population of healthy workers (i.e., without children, seniors, or other sensitive individuals). Such occupational values may also consider cost and feasibility, factors that EPA considers the province of the risk manager rather than the risk assessor.

Given these differences among acute values with regard to their purposes, and the different types of acute exposure characterization that may be performed, the acute HI analysis is most informative when limited to acute values from the same source, the same level of effects, and the same duration. Analyses that mix sources, effects levels, and durations are likely to be misleading.

Risk assessors commonly evaluate acute noncancer hazard using a variety of different acute values from different sources, and discuss the resulting hazard estimates considering the purpose for which each of value was developed. This kind of evaluation should only be done by an experienced toxicologist. **The significance of these HQs and HIs would need to be considered in the context of the purpose of the risk assessment and the characteristics of the dose-response values, such as their purpose, averaging time, and health endpoints**. EPA is working to provide more comprehensive guidance on what benchmarks to rely upon and plans to develop a relevant acute benchmark methodology.

13.2.3 Quantifying Risk From Background Sources

In some cases, it may be appropriate to quantify background concentrations of the air toxics of concern. For example, background concentrations may be a critical element in determining the need for further reductions of emissions from a particular source. Background concentrations are the levels of contaminants that would be present in the absence of contaminant releases from the source(s) under evaluation. Background concentrations may occur naturally in the environment or originate from other human sources (e.g., an industrial area upwind from the sources of concern).

The general approach in risk assessments and risk management decisions has often been to assess the incremental risk posed by emissions from a particular source or group of sources. Various EPA programs, however, have taken specific approaches to considering background risks, some of which are summarized in EPA's *Residual Risk Report to Congress*.[7]

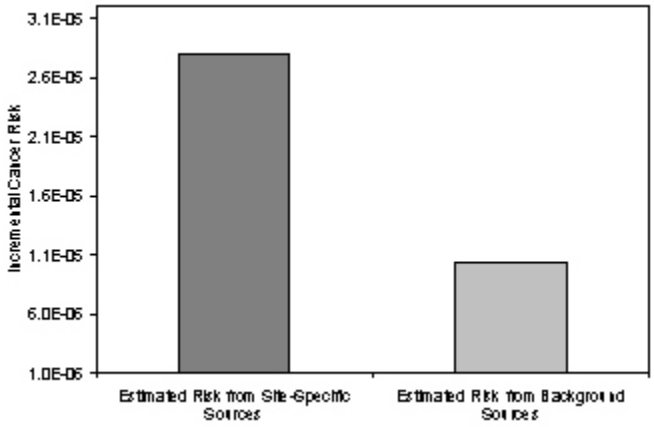
A detailed analysis of background concentrations typically would require extensive data gathering and modeling beyond that required for the incremental risk analysis. For example, numerous nearby (and possibly distant) air toxics sources of varying types would need to be characterized in sufficient detail to support release and exposure modeling. The data needs for assessment of background concentrations may differ depending on what will be done with the data. For example, if the question is simply "what is the risk to the population in a specific place," then an assessment of background may be unnecessary (monitoring data in the study area may be all that is required). On the other hand, if the question is "what is the risk and what can we do about it," then a knowledge of how much risk is contributed from both local and background sources may be necessary. If the risk is unacceptably high, but most of the risk is background in nature, there may be no appropriate risk reduction strategy (especially in regard to local sources).

Interpreting background concentrations may be difficult for anthropogenic chemicals and for chemicals formed through chemical reactions. For example, when trying to estimate background formaldehyde concentrations, it is difficult to screen out the reactive precursors which change in the study area from those that change before entering the study area. Also, if a source of nitrogen oxides (NO_x) is not present, secondary formation of formaldehyde may be slowed.

The presence of high background concentrations of anthropogenic chemicals could increase public concerns in some situations (see Part V of this reference manual for discussion of risk communication). On the other hand, knowledge of background risks could help place the air risks from a particular source or source area in better perspective.

In general, the most appropriate way to evaluate the contribution of background concentrations to the risk estimate is to simply compare the risk attributable to known or estimated (e.g., through monitoring) background concentrations in a bar chart against the risk attributable to the source(s) being evaluated (see Exhibit 13-3). Note that the study-specific risk estimate will be based on a metric of total exposure (when monitoring data are available) or incremental exposure (when modeling data are available. It generally is not appropriate to subtract background concentrations from monitored values.

Exhibit 13-3. Example Comparison of Risk Estimates from Study-specific and Background Sources



In this example, the estimated risk from the specific sources being evaluated (2.8×10^{-5}) and the estimated risk from background sources (1.0×10^{-5}) are compared side-by-side. This places the risk estimates from the sources of concern in an appropriate regional context.

13.3 Interpretation and Presentation of Inhalation Cancer Risks and Noncancer Hazards

In the final part of the risk characterization, risk assessors commonly present estimates of health risk in the context of uncertainties and limitations in the data and methodology. Exposure estimates and assumptions, toxicity estimates and assumptions, and the assessment of uncertainty are usually discussed. Additionally, information relevant to the public health context of the estimated risks is presented.

EPA's *Policy for Risk Characterization*[2] describes a philosophy of transparency, clarity, consistency, and reasonableness (TCCR), and provides detailed approaches to achieving TCCR. Exhibit 13-4 provides an overview of EPA's TCCR principles.

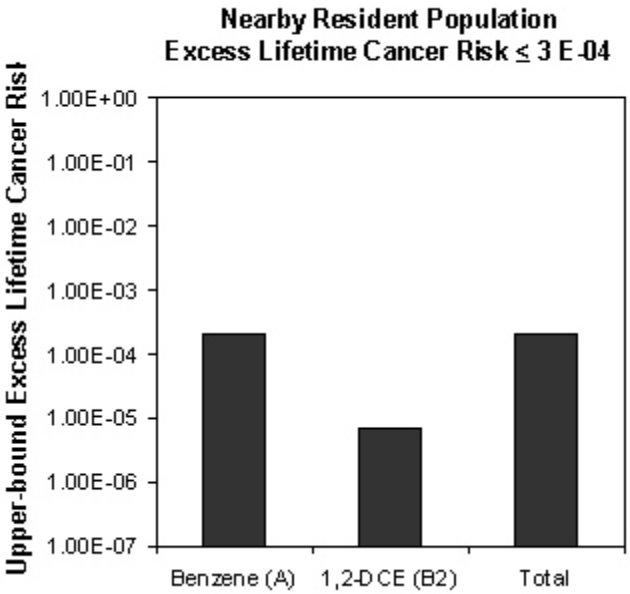
Exhibit 13-4. Transparency, Clarity, Consistency, and Reasonableness Principles		
Principle	**Definition**	**Criteria for a Good Risk Characterization**
Transparency	Explicitness in the risk assessment process	• Describe assessment approach, assumptions, extrapolations, and use of models • Describe plausible alternative assumptions • Identify data gaps • Distinguish science from policy • Describe uncertainty • Describe relative strength of assessment
Clarity	The assessment itself is free from obscure language and is easy to understand	• Employ brevity • Use plain English • Avoid technical terms • Use simple tables, graphics, and equations
Consistency	The conclusions of the risk assessment are characterized in harmony with EPA actions	• Follow statutes • Follow Agency guidance • Use Agency information systems • Place assessment in context with similar risks • Define level of effort • Use review by peers
Reasonableness	The risk assessment is based on sound judgment	• Use review by peers • Use best available scientific information • Use good judgment • Use plausible alternatives
Source: EPA *Risk Characterization Guidance*[3]		

The risk characterization document should allow the risk manager, and the public, to know why risk was assessed the way it was, by clearly summarizing the available data and its analysis, uncertainties, alternative analyses, and the choices made. A good risk characterization will state the scope of the assessment, express results clearly, articulate major assumptions and uncertainties, identify reasonable alternative interpretations, and separate scientific conclusions from science policy judgments. The *Policy for Risk Characterization* calls for the explanation of the choices made to be highly visible.

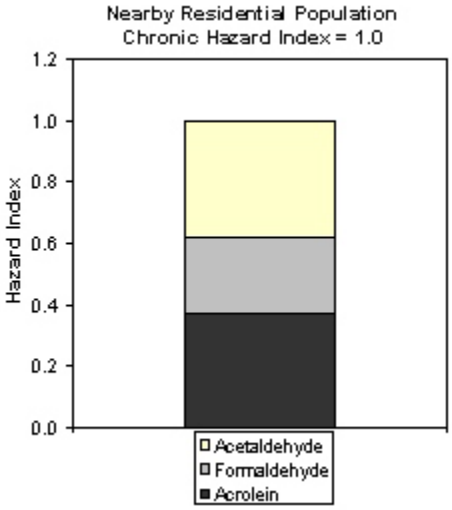
The goal of risk characterization is to clearly communicate the key findings and their strengths and limitations so that decision-makers can put the risk results into context with other information critical to evaluating risk management options (e.g., economics, social values, public perception, policies). The risk characterization will provide a means of placing the numerical estimates of risk and hazard in the context of what is known and what is not about the potential exposures and should include the elements listed in Exhibit 13-5. Exhibit 13-6 provides examples of graphical presentations of risk estimates.

Exhibit 13-5. Elements Commonly Included in the Risk Characterization Discussion
• Agreement that the key contaminants were identified • A discussion of modeled or measured air concentrations relative to background • The magnitude of the estimated cancer risks and noncancer hazard indices, and a description of the types of health risks potentially present, distinguishing between known effects in humans and those found only in experimental animals • The level of confidence in the toxicity data used to estimate risks • A presentation of qualitative information about the toxicity of substances not included in the quantitative risk assessment • Level of confidence in the exposure estimates for key exposure pathways and related exposure parameter assumptions • The major factors driving the risks (e.g., substances, pathways) • The major factors reducing the certainty in the results and the significance of these uncertainties (e.g., a change in the assumption for a certain parameter could increase/decrease the risk estimate). • The exposed population characteristics • A comparison with location-specific health studies, if available

Exhibit 13-6. Example Comparison of Risk Results for a Hypothetical Risk Assessment



The risk of developing cancer is plotted as shown. A risk of 1×10^{-4} (1 E-04) indicates a probability of one chance or less in 10,000 of an individual developing cancer. Risks of 1×10^{-5} (1 E-05) and 1×10^{-6} (1 E-06) correspond to probabilities of one chance or less in 100,000 and one million, respectively. Values in parentheses represent EPA's Weight-of-Evidence classification of the agent as a potential human carcinogen: A = human carcinogen; B2 = probable human carcinogen (with sufficient evidence in animals and inadequate or no evidence in humans).



The hazard index is equal to the sum of the hazard quotients (i.e., exposure concentration/RfC) for each chemical. It is not a probability. A hazard index $\leq$ 1 indicates that it is unlikely for even sensitive populations to experience adverse health effects. Thus, hazard is negligible.

13.3.1 Presenting Risk and Hazard Estimates

Risk and hazard estimates will usually be presented both to risk managers and to the public. Depending on the audience, risk characterizations can present information with different amounts of technical detail as required, although avoiding the use of technical terms generally improves clarity. Presentations may include the assumptions the risk assessment used, as well as the distribution of risks estimated for the assessment. Multiple point estimates and risk ranges could be discussed in both narrative and tabular forms. The discussion of results may include items such as:

- The range of risks estimated within specified distances from the source(s) of concern;
- An estimate of population size associated with different risk levels; and/or
- A comparison of the magnitude of the risk estimate to background risks.

Key issues and conclusions should be clearly highlighted in any summary. Exhibit 13-7 identifies several summary products that can facilitate risk communication. (See also Part V of this Reference Manual for a description of various techniques for communicating risk.)

Exhibit 13-7. Summary Products to Facilitate Risk Communication
• **Executive summary** – a summary with some technical detail, for audiences with some technical knowledge (e.g., first line managers). This executive summary may sometimes be the executive summary of the technical risk characterization itself depending on the audience. • **Bulleted list** – a list highlighting the key issues and conclusions culled from the technical risk characterization with little or no technical detail; for audiences with little or no technical knowledge (e.g., higher-level managers, decision makers). • **Briefing packages** – written products that describe key issues and conclusions for managers, decision makers, and other public officials. • **Fact sheets, press releases, and public relations notices** – written products that describe key issues and conclusions for non-technical audiences (e.g., affected or interested public). • **Slide shows, speeches, and talks** – visual presentations (perhaps accompanied by audio presentations) and transcripts of oral presentations of key issues and their context; for mostly non-technical audiences.

13.3.2 Exposure Estimates and Assumptions

For each exposure pathway evaluated in the risk assessment, check that all information needed to characterize exposure is available. For each exposure pathway evaluated, exposure estimates and assumptions should be reviewed to assure the consistency and validity of key assumptions. These assumptions may include, for example, the period of exposure and the modeling assumptions.

The risk characterization section on exposure may summarize the following exposure information:

- Estimated exposures (chronic, subchronic, and shorter-term, as appropriate); and
- Important exposure modeling assumptions, including:
  - Chemical concentration at the exposure points; and
  - Frequency and duration of exposure.

Other items that could be addressed in the risk characterization summary of the exposure assessment include:

- The most significant sources of environmental exposure:
  - Data on sources of exposure from different media (when multimedia analyses are performed);
  - Estimates of the relative contribution of different sources of exposure; and
  - Identification of the most significant environmental pathways for exposure (when multimedia analyses are performed);

- Descriptions of the populations that were assessed, including the general population, highly exposed groups, and highly susceptible groups;

- Description of the basis for the exposure assessment, including any monitoring, modeling, or other analyses of exposure distributions (e.g., probabilistic techniques – see Part VII of this Reference Manual); and

- Key descriptors of exposure:
  - Description and illustration of the (range of) exposures to: "average" individuals, "high-end" individuals, the general population, and special subpopulations such as children and the elderly;
  - Description of how the central tendency estimate was developed, including the factors and/or methods used in developing this estimate;
  - Description of how the high-end estimate was developed;
  - Description of how population estimates of risk were developed; and
  - Description of how any incidence calculations were performed.

13.3.3 Toxicity Estimates and Assumptions

During the risk characterization step, the risk assessor usually reviews whether all toxicity information needed to characterize risk is available. The risk characterization section on toxicity often summarizes the following information:

- IURs for all carcinogenic chemicals;
- Discussion of weight of evidence and classifications for all carcinogenic chemicals;
- Type of human cancer for Class A carcinogens;
- Chronic and subchronic dose-response values and shorter-term (acute) dose-response values (if appropriate) for all chemicals (including carcinogens and developmental toxicants);
- Critical effect associated with each dose-response value;

- Discussion of uncertainties, uncertainty factors, and modifying factors used in deriving each dose-response value and degree of confidence in dose-response values;
- Whether the dose-response values are expressed as absorbed or administered doses (applies primarily to ingestion exposures - See Chapter 22);
- Pharmacokinetic data that may affect the extrapolation from animals to humans for dose-response values; and
- Uncertainties in any route-to-route extrapolation.

13.3.4 Assessment and Presentation of Uncertainty in Risk Characterization

The risk estimates used in air toxics risk assessments usually are not fully probabilistic estimates of risk but conditional estimates given a considerable number of assumptions about exposure and toxicity. Air toxics risk assessments make use of many different kinds of scientific concepts and data (e.g., exposure, toxicity, epidemiology), all of which are used to characterize the expected risk in a particular environmental context. Informed use of reliable scientific information from many different sources is a central feature of the risk assessment process. Reliable information may or may not be available for many aspects of a risk assessment. Scientific uncertainty is inherent in the risk assessment process, and risk managers almost always must make decisions using assessments that are not as definitive in all important areas as would be desirable. Risk assessments also incorporate a variety of professional and science policy judgements (e.g., which models to use, where to locate monitors, which toxicity studies to use as the basis of developing dose-response values). Risk managers therefore need to understand the strengths and the limitations of each assessment, and to communicate this information to all participants and the public.[2] A critical part of the risk characterization process, therefore, is an evaluation of the assumptions and uncertainties inherent in the risk assessment in order to place the risk estimates in proper perspective.

One of the key purposes of uncertainty analysis is to provide an understanding of where the estimate of exposure, dose, or risk is likely to fall within the range of possible values. Often this is expressed as a subjective confidence interval within which there is a high probability that the estimate will fall. A related analysis, termed “sensitivity analysis” or “analysis of uncertainty importance,” is often performed to identify the relative contribution of the uncertainty in a given parameter value (e.g., emission rate, ingestion rate) or model component to the total uncertainty in the exposure or risk estimate.[8] Often this is used either to identify which parameter values should be varied to provide high-end vs. central-tendency risk estimates, or to identify parameter values where additional data collection (or modeling effort) can increase the confidence in the resulting risk estimate.

The Presidential/Congressional Commission on Risk Assessment and Risk Management (CRARM) recommends that risk assessors respect the objective scientific basis of risks and procedures for making inferences in the absence of adequate data.[9] Risk assessors should provide risk managers and other stakeholders with plausible conclusions about risk that can be made on the basis of the available information, along with evaluations of the scientific weight of evidence supporting those conclusions and descriptions of major sources of uncertainty and alternative views.

The risk characterization typically should address the following:

- Considering the hazard and the exposure, what is the nature and likelihood of the health risk?
- Which individuals or groups are at risk? Are some people more likely to be at risk than others?
- How severe are the anticipated adverse impacts or effects?
- Are the effects reversible?
- What scientific evidence supports the conclusions about risk? How strong is the evidence?
- What is uncertain about the nature or magnitude of the risk?
- What is the range of informed views about the nature and probability of the risk?
- How confident are the risk analysts about their predictions of risk?
- What other sources cause the same type of effects or risks?
- What contribution does the particular source make to the overall risk of this kind of effect in the affected community? To the overall health of the community?
- How is the risk distributed in relation to other risks to the community?
- Does the risk have impacts besides those on health or the environment, such as social or cultural consequences?
- The level of detail considered in a risk assessment and included in a risk characterization should be commensurate with the problem's importance, expected health or environmental impact, expected economic or social impact, urgency, and level of controversy, as well as with the expected impact and cost of protective measures.

Risk characterizations should include sufficient information to enable:

- Risk managers to make a useful risk management decision, and
- Stakeholders to understand the importance and context of that decision.

13.3.4.1 Practical Approaches to Uncertainty Assessment

Sources of Uncertainty

- **Scenario uncertainty**. Information to fully define exposure or risk is missing or incomplete
- **Model uncertainty**. Algorithms or assumptions used in models may not adequately represent reality
- **Parameter uncertainty**. Values for model parameters cannot be estimated precisely
- **Decision-rule uncertainty**. Policy and other choices made during the risk assessment may influence risk estimates

There are numerous sources of uncertainties in air toxics risk assessments, and each merits consideration. The degree to which these sources of uncertainty need to be quantified, and the amount of uncertainty that is acceptable, varies considerably on a study-specific basis. For a screening-level (Tier 1) analysis, a high degree of uncertainty is often acceptable, provided that conservative assumptions are used to bias potential error toward protecting human health. The use of conservative assumptions is intended to result in a situation where the risk assessor is confident that the risk estimate is unlikely to be *greater* than the point estimate of risk. In other words, the point estimate of risk is expected to be at the high-end of the range of possible values. The uncertainty characterization for a Tier 1 analysis commonly is limited to a qualitative discussion of the major sources of uncertainty and their potential impact on the risk estimate. At higher tiers of analysis, sensitivity analysis to quantify the impact of varying input parameter values (or model

algorithms) on the risk estimate, or more complete quantitative uncertainty analysis, commonly are performed to more fully describe the range of possible or plausible values.

Practical approaches to the assessment and presentation of the principal sources of uncertainty in risk assessments are summarized below.[10]

Characterize Scenario Uncertainty. There are uncertainties associated with the estimate of the magnitude and extent of chemical exposure or toxicity, the spatial and temporal aggregation of chemical concentrations to calculate the exposure concentration used in the risk characterization, the completeness of the analysis (e.g., important exposure pathways may not have been evaluated), and the manner in which the exposed population and/or exposure scenario were specified for the analysis. Ideally, the key scenario uncertainties have been discussed during planning, scoping, and problem formulation, and the analysis plan has been developed to address these uncertainties. A limited sensitivity analysis (e.g., on key assumptions associated with exposure) may indicate the magnitude of uncertainty associated with specific aspects of the scenario. At a minimum, the analysis of uncertainty should identify the key scenario uncertainties and indicate the potential impact of each on the direction and magnitude of the risk estimate.

Characterize Model Uncertainty. There are uncertainties associated with the selection of scientific models; these include dose-response models, models of environmental fate and transport, and exposure models. There is always some doubt as to how well an exposure model or its mathematical expression approximates the true relationships between site-specific environmental conditions. Ideally one would like to use a fully validated model that accounts for all the known complexities in the parameter interrelationships for each assessment. Often, however, only partially validated models are available. As a consequence, it is important to identify key model assumptions (e.g., linearity, homogeneity, steady-state conditions, equilibrium) and their potential impact on the risk estimates. In the absence of field data for model validation, the risk assessor could perform a limited sensitivity analysis (i.e., vary assumptions about functional relationships) to indicate the magnitude of uncertainty that might be associated with model form. At a minimum, the analysis of uncertainty should list key model assumptions and indicate the potential impact of each on the direction and magnitude of the risk estimate.

Characterize Model Uncertainties

- List/summarize key model assumptions
- Indicate the potential impact of each assumption on the exposure and risk estimate
  - Direction
  - Magnitude

Characterize Parameter Uncertainty. During the course of a risk assessment, numerous parameter values are included in the calculations of chemical fate and transport and human intake. Significant data gaps might have required that certain parameter values be assumed for the risk assessment. For example, no information on the time spent outdoors may be available for a specific population, and a national average may be used instead. Even if data on the parameter of interest are available, they will be uncertain because the parameter estimates are derived from a sample of the potentially exposed population. A first step in characterizing parameter value uncertainty is to identify the key parameters influencing the risk estimate. This usually can be accomplished by expert opinion or by an explicit sensitivity analysis. In a

sensitivity analysis, the values of parameters suspected of driving the risk estimates are varied, and the degree to which changes in the input variables result in changes in the risk estimates are summarized and compared. It may be possible to reduce parameter uncertainty in the most sensitive parameters by additional, selective data gathering.

Characterize Decision-Rule Uncertainty. There are uncertainties associated with policy and other choices made during the risk assessment. For example, the exposure assessment might have evaluated an exposure duration (e.g., a subchronic exposure) for which no appropriate dose-response value was available. Uncertainty would be associated with the choice of value to use in the hazard characterization (e.g., an acute versus chronic value). In this situation, it might be possible to assess hazard twice, once with the acute value, and once with the chronic value, to may indicate the magnitude of uncertainty associated with this decision. At a minimum, the analysis of uncertainty should identify the key decision-rule uncertainties and indicate the potential impact of each on the direction and magnitude of the risk estimate.

Tracking Uncertainty. Ideally, one would like to quantitatively carry through the risk assessment the uncertainty associated with each parameter in order to characterize the uncertainty associated with the final risk estimates. However, this process can be highly complex and resource intensive and the more practical approach for air toxics risk assessments may be to describe qualitatively how the uncertainties might be propagated through the risk analysis. Three different approaches to tracking uncertainty are described below:

- *Qualitative Approach*. This approach involves developing a quantitative or qualitative description of the uncertainty for each parameter and indicating the possible influence of these uncertainties on the final risk estimates given knowledge of the models used.

- *Semi-Quantitative Approach*. This approach involves: (1) using available data to describe the potential range of values that the parameters might assume; (2) performing sensitivity analysis to identify the parameters with the most impact on the risk estimate; and (3) performing sensitivity analysis to compute the range of exposure or risk estimates that result from combinations of minimum and maximum values for some parameters and mid-range values for others.

- *Quantitative Approach*. Probabilistic techniques such as Monte Carlo simulation analysis can explicitly characterize the extent of uncertainty and variability in risk assessment, especially in the exposure assessment step. Using these techniques, important variables in the exposure assessment, as well as in the other parts of the risk assessment, are specified as distributions (rather than as single values) according to what can be expressed about their underlying variability and/or uncertainty. Values are sampled repeatedly from these distributions and combined in the analysis to provide a range of possible outcomes. While this technique can offer a useful summary of complex information, it must be noted that the analysis is only as certain as the underlying data (and assumed forms of the distribution of data values in the population). It is important that the risk assessor clearly expresses individual modeled variables in a way that is consistent with the best information available. Highly quantitative statistical uncertainty analysis is usually not practical or necessary for most air toxics risk assessments. The general quantitative approach to propagating or tracking uncertainty through probabilistic modeling is described in Chapter 31.

13.3.4.2 Presentation of Uncertainty Assessment

The final discussion of the risk characterization results must place the numerical estimates of risk in the context of the uncertainties inherent in the analysis.[2] The discussion should include:

- Level of confidence in the quantitative toxicity information used to estimate risks;
- Presentation of qualitative information on the toxicity of substances not included in the quantitative assessment;
- Level of confidence in the exposure estimates for key exposure pathways and related exposure parameter assumptions;
- Major factors reducing certainty in the results and the significance of these uncertainties (e.g., adding individual risk estimates for several substances or across multiple exposure pathways); and
- Possible graphical presentation of key parameter and risk uncertainties.

13.3.5 Additional Information

Other studies relevant to the risk assessment being performed may be available, such as community health studies or previous risk assessments. For example, the Agency for Toxic Substances and Diseases Registry (ATSDR) may conduct public health assessments, health consultations, and other activities resulting in evaluations, assessments, and recommendations on specific public health issues related to actual or potential human exposure to hazardous materials (see Chapter 30). ATSDR's recommendations may include additional hazard characterization or risk reduction activities. In addition, these activities can initiate other activities within ATSDR such as exposure investigations, health studies, and health education.

If health or exposure studies have been identified and evaluated as adequate, the study findings may be incorporated into the risk characterization to strengthen the conclusions of the risk assessment. In general, a qualitative comparison of the results of available studies will usually be sufficient.

Additional References Related to Uncertainty Analysis

Hattis. D.B. and D.E. Burmaster. 1994. Assessment of variability and uncertainty distributions for practical risk analyses. *Risk Analysis* 14(5):713-730.

U.S. Environmental Protection Agency. 1997. *Guiding Principles for Monte Carlo Analysis.* Risk Assessment Forum, Washington, DC. March 1997. EPA-630/R-97-001.

U.S. Environmental Protection Agency. 1988. *National Emission Standards for Hazardous Air Pollutants. Federal Register* 53(145):28496-28056, Proposed Rule and Notice of Public Hearing. July 28, 1988.

Finkel, A.M. 1990. *Confronting Uncertainty in Risk Management: A Guide for Decision-Makers.* Center for Risk Management, Resources for the Future. Washington, DC.

U.S. Environmental Protection Agency. 1997. *Policy for Use of Probabilistic Analysis in Risk Assessment.* Office of the Administrator, Washington, DC. May 15, 1997.

National Council on Radiation Protection and Measurements. 1996. *A Guide for Uncertainty Analysis in Dose and Risk Assessments Related to Environmental Contamination*. NCRP Commentary No. 14; available at http://www.ncrp.com/comm14.html.

Information Quality Guidelines

The U.S. Office of Management and Budget (OMB) has directed all federal agencies to develop information quality guidelines for risk-related and other information; EPA has developed draft guidelines pursuant to the OMB directive. While these guidelines do not apply to S/L/T governments, they provide useful principles for developing and communicating the information developed for the risk characterization.

The OMB guidelines denote four substantive qualifiers for information disseminated by federal agencies. **Quality** is defined as the encompassing term, of which utility, objectivity, and integrity are the constituents. **Utility** refers to the usefulness of the information to the intended users. **Objectivity** focuses on whether the disseminated information is being presented in an accurate, clear, complete, and unbiased manner, and as a matter of substance, is accurate, reliable, and unbiased. **Integrity** refers to security – the protection of information from unauthorized access or revision, to ensure that the information is not compromised through corruption or falsification.

The guidelines provide some basic principles for agencies to consider when developing their own guidelines, including:

- Guidelines should be flexible enough to address all communication media and variety of scope and importance of information products.
- Some agency information may need to meet higher or more specific expectations for objectivity, utility, and integrity.
- Ensuring and maximizing quality, objectivity, utility, and integrity comes at a cost, so agencies should consider using a cost-benefit approach.
- Agencies should adopt a common-sense approach that builds on existing processes and procedures. It is important that agency guidelines do not impose unnecessary administrative burdens.

EPA developed draft information quality guidelines in response to the OMB directive (www.epa.gov/oei/qualityguidelines). EPA's guidelines include two components of particular relevance to air toxics risk management: (1) guidelines to ensure and maximize the quality of "influential" information; and (2) guidelines to ensure and maximize the quality of "influential" scientific risk assessment information.

Source: Office of Management and Budget. 2002. *Guidelines for Ensuring and Maximizing the Quality, Objectivity, Utility, and Integrity of Information Disseminated by Federal Agencies.* 67 *Federal Register* 36:8451. February 22, 2002 (www.whitehouse.gov/omb/fedreg/reproducible.html).

References

1. U.S. Environmental Protection Agency. 2003. *Framework for Cumulative Risk Assessment (External Review Draft).* Office of Research and Development, National Center for Environmental Assessment, Washington, D.C., April 23, 2002. EPA/630/P-02/001F. Available at: http://cfpub.epa.gov/ncea/raf/recordisplay.cfm?deid=54944.

   U.S. Environmental Protection Agency. 1997. *Guidance on Cumulative Risk Assessment, Part 1, Planning and Scoping*. Science Policy Council, Washington, D.C.

   U.S. Environmental Protection Agency. 1984. *Risk Assessment and Management: Framework for Decision Making*, Washington, D.C. EPA 600/9-85-002.

2. U.S. Environmental Protection Agency. 1995. *Policy for Risk Characterization* ("Browner Memorandum"). Science Policy Council, Washington, D.C., March 1995. Available at: http://64.2.134.196/committees/aqph/rcpolicy.pdf.

3. U.S. Environmental Protection Agency. 1995. *Guidance for Risk Characterization*. Science Policy Council, Washington, D.C., February 1995. Available at: http://www.epa.gov/osa/spc/htm/rcguide.htm.

4. U.S. Environmental Protection Agency. 1992. *Guidance on Risk Characterization for Risk Managers and Risk Assessment.* Memorandum. Office of the Administrator, Washington, D.C.

5. U.S. Environmental Protection Agency. 1986. *Guidance Mutagenicity Risk Assessment of Chemical Mixtures*. Risk Assessment Forum, Washington, D.C., September 1986. EPA/630/R-98/003. (Published in the *Federal Register* 51: 33997-8, Sept 24, 1986). Available at: http://cfpub.epa.gov/ncea/raf/pdfs/mutagen2.pdf.

6. U.S. Environmental Protection Agency. 1986. *Guidance for Conducting Health Risk Assessment of Chemical Mixtures*. Risk Assessment Forum, Washington, D.C. EPA/630/R-98/002. Available at: http://cfpub.epa.gov/ncea/raf/recordisplay.cfm?deid=20533.

   U.S. Environmental Protection Agency. 2000. *Supplementary Guidance for Conducting Health Risk Assessment of Chemical Mixtures.* Risk Assessment Forum, Washington, D.C. EPA/630/R-00/002. Available at: http://cfpub.epa.gov/ncea/raf/recordisplay.cfm?deid=20533.

7. U.S. Environmental Protection Agency. 1999. *Residual Risk Report to Congress*. Office of Air Quality Planning and Standards, Research Triangle Park, NC, March 1999. EPA-45/R-99/001. Available at: http://www.epa.gov/ttn/oarpg/t3/reports/risk_rep.pdf.

8. Morgan, G. and Henrrion. M. 1990. *Uncertainty: A Guide to Dealing with Uncertainty in Quantitative Risk and Policy Analysis.* Cambridge University Press, Cambridge, U.K.

9. Commission on Risk Assessment and Risk Management (CRARM). 1996. *Risk Assessment and Risk Management in Regulatory Decision-Making* (The "White Book"). Draft Report, Washington, D.C.

   CRARM. 1997. *Framework for Environmental Health Risk Management. Final Report, Volume 1*. Washington, D.C.

   CRARM. 1997. *Risk Assessment and Risk Management in Regulatory Decision-Making. Final Report, Volume 2*. Washington, D.C.

10. U.S. Environmental Protection Agency. 1989. *Risk Assessment Guidance for Superfund: Volume I. Human Health Evaluation Manual (Part A).* Office of Emergency and Remedial Response. Washington, D.C., December 1989. EPA/541/1-89/002. Available at: http://www.epa.gov/superfund/programs/risk/ragsa/index.htm.

PART III

HUMAN HEALTH RISK ASSESSMENT: MULTIPATHWAY

Chapter 14 Overview and Getting Started: Planning and Scoping the Multipathway Risk Assessment

Table of Contents

14.1 Introduction 1

14.2 Overview of Multipathway Air Toxics Risk Assessment 3
- 14.2.1 Planning, Scoping, and Problem Formulation 3
- 14.2.2 Analysis 4
- 14.2.3 Risk Characterization 5

14.3 Overview of Multipathway Exposure Assessment 5

14.4 Planning and Scoping 9
- 14.4.1 Identifying the Concern 9
- 14.4.2 Identifying The Participants 9
- 14.4.3 Determining the Scope of the Risk Assessment 9
- 14.4.4 Describing the Problem 10
- 14.4.5 Determining How Risk Managers Will Evaluate the Concern 10

14.5 Tiered Multipathway Risk Assessments 10

References 11

14.1 Introduction

Part II of this Reference Manual discussed how to plan for and conduct a human health risk assessment via the direct inhalation pathway. Part III provides the same general discussion of the various aspects of the risk assessment process; however, the discussion is focused specifically on multipathway human health risk assessment. ***As noted earlier, all air toxics risk assessments evaluate the direct inhalation pathway. In addition, multipathway risk assessment may be appropriate generally when air toxics that persist and which also may bioaccumulate and/or biomagnify are present in releases. These generally will focus on the persistent bioaccumulative hazardous air pollutant (PB-HAP) compounds (Exhibit 14-1), but specific risk assessments may need to consider additional chemicals that persist and which also may bioaccumulate and/or biomagnify***. For these compounds, the risk assessment generally will need to consider exposure pathways other than inhalation – in particular, pathways that involve deposition of air toxics onto soil and plants and into water, subsequent uptake by biota, and potential human exposures via consumption of contaminated soils, surface waters, and foods. Substances that persist and bioaccumulate readily transfer between the air, water, and land. Some may travel great distances, and linger for long periods of time in the environment.

The discussion of multipathway risk assessment follows the same general framework presented in Part II. This chapter presents an overview of multipathway risk assessment and discusses the initial planning, scoping and problem formulation activities. The remaining chapters of this Part focus on Exposure Assessment (Chapters 14 to 20), Toxicity Assessment (Chapter 21), and Risk Characterization (Chapter 22). The discussions presented here supplement the information provided earlier – readers are encouraged to refer back to the corresponding Chapters in Part II for additional background materials.

Bioconcentration is the net accumulation of a substance by an organism as a result of uptake directly from an environmental medium (e.g., net accumulation by an aquatic organism as a result of uptake directly from ambient water, through gill membranes or other external body surfaces).

Bioaccumulation is the net accumulation (storage in tissue and/or organs) of a substance by an organism as a result of uptake from all environmental sources – the medium in which they live, the water they drink, and the diet they consume – over a period of time.

Biomagnification or Biological Magnification is the process whereby certain substances, such as pesticides or heavy metals, transfer up the food chain and increase in concentration. A biomagnifying chemical deposited in rivers or lakes absorbs to algae, which are ingested by aquatic organisms, such as small fish, which are in turn eaten by larger fish, fish-eating birds, terrestrial wildlife, or humans. The chemical tends to accumulates to higher concentration levels with each successive food chain level. Biomagnification is illustrated in Chapter 23.

Exhibit 14-1. PB-HAP Compounds			
PB-HAP Compound	**Pollution Prevention Priority PBTs**	**Great Waters Pollutants of Concern**	**TRI PBT Chemicals**
Cadmium compounds		X	
Chlordane	X	X	X
Chlorinated dibenzodioxins and furans	X[a]	X	X[b]
DDE	X	X	
Heptachlor			X
Hexachlorobenzene	X	X	X
Hexachlorocyclohexane (all isomers)		X	
Lead compounds	X[©]	X	X
Mercury compounds	X	X	X
Methoxychlor			X
Polychlorinated biphenyls	X	X	X
Polycyclic organic matter	X[d]	X	X[e]
Toxaphene	X	X	X
Trifluralin			X

[a] "Dioxins and furans" ("" denotes the phraseology of the source list)
[b] "Dioxin and dioxin-like compounds"
[c] Alkyl lead
[d] Benzo[a]pyrene
[e] "Polycyclic aromatic compounds" and benzo[g,h,i]perylene
See Appendix D for a discussion of the derivation of this list of PB-HAPs.

14.2 Overview of Multipathway Air Toxics Risk Assessment

The multipathway risk assessment is organized in the same way as the direct inhalation risk assessment into three general phases:

1. Planning, scoping, and problem formulation;
2. Analysis, consisting of exposure assessment and toxicity assessment; and
3. Risk characterization.

14.2.1 Planning, Scoping, and Problem Formulation

The planning, scoping, and problem formulation phase of multimedia risk assessment focuses on developing a common understanding of what needs to be added to the risk assessment (beyond the direct inhalation assessment) to assess risks associated with pathways involving deposition (i.e., transfer of the compounds to soil, water, sediment, and biota) and subsequent ingestion exposure. The scope of the multimedia risk assessment generally is more extensive than that for inhalation assessment, and therefore significant additional effort is likely.

For purposes of this Reference Manual, we discuss planning, scoping, and problem formulation for multipathway human health risk assessment separately from the corresponding phase for inhalation risk assessment. In reality, the planning, scoping, and problem formulation phase for the multipathway assessment would be integrated with the inhalation analysis as early as feasible.

It may be necessary to include on the **planning and scoping team** experts in multimedia modeling, bioaccumulation, human exposure factors, and ingestion toxicology. The focus on additional exposure pathways may influence many aspects of the risk assessment, including the size of the study area; emission sources to be considered; the temporal and spatial resolution required; the appropriate level of detail and documentation; trade-offs between depth and breadth in the analysis; QA/QC requirements; analytical approaches to be used; and the staff and monetary resources to commit. The **study-specific conceptual model** would also reflect the specific concerns of air toxics that persist and which also may bioaccumulate. As with the inhalation risk assessment, the planning, scoping, and problem formulation process is an iterative process that reflects changing information and concerns as the multimedia risk assessment unfolds.

The reader should become familiar with Part II of this manual before reading this Part, since Part III focuses primarily on those aspects of the risk assessment that are unique to multipathway analyses, including:

- How the study area is defined;
- Potentially exposed populations;
- Exposure pathways and exposure routes;
- How exposure is assessed;
- Dose-response values for non-inhalation pathways; and
- How risks are characterized.

14.2.2 Analysis

The analysis phase of the multipathway assessment is divided into two components: exposure assessment and toxicity assessment. **Exposure assessment** is likely to be considerably more complicated than the corresponding inhalation exposure assessment for several reasons:

- People can be exposed to air toxics in many more ways, including in the food they eat, the milk they drink, and the soils on which they play.

- Time is a critical variable. Air toxics that persist and which also may bioaccumulate can slowly build up in soils, sediments, and biota over time. With sufficient time, even relatively small releases have the potential to result in high exposures.

- The spatial distribution of the air toxics can be complex. Chemicals can move away from deposition points due to runoff, erosion, and the movement of contaminated animals. Chemicals deposited over a wide area (e.g., a watershed) can concentrate in smaller areas (e.g., a pond).

- Multimedia models often use more extensive input variables.

- Sampling and analysis may involve a wider range of media (e.g., soil, sediment) and different types of biota (e.g., fish, shellfish, plants). Each type of sampling and analysis has its own methods, protocols, and QA/QC procedures.

- Whereas the exposure concentration in air is the quantitative metric of exposure for inhalation, intake is the quantitative metric of ingestion exposure in multipathway analyses. To quantify intake, it is necessary to (1) estimate the concentrations of chemicals of potential concern (COPC) in water, soil, sediment, and/or food items; (2) determine how much water, soil, sediment, and food are ingested; (3) determine the duration and temporal patterns over which ingestion occurs; and (4) adjust for body weight, to account for the different types of people in the population who interact with the contaminated media. Multimedia exposure assessment uses a number of different **exposure factors** that provide quantitative estimates of the physical and behavioral attributes of potentially exposed populations (e.g., how much fish a person eats per day). Exposure factors can be treated as either constants or variables in the exposure assessment, depending on whether a deterministic or probabilistic analysis is being performed.

The multipathway **toxicity assessment** is similar to the toxicity assessment for inhalation. It considers the same general information: (1) the types of potential adverse health effects associated with chemical exposures; (2) dose-response relationships; and (3) related uncertainties such as the weight of evidence for carcinogenic effects. There are two primary differences:

- A chemical's toxicity is influenced by the route of exposure. That is, the same chemical can result in different toxic effects (and have different dose-response values) depending on whether the chemical is inhaled or ingested. There are a number of reasons why this may occur. For example, when a chemical is inhaled into the respiratory tract, the primary toxic effect may occur in the respiratory tract as a result of the inhaled chemical (a portal of entry effect). When swallowed, on the other hand, many chemicals are absorbed into the

bloodstream through the gastrointestinal tract where they are carried directly to the liver. Chemicals in the liver are often metabolized extensively (either to more or less toxic substances) before being transported by the bloodstream to other parts of the body.

- The specific dose-response values used for the ingestion pathway – reference doses (RfDs) for non-cancer effects and oral cancer slope factors (CSFs) – differ in form and derivation from those used for inhalation assessments. Specifically, RfDs and CSFs are developed to match the metric of exposure for ingestion and are expressed (usually) in terms of amount of chemical ingested per unit of body weight per day (i.e., mg/kg-d for RfDs) and risk per amount of chemical ingested per unit body weight per day (i.e., $(mg/kg\text{-}d)^{-1}$ for CSFs).

14.2.3 Risk Characterization

The risk characterization for multipathway assessments also may be more complicated than that for the inhalation risk assessment.

- Ingestion risk estimates are first added across all ingestion pathways and then added to inhalation risk estimates to calculate total (i.e., cumulative) risk. Although the summation process is relatively simple for screening-level analyses, it can become complex for more advanced tiers of risk assessment.

- The uncertainty analysis for multipathway risk assessments may be considerably more complex if multiple pathways are important because many more exposure factors and variables will be involved in the quantification of risk. As noted earlier, many more specific exposure factors can be treated as variables for probabilistic multipathway risk assessments.

- The uncertainty analysis for multipathway analysis is also much more complex due to the larger number of pathways assessed and the larger number of measurement and modeling inputs that are needed.

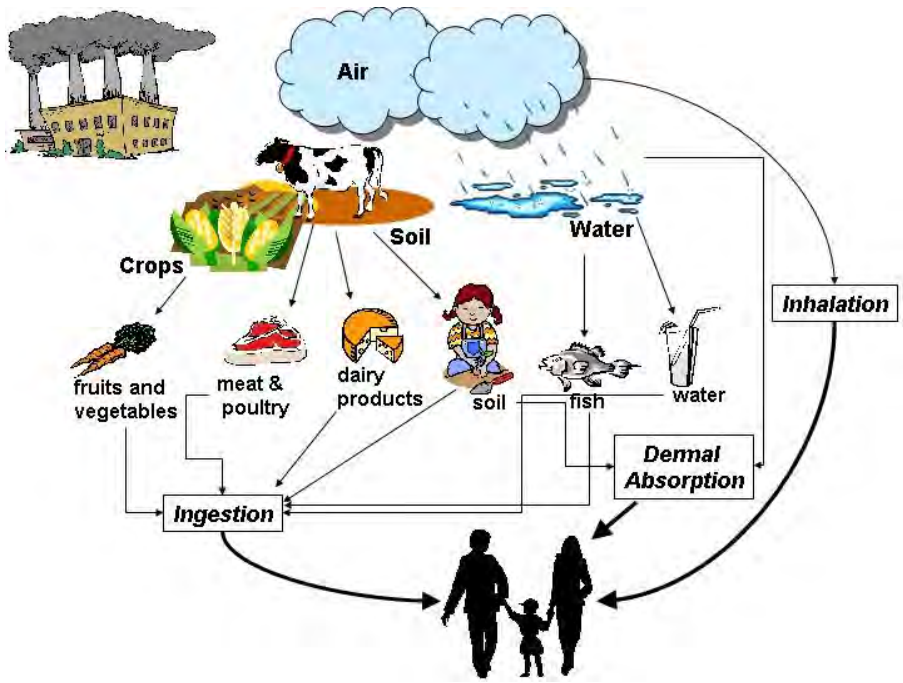
14.3 Overview of Multipathway Exposure Assessment

As with inhalation risk assessments, the exposure assessment for multipathway risk assessments includes identifying sources, characterizing releases to the air, estimating concentrations of air toxics in the environment, characterizing potentially exposed populations, and developing metrics of exposure. This section provides an overview of exposure assessment for multipathway risk assessments. Familiarity with EPA's *Guidelines for Exposure Assessment*[1] prior to beginning the multipathway exposure assessment would be helpful.

The multipathway exposure assessment covers a broader scope and may be more complex than direct inhalation exposure assessment.

- Exposure pathways to be evaluated include multiple media (soil, water, sediment, biota) and exposure routes in addition to inhalation (e.g., ingestion). Therefore, the exposure setting may need additional characterization (e.g., the location and nature of water bodies and/or agricultural crops).

Overview of Multipathway Exposure Pathways/Routes



- The evaluation of chemical fate and transport accounts for the transfer of contaminants from air to soil and water and subsequent transport and transfer to other media. For example, air toxics that persist and which also may bioaccumulate are deposited onto soils and can enter surface waters via runoff; some of the compounds that deposit into water predominantly partition into sediments. **Bioaccumulation** – a concentration of contaminants in biological tissues – and subsequent transfer to humans via ingestion often play a major role in the exposure assessment. Multimedia models can be used to describe contaminant fate and transport through the use of partition coefficients and mass-balance techniques (see Chapter 6). Different monitoring methods (e.g., sediment or fish tissue sampling and analysis) may be included to augment or assist in the evaluation of modeling outputs.

- In contrast to the direct inhalation assessment, in which the quantitative metric of exposure is the ambient air concentration at the exposure point, ingestion exposures are quantified using the **chemical intake rate** – the amount of chemical ingested per unit time – generally expressed in units of milligrams of chemical per kilogram of body weight per day. Calculation of chemical intake rate requires information on COPC concentrations in items ingested as well as information about the type and amount of different items eaten each day, body weight, and exposure durations for the sub-populations of interest. **Intake rate** is simply the amount of food (or other media), containing the contaminant of interest, that an individual ingests during some specific time period (units of mass/time). Intake rate can be expressed as a total amount (e.g., mg); as a dose rate (e.g., mg/day); or as a rate normalized to body mass (e.g., mg/kg-day). For most chemicals, the dose-response value (e.g., reference dose, or RfD) is based on the potential dose (i.e., the amount of chemical taken in), with no

explicit correction for the fraction absorbed. For some chemicals, it may be necessary to adjust for such differences using physiologically based pharmacokinetic (PB-PK) models, mathematical dosimetry models, and/or adjustment factors (see Chapter 8).

Because exposure is quantified using chemical intake rate, different types of people within a population (e.g., childhood exposures) may need to be considered explicitly. Consumption rates, dietary preferences, and body weight vary with age and would be accounted for in the risk assessment. (Note that not only age, but sex, ethnicity, cultural and religious practices may also strongly influence the exposure patterns of people within a potentially exposed population.)

Although it is possible to evaluate acute exposures for the ingestion pathway, EPA does not generally perform acute exposure assessments, because it is unlikely that PB-HAP compounds would concentrate to acutely toxic levels under any typical release scenario that did not pose a much more substantial chronic risk. However, each assessment would consider the available evidence in making this judgement. At a minimum, the risk characterization would state the reasons why an analysis of acute health effects for non-inhalation pathways was not performed.

The multipathway exposure assessment focuses on two general categories of ingestion pathways: incidental ingestion and food chain (Exhibit 14-2). Incidental ingestion pathways consider exposures that may occur from ingestion of soils or surface water while an individual is engaged in other activities (e.g., ingestion of soil while gardening or playing outside; ingestion of surface water while swimming). Food chain pathways consider exposures that may occur if PB-HAP compounds accumulate in the food and water people consume.

Exhibit 14-2. Human Exposure Pathways Considered for Multipathway Air Toxics Assessments

Incidental Ingestion Pathways:
air → soil → human
air → surface water → human
Food Chain Pathways:
air → water → human
air → soil → water → human
air → vegetation → human
air → animal → human
air → vegetation → animal → human
air → soil → vegetation → human
air → soil → animal → human
air → soil → water → animal → human
air → soil → water → fish → human
air → soil → water → sediment → fish → human
air → water → animal → human
air → water → fish → human
air → water → sediment → fish → human

As Exhibit 14-2 suggests, the focus of the multipathway assessment is on ingestion pathways. Other exposure pathways may be important for particular risk assessments, including dermal exposures (i.e., direct contact with contaminated soils, surface waters, or surface water sediments during outside activities such as gardening or swimming); resuspension of dust (e.g., from wind blowing across contaminated soils, or agricultural activities such as tilling) and subsequent inhalation of the dust particles; and ingestion of contaminated groundwater. However, EPA does not have sufficient experience with multipathway air toxics risk assessments to identify the circumstances for which exposures via these additional pathways may represent a potential concern.

- If site-specific circumstances suggest that dermal pathways may be of concern, EPA's *Risk Assessment Guidance for Superfund (RAGS), Part D, Standardized Planning, Reporting and Review of Superfund Risk Assessments*,[2] includes a relatively straightforward methodology for dermal exposure and risk assessment, starting with soil concentrations. The Planning Tables in the document are simple to use and incorporate into the multipathway analysis.

- Relative to the direct inhalation pathway, inhalation of soil resulting from dust resuspension by wind erosion generally is not thought to be a significant pathway of concern for air toxics risk assessments. If site-specific circumstances suggest that resuspension of dust may represent a potential concern, EPA's *Methodology for Assessing Health Risks Associated with Multiple Pathways of Exposure to Combuster Emissions* (MPE) (Chapter 5 Dust Resuspension) discusses the methods for evaluating this pathway.[3]

Analysis of Groundwater Pathways

EPA's Office of Solid Waste has considerable experience in modeling and monitoring the movement of contaminants in groundwater. Much of that experience is based on exposure assessments associated with land-based disposal units (i.e., where the source of contamination is in the subsurface). For example, EPA's Center for Exposure Assessment Modeling (CEAM) distributes multimedia models designed to quantify the movement and concentration of contaminants (from land-based releases at hazardous waste sites) traveling through groundwater, surface water, and food chain media (available at http://www.epa.gov/ceampubl/). In these models, releases to the atmosphere from the subsurface may be considered, but transfer from the air through the subsurface are not.

EPA does not have sufficent experience with air toxics multipathway analysis to identify situations in which the groundwater may be contaminated. EPA's *Methodology for Assessing Health Risks Associated with Multiple Pathways of Exposure*[3] identifies three site-specific conditions that might lead to greater groundwater impacts:

- Deposition rates that are several times greater than the average;
- The existence of more soluble HAPs in emissions; and
- Higher recharge rates such as would occur in areas with very permeable soil and bedrock near the surface.

- If site-specific circumstances suggest that groundwater may represent a potential concern (e.g., the presence of extremely shallow aquifers used for drinking water purposes or a karst environment in which the local surface water significantly affects the quality of ground water used as a drinking water source), Total Risk Integrated Methodology - Fate, Transport, and

Ecological Exposure Module (TRIM.FaTE) has the ability to assess chemicals moving into the groundwater pathway. EPA's *Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities*[3] and EPA's *Draft Technical Background Document for Soil Screening Guidance* discusses methods for evaluating the groundwater pathway.

14.4 Planning and Scoping

As with inhalation analyses, the key steps in the planning and scoping process include (1) identifying the concern; (2) identifying who will be involved; (3) determining the scope of the risk assessment; (4) describing why there may be a problem; and (5) determining how the concern will be evaluated. The planning and scoping process for multipathway risk assessment focuses on developing a common understanding of what needs to be evaluated to assess risks via deposition and transfer of the air toxics to soil, water, and biota, and subsequent ingestion. More detailed discussions of the planning and scoping process can be found in Part II of this Volume and in guidance documents developed by EPA.[4]

14.4.1 Identifying the Concern

The driving concern for the multipathway risk assessment generally would be the same as that for the inhalation risk assessment (e.g., regulatory requirement, community need, health concern). However, a number of additional specific concerns may arise. For example, the potential for bioaccumulation in food and subsequent ingestion may raise specific concerns about areas where people farm, economic issues such as recreational fishing, or additional exposure pathways of potential concern (e.g., infants ingesting mother's milk).

14.4.2 Identifying the Participants

The participants for the multipathway risk assessment generally would be the same as those for the inhalation risk assessment. However,

- A broader range of risk managers would be involved. For example, if there is a potential for a fishery or farm crops to become contaminated with air toxics, different persons or groups may have the authority to make the risk management decisions – the state, local, or tribal (S/L/T) fish and game department or the agriculture department may become involved.

- The risk assessment technical team would include additional experts (e.g., in the areas of multimedia modeling, bioaccumulation, soil chemistry).

- The specific set of interested or affected parties may change or expand (e.g., farmers and fishermen may be more concerned/involved).

14.4.3 Determining the Scope of the Risk Assessment

At a minimum, the scope of the risk assessment will include additional exposure pathways, exposure routes, and potentially exposed populations or sub-populations. The details of scope are developed during the problem formulation step (see Chapter 15).

14.4.4 Describing the Problem

As with inhalation, participants would develop a **problem statement** that clearly articulates the perceived problem to be evaluated. The problem statement may also provide statements of what is and is not included in the multipathway risk assessment and why. (Note that, in general, only one problem statement is necessary to describe all exposure pathways, including inhalation. A separate problem statement for each exposure pathway is not usually necessary.)

14.4.5 Determining How Risk Managers Will Evaluate the Concern

As with inhalation, the multipathway risk assessment would be designed to provide input to risk managers to help inform the decisions they must make. Part of the planning and scoping process is developing an understanding of the types of information needed by the risk managers and the level of uncertainty in that information that can be tolerated.

Example Multipathway Problem Statement

Air toxics emissions may be causing increased long-term health risk to people who eat fish in Puffer Pond that may be contaminated with mercury compound releases from the Big Air Manufacturing Company. A multipathway risk assessment will be performed to evaluate potential long-term human health impacts associated with consumption of contaminated fish. Ingestion risks will be assessed for recreational fishers who eat fish caught in Puffer Pond. In addition, a modeling risk assessment using air dispersion modeling will be conducted to estimate inhalation risks for populations within 50 km of the Acme property boundary using residential exposure conditions.

14.5 Tiered Multipathway Risk Assessments

EPA guidance generally recommends that a tiered approach to risk assessments be taken to identify the key chemicals, sources, and pathways that contribute most to the risk being evaluated.[(5)] A tiered approach can be particularly valuable for multipathway risk assessments because of the potential complexity commonly associated with such analyses. Often, screening-level analyses assume relatively high exposure factors (e.g., all of the fish a person eats comes from a potentially contaminated pond) to determine whether risk associated with a specific pathway appears to be significant enough to warrant more robust analysis. Subsequent tiers of analysis, using more realistic exposure factors and perhaps involving more complex modeling and perhaps sampling and analysis, are generally undertaken only if lower-tier analyses continue to indicate the potential for risk. As with inhalation risk assessments, an iterative process of evaluation, deliberation, data collection, work planning and communication is used to decide:

- Whether or not the risk assessment, in its current state, is sufficient to support the risk management decision(s); and

- If the assessment is determined to be insufficient, whether or not progression to a higher tier of complexity (or refinement of the current tier) would provide a sufficient benefit to warrant the additional effort.

References

1. U.S. Environmental Protection Agency. 1992. *Guidelines for Exposure Assessment. Federal Register* 57:22888-22938, May 29, 1992. Available at http://cfpub.epa.gov/ncea/raf/recordisplay.cfm?deid=15263

2. U.S. Environmental Protection Agency. 2001. *Risk Assessment Guidance for Superfund (RAGS): Volume I - Human Health Evaluation Manual (Part D, Standardized Planning, Reporting and Review of Superfund Risk Assessments), Final.* Office of Emergency and Remedial Response, Washington, D.C., December 2001. EPA 9285.7-47.

3. U.S. Environmental Protection Agency. 1998. *Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities, Peer Review Draft.* Office of Solid Waste and Emergency Response, July 1998. EPA/530/D-98/001 A, B, and C and subsequent Errata (EPA Memo, July 1999). Available at: http://www.epa.gov/earth1r6/6pd/rcra_c/pd-o/comb_risk.htm

4. U.S. Environmental Protection Agency. 1997. *Guidance on Cumulative Risk Assessment. Part 1. Planning and Scoping.* Science Policy Council, Washington, D.C., July 3, 1997. Available at: http://www.epa.gov/osp/spc/cumrisk2.htm

   U.S. Environmental Protection Agency. 2003. *Framework for Cumulative Risk Assessment.* Office of Research and Development, National Center for Environmental Assessment, Washington, D.C., 2003. EPA/600/P-02/001F. Available at: http://cfpub.epa.gov/ncea/raf/recordisplay.cfm?deid=54944

   U.S. Environmental Protection Agency. 2001. *Risk Assessment Guidance for Superfund (RAGS): Volume I - Human Health Evaluation Manual (Part D, Standardized Planning, Reporting and Review of Superfund Risk Assessments) Final.* Office of Emergency and Remedial Response, Washington, D.C., December 2001. Publication 9285.7-47. Available at: http://www.epa.gov/superfund/programs/risk/ragsd/index.htm

5. U.S. Environmental Protection Agency. 2001. *Risk Assessment Guidance for Superfund (RAGS). Volume III - Part A. Process for Conducting Probabilistic Risk Assessment.* Office of Emergency and Remedial Response, Washington, D.C., December 2001. EPA/540/R-02/002. Available at: http://www.epa.gov/superfund/programs/risk/rags3a/index.htm

Chapter 15 Problem Formulation: Multipathway Risk Assessment

Table of Contents

15.1 Introduction 1

15.2 Developing the Multipathway Conceptual Model 1

15.3 Developing the Multipathway Analysis Plan 2
- 15.3.1 Identification of the Sources 3
- 15.3.2 Identification of the Chemicals of Potential Concern 3
- 15.3.3 Identification of the Exposure Pathways/Routes 3
  - 15.3.3.1 Characteristics of the Assessment Area 4
  - 15.3.3.2 Scale of the Assessment Area 5
  - 15.3.3.3 Use of Modeling vs. Monitoring 5
  - 15.3.3.4 Quantitation of Exposure 6
  - 15.3.3.5 Evaluation of Uncertainty 7
  - 15.3.3.6 Preparation of the Documentation 7
- 15.3.4 Identification of the Exposed Population 7
- 15.3.5 Identification of Endpoints and Metrics 7

15.4 Exposure Assessment Approach 7
- 15.4.1 Scenario Approach 8
- 15.4.2 Population-Based Approach 11

References 12

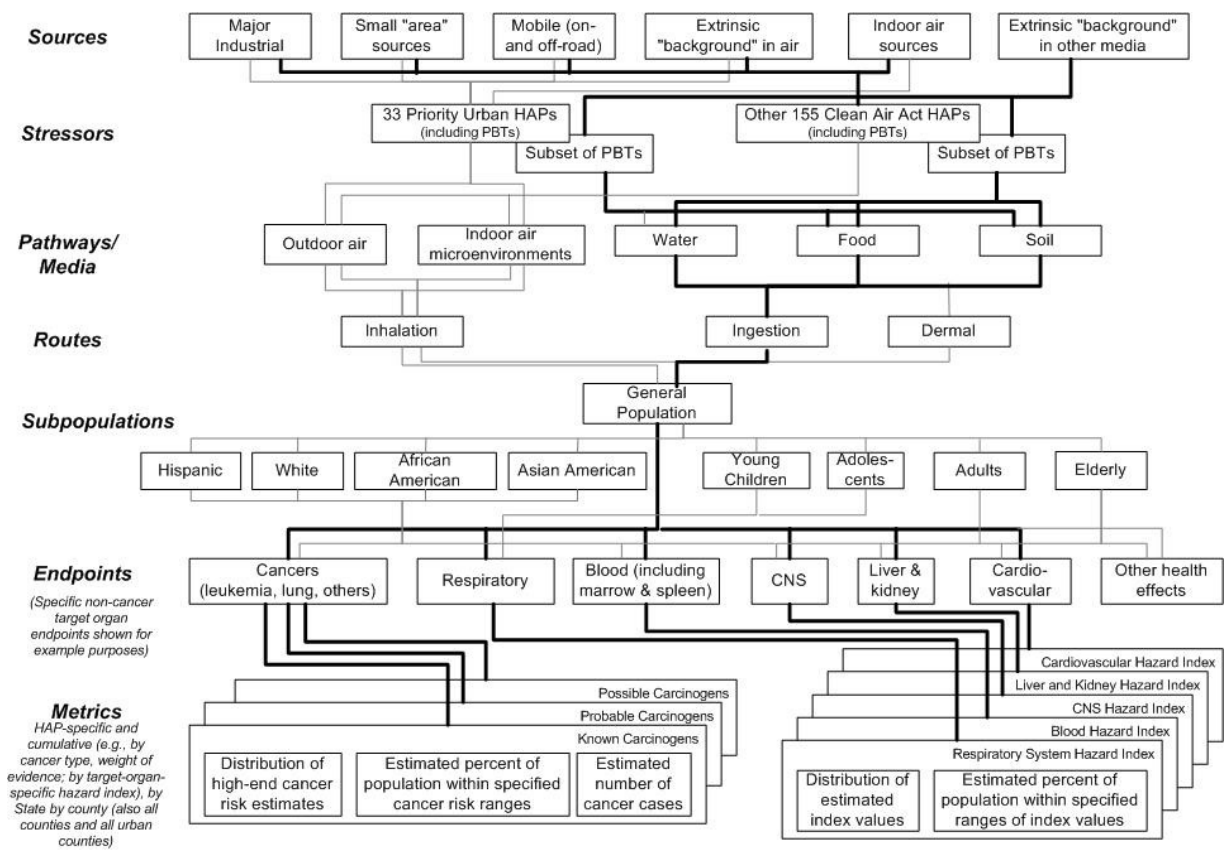
15.1 Introduction

This chapter discusses the problem formulation step of the multipathway risk assessment, which takes the results of the planning and scoping process and translates them into two critical products: the conceptual model, and the analysis plan.

15.2 Developing the Multipathway Conceptual Model

As with inhalation analyses, the conceptual model (Exhibit 15-1) focuses the multipathway risk assessment on several key elements, including sources, chemicals released, fate and transport mechanisms, potentially exposed populations, potential exposure pathways and routes of exposure, and potential adverse effects. Although discussed separately here, as noted in Chapter 6, ***the elements of the conceptual model that are unique to the multipathway human health risk assessment should be integrated with those for the inhalation assessment as early as feasible***.

Exhibit 15-1. Generalized Conceptual Model for Multipathway Analyses



This figure highlights the multipathway components of the general air toxics risk assessment conceptual model introduced in Chapter 6. The conceptual model for a specific multipathway risk assessment may consider only part of this general model, or may focus more closely on specific sub-populations of concern.

Elements of the conceptual model that may be unique to the multipathway assessment include:

- **Sources**. The specific sources included in the analysis may be focused on the subset of all sources that release most or all of the identified air toxics that persist and which also may bioaccumulate.

- **Chemicals of potential concern**. The specific COPC will focus on those air toxics that persist and which also may bioaccumulate (i.e., persistent bioaccumulative hazardous air pollutants (PB-HAPs) and other non-HAP chemicals that may be of concern for persistence and bioaccumulation).

- **How the COPC move through the environment**. The conceptual model will need to consider the mechanisms by which PB-HAPs move through the environment, which include dispersion in the air; deposition (including vapor phase transfer) to soils, surface waters, and plant surfaces; erosion and other runoff phenomena; and uptake and bioconcentration by biota. The physical boundaries of the study area may need to include geographic areas where COPC may be transported after deposition (e.g., PB-HAPs may have the potential to be deposited in a watershed and be carried out of the geographic area defined for the inhalation pathway modeling).

- **The exposure pathways/media of concern**. The potential exposure pathways will include a number of different ingestion pathways and, in some cases, dermal absorption pathways.

- **The human populations potentially receiving exposure**. The potentially exposed populations may need to include persons who do not live within the study area but consume food products that have the potential to become contaminated (e.g., recreational fisher). Additionally, different sensitive sub-populations may be identified (e.g., people who consume large amounts of locally-caught fish because of cultural reasons).

- **The potential adverse health effects (endpoints) that may result from exposure**. The general types of chronic health risks (cancer, non-cancer) may or may not change, depending on the specific COPC being evaluated. However, acute exposures generally are not a concern for multipathway analyses because it would be unlikely for air toxics to accumulate in soil, sediment, or food items to concentrations that would pose, in the absence of a chronic hazard, an acute hazard through the ingestion or dermal pathway.

- **Metrics**. The metrics used to characterize exposure and estimate risk may or may not be different from those used in the inhalation risk assessment. For example, the inhalation assessment may stop at a Tier 1 analysis, while the multipathway assessment may go all the way to a Tier 3 analysis.

15.3 Developing the Multipathway Analysis Plan

As noted in Chapter 6, the analysis plan matches each element of the conceptual model with the analytical approach that the assessor will use to develop data about that element. This section describes the elements of the analysis plan that are unique to the multipathway assessment, including (1) identification of sources; (2) identification of COPC; (3); identification of exposure

pathways/routes; (4) identification of exposed populations; and (5) identification of endpoints and metrics.

15.3.1 Identification of the Sources

This part of the analysis plan identifies the sources to be included in the risk assessment. As noted earlier, the focus of multipathway analysis is on sources of the air toxics that persist and which also may bioaccumulate. Within that subset, certain sources may be most important for a specific risk assessment. A tiered approach is recommended for focusing the risk assessment from the initial set of sources to the sources that will drive risk management decisions. The initial tier of analysis generally includes all sources of PB-HAPs. In subsequent tiers of analysis, it may be possible to remove specific sources from the analysis that contribute a very small fraction to the total risk estimate.

15.3.2 Identification of the Chemicals of Potential Concern

This part of the analysis plan identifies the chemicals that will be evaluated in the risk assessment. As noted earlier, the focus of multipathway analysis is on the subset of air toxics that persist and which may also bioaccumulate. Within that subset, certain chemicals may be most important for a specific risk assessment. A tiered approach is recommended for focusing the risk assessment from the initial list of COPC to the set of contaminants that will drive risk management decisions. The initial tier of analysis generally includes all of the air toxics released from the identified important sources. In subsequent tiers of analysis, it may be possible to remove specific chemicals from the analysis if they contribute a very small fraction to the total risk estimate.

15.3.3 Identification of the Exposure Pathways/Routes

This part of the analysis plan identifies the exposure pathways/routes to be evaluated. As noted in Chapter 6, an exposure pathway consists of four elements:

- A source and mechanism of chemical release;
- One or more environmental media (i.e., air, water, soil) in which the chemical is transported from the source;
- A point of potential human contact with the contaminated medium (referred to as the exposure point); and
- An exposure route (e.g., inhalation, ingestion) at the contact or exposure point. The route may be actual or potential, depending on the purpose of the assessment.

The exposure pathway is complete if all four elements can be identified; otherwise the exposure pathway is incomplete and not considered further (see Exhibit 14-2, which presents the potential exposure pathways considered for multipathway assessments).

The exposure points selected for the multipathway risk assessment also will depend on the choice of multipathway assessment approach and may or may not be identical to those used in the inhalation risk assessment. In the example presented in Chapter 11, Mr. McDonald's house was selected as the point of maximum inhalation concentration at a receptor location. The multipathway assessment would likely also evaluate potential exposures via crops, meat, milk,

and other foods. However, the focus would be on other exposure points or areas within the farm (e.g., the area where forage fed to dairy cows is grown or where vegetable crops are planted), not on the farmer's house.

15.3.3.1 Characteristics of the Assessment Area

The physical setting is important both in developing the study-specific conceptual model and selecting and providing input parameters for the appropriate multimedia models. As described earlier, the physical setting includes information such as urban vs. rural setting, simple vs. complex terrain, climate and meteorology, and other important geographic features (see Chapter 6). The most important additional information required for multipathway analyses is information on land use, soils, and surface water bodies within the assessment area.[(a)] Many of the general physical characteristics of the setting will influence the scope of multimedia modeling required. For example, if the sources being evaluated are located in heavily industrialized area, there may be few, if any, agricultural areas or water bodies close enough to receive significant deposition. In this example, deposition to soils in nearby residential areas and subsequent exposure pathways may be the most significant exposure pathways to examine.

- **Land use**. Information on land use is an important part of the physical characteristics of the assessment area discussed in Chapter 6. For multipathway analyses, it is important to identify specific types of land uses that may lead to exposures via ingestion pathways, especially agriculture, fishing, recreation, and residential (indoor and outdoor, including gardening), as well as the location of particular areas where exposures via soil may be of concern (e.g., playgrounds, schools, day care centers). Sources for land use data are discussed in Chapter 6.

- **Soils**. The type and characteristics of soils (e.g., sandy, organic, acidic, alkaline) in the assessment area affects physical phenomena such as soil erosion rates, the types and density of plants supported by the soils, and the physical and chemical characteristics that govern contaminant fate and transport. For example, the bioavailability of a compound may depend partially on soil pH. The specific information needed will depend in part on the input requirements of the multimedia fate and transport models selected for the analysis (see Chapter 19).[(b)]

- **Water bodies and their associated watersheds**. Water bodies and their associated watersheds are important factors in evaluating some of the major exposure pathways/routes considered in multipathway analyses. For example, the identification of surface water bodies at locations in the assessment area receiving deposition from emission sources indicates the potential for exposures to contaminants from ingestion of fish, and possibly drinking water (drinking water is usually evaluated only if the local population obtains drinking water from

---

[a]Maps, aerial photos, and tools such as Geographic Information Systems (GIS) can be very helpful tools for characterizing the exposure setting (see Part VI of this Reference Manual).

[b]Sources of this information may include any existing site descriptions, preliminary risk assessments, county soil surveys, wetlands maps, aerial photographs, U.S. Geological Survey topographic maps, U.S. Department of Agriculture Soil Conservation Service reports, and information from state natural resources agencies.

surface water sources).[c] Information on fishing activity will also be useful in characterizing the potentially exposed population.

Land use and human activities should be characterized in much the same way as Chapter 6 described, except that a broader range of activities/uses needs to be considered. It is important to identify all activities within the assessment area that could result in exposure to contaminants via non-inhalation pathways. These would include hunting, fishing, growing crops (e.g., commercially, as animal feed, or for private consumption), and incidental ingestion of soils. As noted earlier, the multipathway assessment may need to specifically address special populations that are located in impacted areas because of unique characteristics of the exposure setting or to address particular community concerns. For example, a day care center or traditional Tribal fishing/hunting area may be located in an area that is impacted by releases from a facility or source area. Consequently, due to the site-specific exposure characteristics, exposure to children at the day care center or tribal members may need to be addressed, because they may be especially sensitive to the adverse effects and/or the exposure setting may be particularly conducive to exposure. EPA has developed a policy focused on consistently and explicitly evaluating environmental health risks to infants and children in all risk assessments.[1]

15.3.3.2 Scale of the Assessment Area

For inhalation assessments, the study area generally is limited to a 50-km radius from the emissions sources (based on the dispersion models being used). The study area for the multipathway risk assessment generally will be limited similarly to the area in which deposition is modeled. However, certain potential exposure scenarios may require expansion of the study area beyond the modeled deposition area. Examples include:

- The watershed for a lake or pond is within the modeled deposition area, but the lake or pond (where contaminants may accumulate) is outside the deposition area.
- A commercial farm is within the deposition area, and a portion of the crops are consumed by persons living outside the deposition area.
- A popular fishing area is located within the deposition area, and people from outside the deposition area come there to fish.

15.3.3.3 Use of Modeling vs. Monitoring

As this document has previously noted, risk assessors can base estimates of current exposure concentrations on either actual

Multimedia Assessments: Modeling vs. Monitoring

Most multimedia air toxics risk assessments will develop estimates of exposure concentration for non-inhalation pathways primarily through modeling. In some instances, analysts may use monitoring to evaluate the model. In more rare instances, however, analysts will use monitoring to develop exposure concentrations.

[c] Use, area, and location of water bodies and their associated watersheds can typically be identified by reviewing the same land-use land classification maps, topographic maps, and aerial photographs used in identification of land use discussed in Part II of this Reference Manual. Additional information on water body use can also be obtained through discussions with local authorities (e.g., state environmental agencies, fish and wildlife agencies, or local water control districts) about viability to support fish populations and drinking water sources, or current postings of fish advisories.

measurements (i.e., monitoring data) or modeling (in this case, multimedia models). In many cases, monitoring can be helpful in reducing uncertainties in the exposure assessment, because multimedia modeling is more complex and involves more uncertainties. Note, however, that the scope of potential monitoring for multipathway analysis is considerably greater than that for inhalation analyses. A wide range of types of sampling and analysis could be conducted, including sampling of soils, surface waters, sediments, and biota (human food items). Each type of sample has its own methods, protocols, and QA/QC requirements (see Chapter 19). Multimedia sampling and analysis may require additional expertise and effort. The analysis plan, including the quality assurance protection plan (QAPP), will need to be modified accordingly.

15.3.3.4 Quantitation of Exposure

In contrast to the inhalation assessment, in which the quantitative metric of exposure is the ambient air concentration at the exposure point, ingestion exposures are quantified using the **chemical intake rate** – the amount of chemical ingested per unit time – generally expressed in units of milligrams of chemical per kilogram of body weight per day. The fundamental equation for dietary intake and ingestion pathways in general is given as:

$$I = \frac{EC \times CR}{BW} \times \frac{EF \times ED}{AT} \qquad \text{(Equation 15-1)}$$

where

I	=	Chemical intake rate, expressed in units of mg/kg-day. For evaluating exposure to non-carcinogens, the intake is referred to as average daily dose (*ADD*); for evaluating exposure to carcinogenic compounds, the intake is referred to as lifetime average daily dose (*LADD*).
EC	=	Exposure concentration of the chemical in the medium of concern for the time period being analyzed, expressed in units of mg/kg for soil and food or mg/L for surface water or beverages (including milk).
CR	=	Consumption rate, the amount of contaminated medium consumed per unit of time, event, or other measure. (e.g., kg/day for soil and food; L/day for water).
EF	=	Exposure frequency (number of days exposed per year).
ED	=	Exposure duration (number of years exposed).
BW	=	Average body weight of the receptor over the exposure period (kg).
AT	=	Averaging time, the period over which exposure is averaged (days). For carcinogens, the averaging time is usually 25,550 days, based on an assumed lifetime exposure of 70 years; for non-carcinogens, averaging time equals *ED* (years) multiplied by 365 days per year.

As noted above, modeling and/or monitoring (sampling and analysis) can be used to determine the exposure concentration (EC) at specified exposure points. However, a variety of approaches and assumptions can be used to determine the remaining variables in the equation, as will be discussed in subsequent chapters. For example, calculation of the intake rate requires assumptions about diet (i.e., how much the exposed individual eats and drinks each day) and body weight (how much the individual weighs). Dietary assumptions need to be specific to the type of food consumed (e.g., fish, milk, beef).

As noted in Section 6.3.3.4, the exposure duration (ED) used to calculate chemical intake rate (I) will have an impact on the choice of toxicity values (e.g., acute vs. chronic) used to characterize risk and hazard. As a general rule, the ED values should match the exposure assumptions used in developing the dose-response values.

15.3.3.5 Evaluation of Uncertainty

As with the inhalation assessment, the evaluation of uncertainty includes both a **summary** of the values used to estimate exposure, including their range, midpoint,and other values; and a qualitative or quantitative **discussion** that evaluates which variables or assumptions have the greatest potential to affect the overall uncertainty in the exposure assessment.

15.3.3.6 Preparation of the Documentation

The analysis plan needs to specify the approach used to document the multipathway exposure assessment, as discussed in Chapter 20.

15.3.4 Identification of the Exposed Population

This part of the analysis plan identifies the exposed population that will be evaluated in the risk assessment. The procedure for characterizing the potentially exposed population generally will be similar to that described for the inhalation pathway (Chapter 6). As noted previously, it may be necessary to include individuals who live outside the modeled deposition area. The manner in which potentially exposed populations are characterized depends on the general approach used for the multipathway assessment (see Section 15.4 below).

15.3.5 Identification of Endpoints and Metrics

This part of the analysis plan identifies the specific human health endpoints that will be evaluated in the risk assessment and the metrics used to quantify exposure and risk. The multimedia assessment uses the same general endpoints (i.e., cancer and non-cancer) and presents the central tendency and high-end tendency descriptors required as the range of risk estimates of the distribution. Risk characterization is discussed in more detail in Chapter 22.

15.4 Exposure Assessment Approach

A variety of approaches are available for multipathway exposure assessments. This section describes two representative approaches that range from a relatively simple approach based on scenarios to a very complex and data-driven approach based on mass-balance models. This discussion is intended to illustrate some of the potential approaches available for multipathway exposure assessment. A given risk assessment might incorporate features of either of the two approaches outlined below, or might feature a different approach.

Regardless of the specific approach taken, EPA recommends a tiered approach to multipathway exposure assessment, in which the exposure assessment moves from relatively simple to more complex as warranted by the quality of available information and its ability to be used to support the risk management decision(s). Chapter 3 provides an overview of tiered approaches to risk assessment.

Childhood exposures need to be considered explicitly in any non-inhalation scenario. This can be done with a separate scenario (e.g., a "resident child") or by incorporating changes in consumption rates, dietary preferences, and body weight with age in the exposure factors incorporated into the scenario. EPA's Risk Assessment Forum recently published guidance on selecting appropriate age groups for assessing childhood exposures.[2]

15.4.1 Scenario Approach

Multipathway exposures may be evaluated by developing a number of scenarios that describe the potential human exposures that might occur via each of the potential exposure pathways identified in the conceptual model. An **exposure scenario** is a combination of exposure pathways by which a single defined human receptor might be exposed to air toxics that persist and which also may bioaccumulate. The specific exposure scenarios defined for a given risk assessment would be based on the characteristics of the exposure setting, potential exposure pathways, potential exposure points or areas, and predominant land uses and activities associated with the potentially exposed population. For example, if the study area included a small lake where fishing might occur, the assessment might include a "fisher" scenario that included ingestion of fish caught in the lake.

The scenario approach generally involves relatively simple modeling and fewer data requirements as modeling inputs. This can be performed by using "linked modeling systems" which can either be relatively simple or incorporate highly sophisticated single-medium models into a single multimedia system. However, these types of models do not assure conservation of mass and therefore may under- or over-estimate exposure concentrations for particular scenarios. The general scenario approach involves:

- Identifying the potential exposure pathways that may be important, including the areas where contaminants have the potential to accumulate in soils, surface waters, sediments, and biota; and specific activities that may result in ingestion of these contaminants (either via incidental ingestion of soil while in the contaminated areas or by consuming the contaminated plants, animals, or surface water).

- Developing a set of scenarios that describe reasonable sets of potential exposure pathways, given the types of people and activities that occur within the study area. The scenarios would include specific exposure factors (e.g., body weight, fish consumption) based on the particular activities identified above. The exposure scenarios should consider children, either as a separate scenarios (e.g., a "resident child") or as part of an overall scenario (e.g., someone who is born in the exposure area and lives there for 30 years, and thus experiences exposure both during childhood and as an adult). The exposure factors could be set initially at conservative levels for screening-level assessments and then at more site-specific levels for higher tiers of analysis. Each exposure scenario also should appropriately consider study-specific sub-populations that may experience different exposure conditions (e.g., because they eat different foods or parts of foods at different rates than the general population).

- Using relatively simple multimedia modeling techniques (e.g., "linked modeling systems" described in Chapter 18) to estimate exposure concentrations in the media and biota of interest to each scenario. Monitoring (sampling or analysis) could be used to augment the modeling effort. For screening-level analyses, the scenarios can be based on the locations

with the highest modeled concentrations or deposition rates. For example, a scenario involving consumption of fish could be based on the same location as a residential scenario. In some cases, it may be appropriate to evaluate an exposure scenario assuming exposure through ingestion of fish from one water body and drinking water from a different water body. Such assumptions may need to be refined in subsequent modeling tiers.

- Quantifying the dietary intake for each scenario based on the modeled or estimated exposure concentrations and the specific exposure factors for each scenario.

Several exposure scenarios are commonly used in multipathway risk assessments (Exhibit 15-4). A recent description of several scenarios is provided in EPA's risk assessment guidance for hazardous waste incinerators.[3] In addition to the commonly assessed scenarios provided in Exhibit 15-4, other scenarios may be appropriate, depending on study-specific conditions. For example, if a contaminated surface water body is used for bathing and swimming, incidental ingestion, and dermal exposure from these activities may need to be considered. As another example, the resuspension of contaminated soils (i.e., windblown dust) may be important in some study areas. Note also that exposure of an infant to chlorinated dioxins/furans (and other lipophilic contaminants) via the ingestion of breast milk may be evaluated as an additional exposure pathway, separately from adult exposures, in each of the scenarios outlined below. ***Note also that in each of these scenarios, the risk assessment needs to look at both an adult and child (for example, the "farmer" includes both an adult farmer and a farmer child)***.

Farmer. The farmer scenario is commonly evaluated to account for the combination of exposure pathways to which a person may be exposed in a farm or ranch exposure setting. As indicated in Exhibit 15-4, the farmer is commonly assumed to be exposed to air toxics through one or more of the following exposure pathways:

- Direct inhalation of vapors and particles;
- Incidental ingestion of soil;
- Ingestion of drinking water from surface water sources;
- Ingestion of homegrown produce;
- Ingestion of homegrown beef;
- Ingestion of dairy products from homegrown livestock;
- Ingestion of homegrown chicken;
- Ingestion of eggs from homegrown chickens;
- Ingestion of homegrown pork; and
- Ingestion of breast milk (evaluated separately for an infant [for PCBs, dioxins, and furans]).

In a Tier 1 assessment, the farmer commonly is assumed to consume a certain amount daily (e.g., grams/day) of each food group (beef, pork, poultry, eggs, and milk) to make up a total consumption rate, and amounts consumed are assumed to be homegrown. If site-specific information is available that demonstrates that a farmer does not raise beef, poultry, or pork, and that raising any of these livestock would not occur for a reasonable potential future farmer at a location, then elimination of one or more of these exposure pathways could be justified. The farmer scenario often does not include the fish ingestion exposure pathway. However, in some areas of the country, it is common for farms to also have stock ponds which are fished on a regular basis for the farmer's consumption. Also, ingestion rates (e.g., food, incidental soil ingestion) often are age-dependent.

Exhibit 15-4. Common Exposure Scenarios Used in Multipathway Exposure Assessments				
Exposure Pathways	**Scenarios[a]**			
	Farm Resident	**Resident**	**Resident with Garden**	**Local Fish Consumers**
Inhalation of Vapors and Particulates[b]	•	•	•	•
Incidental Ingestion of Soil	•	•	•	•
Ingestion of Drinking Water from Surface Water Sources	•	•	•	•
Ingestion of Homegrown Produce	•	–	•	•
Ingestion of Homegrown Beef	•	–	–	–
Ingestion of Milk from Homegrown Cows	•	–	–	–
Ingestion of Homegrown Chicken	•	–	–	–
Ingestion of Eggs from Homegrown Chickens	•	d	d	d
Ingestion of Homegrown Pork	•	–	–	–
Ingestion of Fish	d	d	d	•
Ingestion of Breast Milk	c	c	–	c

Notes:

• Pathway is included in exposure scenario.

– Pathway is not included in exposure scenario.

[a] Exposure scenarios are defined as a combination of exposure pathways evaluated for a receptor at a specific exposure scenario location. Note that these scenarios are not exhaustive (i.e., additional or other scenarios may be relevant to a particular exposure assessment). Note also that within each scenario, the quantitative exposure estimates will vary across age groups.

[b] Note that inhalation is included in the overall exposure assessment, but the inhalation exposure assessment is performed separately (as described in Part II of this Reference Library).

[c] Infant exposure to dioxins and/or furans via the ingestion of their mother's breast milk is evaluated for infants as an additional, separate exposure pathway.

[d] Regional specific exposure setting characteristics (e.g., presence of ponds on farms or within semi-rural residential areas, presence of livestock within semi-rural residential areas) may warrant inclusion of this exposure pathway when evaluating a recommended exposure scenario.

Resident. The resident scenario is commonly evaluated to account for the combination of exposure pathways to which a person may be exposed in an urban or rural (non-farm) setting. As indicated in Exhibit 15-4, the resident is commonly assumed to be exposed to air toxics through the following exposure pathways:

- Direct inhalation of vapors and particles;
- Incidental ingestion of soil;
- Ingestion of drinking water from treated surface water sources; and
- Ingestion of breast milk (evaluated separately for an infant [for PCBs, dioxins, and furans]).

The resident scenario often does not include the fish ingestion exposure pathway. However, in some areas of the country, ponds within semi-rural residential areas support fish for human consumption.

Resident with Garden. The resident with garden scenario is commonly evaluated to account for people who may be exposed while gardening or through the consumption of produce grown in their garden in an urban or rural (non-farm) setting. As indicated in Exhibit 15-4, the resident with garden is commonly assumed to be exposed to air toxics through the same exposure pathways as the resident scenario, with one additional exposure pathway:

- Ingestion of homegrown produce.

Local Fish Consumer. The local fish consumer scenario is evaluated to account for the combination of exposure pathways to which a receptor may be exposed in an urban or rural setting where fish is the main component of the person's diet. As indicated in Exhibit 15-4, the local fish consumer is commonly assumed to be exposed to air toxics through the following exposure pathways:

- Direct inhalation of vapors and particles;
- Incidental ingestion of soil;
- Ingestion of drinking water from surface water sources;
- Ingestion of homegrown produce;
- Ingestion of fish; and
- Ingestion of breast milk (evaluated separately for an infant [for PCBs, dioxins, and furans]).

In many cases, local fish consumers are assumed to grow some of their own produce, but this may or may not be relevant to a particular risk assessment. Also note that in some parts of the country, a primary reliance on fishing as a source or dietary protein is common - a circumstance known as "subsistence fishing." Subsistence hunting may also be important for some groups.

15.4.2 Population-Based Approach

Multipathway exposures may be evaluated by tracking individual members of a population and their inhalation and ingestion through time and space. Such analyses may incorporate a user-specified number of **simulated individuals** or population groups (**cohorts**) to represent the population in the study area. A cohort is defined here as a group of people within a population with the same demographic variables who are assumed to have similar exposures. In this approach, the exposure analysis process consists of relating chemical concentrations in environmental media (e.g., air, soil, water) to chemical concentrations in the exposure media with which a person or population has contact (e.g., soil, food, household dust). Exposure is estimated by tracking the movement of a population cohort through locations where chemical exposure can occur according to a specific activity pattern.[4] Models such as the Stochastic Human Exposure and Dose Simulation Model (SHEDS), Calendex, the Hazardous Air Pollution Exposure Model (HAPEM), and the Total Risk Integrated Methodology, Exposure Event Model (TRIM.Expo) incorporate this approach (see Chapter 19). The general approach, which is analogous to the inhalation exposure modeling techniques described in Chapter 9, involves:

- Defining **exposure districts** – geographic locations within the study area where there is potential contact between humans and a pollutant – and estimating chemical concentrations within each exposure district (through modeling and/or measurement).

- Preparing inventories of chemical concentrations in each microenvironment in each exposure district at selected time intervals (e.g., days, hours). These inventories may be developed using mass-balance approaches that predict the partitioning of chemicals throughout the environment or through the use of microenvironment factors.

- Identifying the characteristics and activity patterns of each population cohort or set of representative individuals (e.g., the demographic characteristics of the individual; the locations where the individual lives, works, etc.; the amount of time spent in each location, and the individual's activities within a location). These activity patterns should be representative of the exposed population of concern. For representative individuals or cohorts, each simulated person/cohort is represented by a "personal profile" or "activity pattern" developed by selecting from a set of variables that include:

  – Demographic variables (e.g. age, sex), which are generated based on census data;
  – Residential variables (e.g., where the person lives, works, etc., which are generated based on sets of distribution data;
  – Daily varying variables (e.g., how long a person works in a garden), which are generated based on distribution data that change daily during the simulation period;
  – Physiological variables (e.g., height, weight), which are generated based on age group-specific distribution data; and
  – Dietary variables (e.g., amount and type of food consumed), which are generated based on sets of distribution data.

  Profiles or activity patterns may be developed using probability density functions, allowing the analysis to incorporate probabilistic techniques such as Monte Carlo analysis (see Part VII of this reference manual).

- Summing the exposures for each exposure event over the assumed duration of exposure (e.g., lifetime or portion of a lifetime).

The primary advantage of the population-based approach is that it is a more realistic exposure assessment that simulates how people actually live and work within the study area. It therefore can provide a more complete characterization of the spatial and temporal patterns of exposure. The primary disadvantage of the cohort approach is the time and resources it requires, including significant input data requirements.

References

1. U.S. Environmental Protection Agency. 1995. *New Policy on Evaluating Health Risks to Children*. Memorandum to Assistant Administrators, General Counsel, Inspector General, Associate Administrators, Regional Administrators. From Carol M. Browner, Administrator, and Fred Hansen, Deputy Administrator. Washington, DC., October 20, 1995.

2. U.S. Environmental Protection Agency. 2003. *Guidance on Selecting the Appropriate Age Groups for Assessing Childhood Exposures to Environmental Contaminants (External Review Draft).* Risk Assessment Forum, Washington, D.C., February 1, 2003. EPA/630/P-03/003A. Available at: http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=55887

3. U.S. Environmental Protection Agency. 1998. *Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities, Volume 1. Peer Review Draft.* Office of Solid Waste and Emergency Response, Washington, D.C., July 1998. EPA/530/D-98/001A. Available at: http://www.epa.gov/combustion/risk.htm.

4. See the Human Exposure Modeling page of EPA's Fate, Exposure and Risk Analysis (FERA) website: http://www.epa.gov/ttn/fera/human_gen.html.

   See the TRIM.Expo web page at: http://www.epa.gov/ttn/fera/human_apex.html.

   U.S. Environmental Protection Agency. 1999. *Total Risk Integrated Methodology. TRIM.Expo Technical Support Document. External Review Draft.* Office of Air Quality Planning and Standards, Research Triangle Park, NC, November 1999. EPA/453/D-99/001. Available at http://www.epa.gov/ttn/fera/trim_fate.html#1999historical.

Chapter 16 Quantification of Exposure: Development of the Emissions Inventory for the Multipathway Risk Assessment

Table of Contents

16.1 Introduction 1

16.2 Developing the Emissions Inventory 1

16.1 Introduction

Chapter 7 provides an overview of the process used to develop an emissions inventory for an air toxics risk assessment. As noted in that chapter:

- Emissions data are a source term for the risk assessment, primarily as a key input for computer models that estimate the transport of chemicals in the atmosphere; if and how they will be transformed by chemical or physical processes; how and where they will be deposited; and how they will continue to partition and move through environmental media following deposition.

- Developing the emissions inventory involves identifying the specific air toxics released from the source and quantifying release characteristics (e.g., release rates, temperature, release velocity).

- Local enhancements of existing air toxics emissions inventories may be advantageous to a particular air toxics assessment effort as a very critical initial step, because air toxics inventories are not always at the quality that would provide the results desired in a modeling assessment.

16.2 Developing the Emissions Inventory

The process used to develop the emissions inventory for the multipathway risk assessment is similar to the process for inhalation analyses (see Chapter 7 for a description of this process). However, there are a few additional considerations that may apply to a given multipathway analysis (e.g., information on particulate/particle-bound/vapor fractions if ISCST3 is used). See Chapter 18 for more discussion on inputs for models used in multimedia assessment.

Chapter 17 Quantification of Exposure: Chemical and Physical Properties Affecting Multimedia Fate and Transport

Table of Contents

17.1 Introduction 1
17.1.1 Measures of Persistence 1
17.1.2 Metrics of Bioaccumulation 5

17.2 Chemical and Physical Properties that Affect Persistence and Bioaccumulation 8

17.3 Evaluating Persistence and Bioaccumulation in Exposure Assessments 10

References 11

17.1 Introduction

This chapter provides both an overview of the physical and chemical properties of chemicals that persist in the environment long enough to be of potential concern for multimedia exposure and information on their potential to bioaccumulate in biological tissues to levels that may result in significant exposures. This chapter also provides a brief discussion of various approaches for evaluating persistence and bioaccumulation in an air toxics exposure assessment.

- The term **persistence** refers to air toxics that are slow to degrade in the atmosphere or in the soils, water, and/or sediments onto which they deposit and partition. Under certain circumstances, persistent chemicals can increase in concentration over time. In addition, some of these pollutants can bind tightly to soils and sediment and move from place to place by erosion. Examples of persistent chemicals are metals (which never degrade) and polychlorinated biphenyls (which degrade, but only over very long periods of time). Measures of persistence are described in Section 17.1.1.

- The term **bioaccumulation** refers to persistent chemicals that build up in the tissues of living organisms to concentrations that are higher than in the surrounding environment. These pollutants are often lipophilic in nature, which allows them to be taken up in and stored by fat tissues. As an example, the group of chemicals known collectively as dioxins (usually some mixture of chlorinated dioxins and furans) persists for long periods of time in the environment and is strongly lipophilic. Repeated exposure to dioxin in food can lead to increased body burdens in fat tissue and human milk. Measures of bioaccumulation are discussed in 17.1.2.

- The term **biomagnification** refers to persistent chemicals that increase in concentration as they transfer up the food chain so that they accumulate to higher concentration levels with each successive food chain level (see Section 17.1.2).

The air pollutant's physical and chemical properties and the characteristics of the environment to which the pollutant is emitted affect its potential to persist and bioaccumulate. One of the most important determinants in predicting persistence and accumulation in biota and other environmental media is the partitioning behavior of the pollutant. Partitioning refers to where in the environment a chemical will tend to reside and in what relative quantities. When released to the air, chemicals may partition to air, water, soils, sediments, or biota, depending on a number of chemical and site-specific factors. Section 17.2 highlights measures of partitioning and other chemical and physical properties. Section 17.3 discusses how measures of persistence, bioaccumulation, and partitioning are used in exposure assessment.

17.1.1 Measures of Persistence

Estimating the persistence of chemicals in the environment is a challenging exercise. Persistence depends on basic processes such as how the chemicals are released (i.e., which environmental media they are released to initially, in this case, air); how they move in the environment (i.e., to which environmental media they tend to partition); and their tendency to degrade within those specific media (i.e., their persistence in air vs. in water). These basic processes, in turn, depend on a number of chemical-specific properties and site-specific conditions. However, despite the

complexity of the overall process, some general screening methods are available to get a sense of persistence of chemicals in certain media.

Within each environmental medium, several different degradation processes can influence the persistence of chemicals. However, regardless of the specific degradation process, what generally occurs is that organic chemicals (or organo-metallic compounds) are reduced in size and complexity, and, if complete degradation occurs, converted to carbon dioxide, methane, ammonia, water, sulfates, phosphates, nitrates, and other end products. Explanations of the specific degradation processes and their applications within different environmental media are summarized below and in Exhibit 17-1:

- **Aerobic biodegradation** is the breakdown of chemicals by microorganisms that utilize oxygen;
- **Hydrolysis** is the breakdown of chemicals by reaction with water;
- **Photolysis or photodegradation** is the process by which chemicals can be degraded by the energy in artificial light or sunlight;
- **Oxidation or reduction (or redox)** reactions involving the exchange of electrons between the pollutant and reactive compounds found in the environment;
- **Photooxidation** is a process by which oxidation and photolysis work jointly to break down chemicals and refers to a reaction with oxygen in the presence of light (usually sunlight); and
- **Anaerobic biodegradation** is the breakdown of chemicals by microorganisms without the use oxygen (for example, in sediments).

Exhibit 17-1. Degradation Processes in Environmental Media

Environmental Medium	Applicable Degradation Process(es)
Air	Photolysis
	Photooxidation
Surface water	Aerobic biodegradation
	Hydrolysis
	Photolysis
	Photooxidation
Soils	Aerobic biodegradation
	Hydrolysis
	Photolysis (surface soil)
	Oxidation - reduction reactions
Sediments	Anaerobic biodegradation

Persistence is usually described using a term called **half-life**, which is the time required for one-half of the original mass of the chemical to be degraded, transformed, or destroyed in a given medium. Half-life values may be measured directly or estimated (e.g., with computer models that predict half-life based on chemical structure). Alternatively, the literature may report a degradation or transformation **rate constant** in units such as 1/day or day^{-1}. Assuming the reaction is first-order, the rate constant can be converted to the half-life and vice versa using the equation $t_{½} = 0.693/k$, where $t_{½}$ is the half-life (days) and k is the first-order rate constant (day^{-1}). Exhibit 17-2 provides a list of data sources for degradation half-life or rate constant values.

Exhibit 17-2. Data Sources for Half-Life and Rate Constant Values		
Data Source	**Data Elements**	**Comments**
Howard et al. Handbook	• High and low compartment half-life values for soil, air, surface water, and sediment/ground water based on measurements and modeling estimations; and • High and low measured and estimated process-specific half-life values, including hydrolysis, reduction, photolysis, photooxidation (in water and air), and biodegradation.	Howard et al.[1] compiled measured and estimated environmental degradation rates for organic chemicals, including polycyclic aromatic hydrocarbons (PAHs), pesticides, and solvents.
Mackay et al Handbooks	• Measured and estimated half-life and rate constant data for air, water, soil, and sediment.	Mackay et al.[2] compiled measured and estimated environmental degradation rates for organic chemicals, including monoaromatic hydrocarbons, chlorobenzenes, polychlorinated biphenyls, polynuclear aromatic hydrocarbons, polychlorinated dioxins and dibenzofurans, volatile organic compounds (VOCs), pesticides, and oxygen, nitrogen, and sulfur organic compounds.
Verschuren	• Measured and estimated half-life values in surface water, ground water, sediment, soil, and biota.	Verschuren[3] compiled measured and estimated degradation rates for organic chemicals.
Mercury Study Report to Congress	• Measured demethylation rate constants converted to half-life data for mercury (water, soil, sediment).	The *Mercury Study Report to Congress*[4] was developed for a rulemaking and has undergone extensive Agency peer review.
HYDROWIN	• Estimated hydrolysis rate for water.	HYDROWIN, the hydrolysis estimation program, is part of EPA's Estimation Program Interface (EPI) Suite.[5] HYDROWIN uses a chemical's structure to estimate the acid- and base-catalyzed rate constants for certain chemical classes (esters, carbamates, epoxides, halomethanes, and certain alkyl halides). Chemicals can be catalyzed (broken down) by acids (hydronium) or bases (hydroxide ions). The rate constants are used to calculate hydrolysis half-lives at selected pHs.

Half-life values for the same substance in different media, and for different substances in the same medium, can differ by several orders of magnitude. This is illustrated in Exhibit 17-3.

Exhibit 17-3. Example Illustrating Ranges of Half-life Values in Different Environmental Media

HAP	Measured or Estimated Half-life Value (Hours)[a]			
	Air	Water	Soil	Sediment
acrolein	19	672	672	1,776
benzene	276	252	252	9,984
chlorobenzene	401	2,616	50	1,800
formaldehyde	4	192	96	384
methyl bromide	8,980	420	420	1,680
2,3,7,8-TCDD	200	768	12,096	14,400

Note: Values are for Illustrative Purposes Only

(a) Values represent the maximum measured or estimated values from the references cited in Exhibit 17-2.

The fate and transport models used for characterizing multipathway exposure use metrics of persistence to account for loss of the chemical through degradation or transformation processes described above. Typically, for each modeled media compartment, the fate and transport models require an overall metric of persistence for that media compartment (e.g., a half-life value for air, a half-life value for soil, a half-life value for surface water). The media compartment half-life is the half-life associated with the most important or fastest degradation process or reaction. That is, the process-specific half-life of the fastest degradation process is usually selected as the overall media compartment half-life.

Metals may transform among different compounds or species (e.g., divalent mercury can undergo methylation to yield methyl mercury, and it can be reduced to form elemental mercury). Some metal species are more persistent and/or may be more toxic than others. Transformation half-life values in different media are more useful for evaluating the persistence of metal pollutants of concern. The speciation of metals is highly dependent on geochemical environment (and in some cases, the presence of certain microbes). One species may dominate in water of one pH, Eh, DOC concentration, etc., and an entirely different species may dominate in water of different geochemistry. [Also note that a further complicating factor is that analytical laboratories often report the total amount of a metal present in a sample, rather than the amount of the various individual metal species present (e.g., Cr^{6+} versus Cr^{3+}), which usually have different toxicities and different persistence values. An understanding of the concentrations of various metal species is therefore desirable; however, it is often not analytically achievable.][6] There are a number of concerns regarding the assessment of risks posed by metals in the environment. Information on this subject can be found at the following website: http://cfpub.epa.gov/ncea/raf/recordisplay.cfm?deid=51736.

What is a Persistent Chemical?

Because environmental persistence is a complicated phenomenon, no single value is universally accepted as indicating a "persistent" chemical. EPA has used a half-life value of two months (1,440 hours) in the Toxics Release Inventory (TRI) PBT final rule and the Premanufacturing Notice evaluation process.[7] Other authors have suggested a half-life value of one month (720 hours), using the following logic: Assuming that a chemical will degrade in approximately six half lives (when less than two percent of the mass remains), a chemical with a half-life of one month would persist in the environment for six months. This six month period would be long enough to encompass the sensitive developmental life stages of many organisms.[8]

Note that **the use of any single threshold value to define "persistent" chemicals may be misleading**. An important consideration is data quality (e.g., whether the half-life value is measured or predicted and the overall quality of the experimental study or computer algorithm used to develop the value). Assessors must consider other factors including the tendency of the chemical to partition into various media. For example, vinyl chloride has a half-life of six months in surface water and two years in sediment – values that might suggest this chemical is "persistent." However, its half-life in air is 53.4 hours and in soil is 10 days. Thus, depending on its relative tendency to partition between air, soil, water, and sediment, a significant amount of the mass in air emissions might be degraded (in the air and/or soil) prior to the chemical reaching surface water and sediment.

Ultimately, it is often difficult to select a single and reliable half-life because the half-life of a chemical depends not only on its physical properties (see section 16.4), but also on the environmental conditions of the site to which the chemical is emitted. Temperature, sunlight intensity, the nature of the microbial community, and concentrations of reactive species such as oxygen radicals can all affect the reactivity of a compound.[3]

17.1.2 Metrics of Bioaccumulation

Several chemical-specific metrics can be used to evaluate the potential for a chemical to bioaccumulate in plants and animals and biomagnify in food webs.[9] These values may be measured in laboratory tests or estimated with computer models based on chemical structure. These metrics include:

Note that BAFs and BCFs are not calculated for humans; rather, they are sometimes used to estimate air toxics concentrations in the food items eaten by people.

- **Bioaccumulation Factor (BAF)**. The concentration of a substance in tissue of an organism divided by its concentration in an environmental medium in situations where the organism and its food are exposed (i.e., accounting for food chain exposure as well as direct chemical uptake). Such values are often used to characterize the transfer of pollutants through consumption of fish, beef, or dairy products.[10]

- **Bioconcentration Factor (BCF)**. The concentration of a substance in the tissue of an organism divided by the concentration in an environmental medium (e.g., the concentration of a substance in an aquatic organism [µg/kg body weight] divided by the concentration in the ambient water [µg/L water], in situations where the organism is exposed through the water only). The most commonly given value is an estimate of the relative concentrations in water and whole fish (or, in some cases, fish fillets, since most people in the U.S. do not eat

whole fish), but BCFs for other organisms are also available. For plants, the Root Concentration Factor (RCF) indicates how readily pollutants are taken up from soil into underground tissues.

- **Octanol/water partition coefficient**. The ratio of a chemical's solubility in n-octanol to its solubility in water at equilibrium (in this test, n-octanol is used as a surrogate for lipophilic tissue). This metric, usually expressed as a logarithm (log K_{ow} or log P), is often used as a surrogate for (and is an important basis for estimating) BAF or BCF.

As with persistence, bioaccumulation is a complicated phenomenon, and no single value is universally accepted as indicating that a chemical has a high tendency to bioaccumulate, although EPA's Office of Pollution Prevention and Toxics has used a threshold BCF value of 1,000 in evaluating new and existing chemicals.[11] Exhibit 17-4 provides a list of sources for bioaccumulation data.

Importance of Trophic Levels in Evaluating Bioaccumulation in Aquatic Food Chains

An organism's trophic position in the aquatic food web can have an important effect on the magnitude of bioaccumulation of certain chemicals. Certain pollutants have the potential to biomagnify, or increase in concentration at successive trophic levels through a series of predator-prey associations. Chapter 5 (Bioaccumulation) of EPA's *Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health* provides guidance about deriving trophic level-specific bioaccumulation factors.[17]

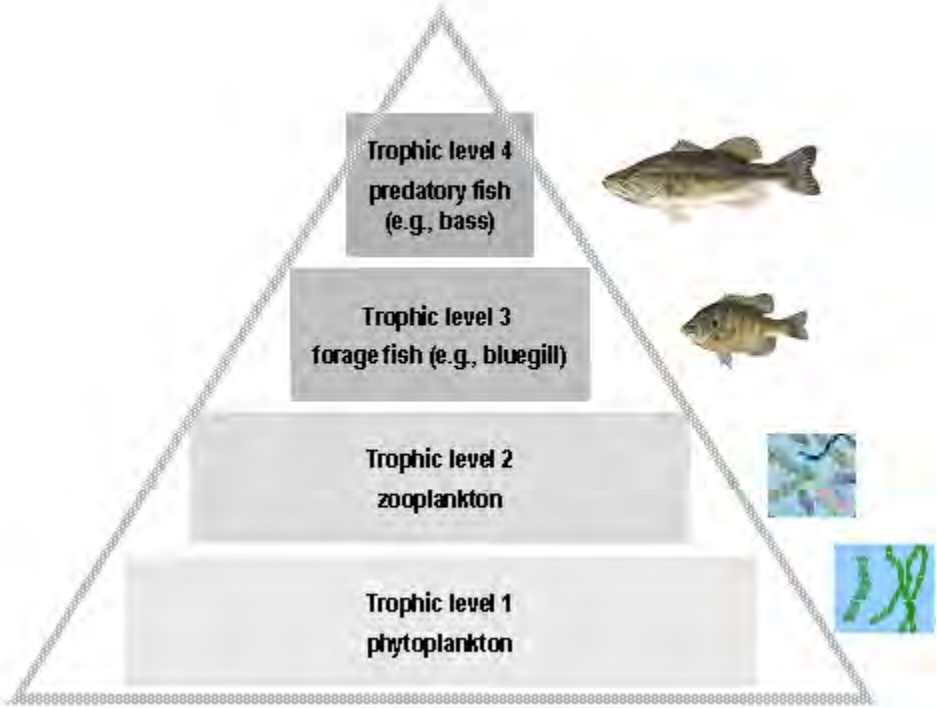


Exhibit 17-4. Sources of BCF and BAF Values		
Data Source	**Data Element**	**Comments**
HWIR Technical Support Document	Measured BAF Measured BCF Predicted BAF Predicted BCF	The Hazardous Waste Identification Rule (HWIR) Technical Support Document[12] data were developed for a rulemaking. Estimates are available for different classes of organisms (fish and invertebrates).
Mercury Study Report to Congress	Measured BAF for mercury	The *Mercury Study Report to Congress*[4] was developed for a rulemaking and has undergone extensive Agency peer review.
Ambient Water Quality Criteria documents	Measured BCF	Ambient Water Quality Criteria documents[13] are developed for rulemakings that establish concentration limits for chemicals in the surface waters of the United States. They have undergone extensive Agency peer review.
AQUIRE Database	Measured BCF	EPA's Office of Research and Development, Environmental Research Laboratory in Duluth, MN, maintains a database of citations and aquatic bioassay data including residue measures.[14]
BCFWIN	Predicted BCF	BCFWIN, the Bioconcentration Factor Estimation Program, is part of EPA's EPI Suite.[5] This program estimates BCFs based on log K_{ow} data using the estimation methodology presented in a 1997 study prepared for EPA by Meylan et al.[15] The methodology was formulated using a training set of 694 compounds with measured BCF values for fish. In order of preference, the program uses (1) the log K_{ow} entered by the user, (2) the experimental log K_{ow} from the experimental log K_{ow} database for KOWWIN, and (3) the KOWWIN estimated log K_{ow} value.

BAF or BCF values are used in some fate and transport models to calculate how much of the chemical will partition into organisms, such as fish or shellfish, that are consumed by humans or ecological receptors of concern. The concentrations in these biota can then be used to calculate the intake of the chemical by humans or ecological receptors of interest. It is recommended that BAFs be derived separately for species of different trophic levels to account for different levels of accumulation for members of different trophic levels.[16] EPA presents specific guidelines for deriving BAF values, including how to estimate BAFs using BCF values in its revised methodology for developing Ambient Water Quality Criteria.[17] National-level BAFs developed by the Office of Water can be used in screening analyses. These can be refined based on site-specific characteristics in subsequent tiers of the assessment, if necessary.

As with half-life values, BCF values vary significantly among air toxics. Exhibit 17-5 presents a few representative BCF values for several HAPs. Note that these represent the highest values identified in the references cited in Exhibit 17-4 and are meant for illustrative purposes only.

Exhibit 17-5. Example BCF Values	
HAP	**BCF Value[a]**
acrolein	3
carbon tetrachloride	30
chloroform	6
pentachlorophenol	776
2,3,7,8-TCDD	5,754
Note: values are for illustrative purposes only	
(a) Values represent the maximum measured or estimated values from the references cited in Exhibit 17-4.	

Note that **the use of any single threshold value to define "bioaccumulative" chemicals may be misleading**. As with persistence, data quality is an important consideration (e.g., measured vs. predicted values; quality of the underlying study/algorithm used to develop the value). Moreover, the specific organisms and tissues in which bioaccumulation is measured or predicted are important determinants of the relevance of a given BCF or BAF value for the exposure assessment. Many substances that bioaccumulate tend to accumulate in lipid tissues (e.g., fat, organs), which may not be the tissues that most people eat (e.g., fish fillet). Therefore, a high BCF may not actually result in high exposures to humans.

17.2 Chemical and Physical Properties that Affect Persistence and Bioaccumulation

Exhibit 17-6 identifies both direct metrics of persistence (i.e., half-life values), as well as the physical and chemical properties that are most important in determining persistence and bioaccumulation.

In Exhibit 17-6, **solubility** and **vapor pressure** determine the propensity for pollutants to dissolve in water and volatilize into the air, respectively. The **Henry's Law constant**, which is simply the ratio of the chemical's water solubility to its vapor pressure, indicates whether a compound will partition into air or water at equilibrium. The speed with which the equilibrium occurs is affected by the diffusion constants in air and water.

The coefficients that are abbreviated with a capital "K" (e.g., the octanol-water partition coefficient, K_{ow}) in Exhibit 17-6 provide information on how strongly organic and inorganic compounds are likely to bind to soil or sediment particles, or to partition into lipid versus aqueous phase liquids. Strong binding indicates a high potential to persist and accumulate in soils and sediments; soil/sediment binding also tends to be correlated with the potential to bioaccumulate.

The 1998 Peer Review Draft of EPA's *Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities* (HHRAP) provides (Appendix A) tables of recommended rate constants and chemical/physical parameters for a large number of air toxics.[18] Also, the user's guide for EPA's Risk Screening Environmental Indicators (RSEI) tool includes physicochemical properties for TRI chemicals and chemical categories.[19] Note, however, the values in these sources may need to be updated.

Exhibit 17-6. Chemical and Physical Property Definitions		
Property	**Definition**	**Significance/Comments**
Name	Unique identifier	Not always reliable (many synonyms)
CAS No.	Unique identifier	Much more reliable than the chemical name; some chemicals have no CAS number; some CAS numbers refer to mixtures
MW	Molecular weight	In absence of data, can be used to calculate D_a, D_w, etc.
V_p	Vapor pressure	Indicates volatility
S	Solubility	Indicates maximum concentration of a chemical that will dissolve in water
H	Henry's Law constant	Ratio of vapor phase concentration to the liquid phase concentration of a gas; high values indicate tendency to volatilize from water solution
D_a	Diffusion coefficient in air	Used to calculate rate of volatilization from air
D_w	Diffusion coefficient in water	Used to calculate rate of volatilization from water
K_{ow}	Octanol-water partition coefficient (log K_{ow} is frequently tabulated)	High value (> 1000, log K_{ow} > 3) indicates strong tendency to bioconcentrate
K_{oc}	Organic carbon partition coefficient (log K_{oc} often tabulated)	High value indicates strong tendency to bind to soil/sediment; K_{oc}, K_{ow} can be estimated from each other
Kd_{soil}	Soil dissociation constant	Indicates potential of inorganic ions/compounds to bind to soil; varies for different ionic species, pH, soil types
Kd_{sed}	Sediment dissociation constant	Indicates potential of inorganic ions/compounds to bind to sediment (similar to Kd_{soil})
$t_{1/2soil}$	Half-life in soil	Indicates persistence in soil; generally soil type, conditions, and degradation pathway(s) must be specified
$t_{1/2sed}$	Half-life in sediment	Indicates persistence in sediment; same considerations as for $t_{1/2soil}$
$t_{1/2sw}$	Half-life in surface water	Indicates persistence in surface water; for moderate to high-vapor pressure compounds
BAF	Bioaccumulation factor	Indicates accumulation of a compound into tissues of an organism from contact with contaminated water, contaminated sediments, and ingestion of contaminated food
BCF	Bioconcentration factor	Indicates accumulation of a compound into tissues of an organism from contact with a contaminated medium.
RCF	Root concentration factor	Ratio of root to soil concentration, measures propensity to take up pollutant from soil for defined plant species
B	Biotransfer factors	Describes propensity of pollutant to be transferred through food chain; defined for specific crops and consuming organisms (e.g., alfalfa => dairy cattle); generally correlates with BCF, K_{ow}; used primarily for detailed pathway modeling

Fugacity as a Determinant in the Fate and Transport of a Pollutant

Once a contaminant is emitted from its source, the fate of the contaminant is determined by a number of factors (e.g., molecular weight, solubility, partition coefficients). In order to characterize the fate of the pollutant, modelers have developed the concept of fugacity[20] to define the tendency of the gas to escape to another phase in order to reach a steady-state equilibrium. This tendency to migrate then leads to partitioning of the pollutant across environmental media based on its chemical and physical characteristics. Fugacity models are distribution-based models incorporating all environmental compartments (media) and are used to quantify steady-state fluxes of pollutants across compartment interfaces. There exist three levels at which fugacity may be modeled. The Level I model depicts the distribution of a known quantity of a chemical in a closed environment at equilibrium, in which no degrading reaction occurs and no advective gain or loss is attained. A Level II model describes a situation in which a chemical is continuously discharged at a constant rate and achieves steady-state equilibrium, at which the input and output rates are equal. In Level II models, reaction and advection may occur. A Level III model is similar to a Level II model in that the modeled chemical is continuously discharged at a constant rate and achieves a steady state condition, in which the input rate equals the output rate. Yet, a Level III model differs in that fugacity for the given chemical is equal within a single compartment for all defined subcompartments, but is not equal between compartments. Therefore, individual inputs for each medium must be defined separately in order to determine appropriate pollutant partitioning among environmental compartments.

17.3 Evaluating Persistence and Bioaccumulation in Exposure Assessments

Characterizing the movements of air toxics that persist and which also may bioaccumulate through various environmental media such as air, soils, water, sediments, and biota can be a highly complex task, and generally available methods for evaluating these fate and transport pathways have only recently been developed. Specifically, EPA and other regulatory agencies have developed a number of models that estimate the concentrations of persistent and bioaccumulative compounds over time in the various environmental "compartments" subsequent to defined patterns of deposition. These models simulate both physical and chemical processes such as air deposition to soil, runoff, leaching (dissolution), soil/sediment adsorption, and chemical speciation (oxidation/reduction, precipitation reactions). Some models also evaluate the biodegradation of organic pollutants by bacteria and other organisms in soil, sediment, and surface water. These models require a large number of site-specific inputs and many measures of the physical and chemical properties of pollutants. It is beyond the scope of this chapter to discuss in detail all the processes that may be modeled in the assessment of indirect exposure pathways; however, Mackay and his colleagues provide a good overview of the multimedia fate and transport[20] and Conell and Emlay describe the principles governing the bioaccumulation of pollutants in the environment.[21] Also, Chapter 18 provides a description of available multimedia models that are generally recommended for use in air toxics risk assessment. The documentation for these models provides detailed descriptions of the specific processes and methods used in each model.

References

1. Howard, P., Boethling, R., Jarvis, W.F., Meylan, W. 1990. *Handbook of Environmental Degradation Rates*. Lewis Publishers, Michigan.

2. Mackay D., Shiu W.Y., and Ma K.C. 1991-1997. *Illustrated Handbook of Physical-Chemical Properties and Environmental Fate for Organic Chemicals*, (5 volumes). Lewis Publishers Inc., Boca Raton, Florida.

3. Verschueren, K. 1996. *Handbook of Environmental Data on Organic Chemicals*. 3rd ed. Van Nostrand Reinhold Co., New York, NY.

4. U.S. Environmental Protection Agency. 1997. *Mercury Study Report to Congress*. Office of Air Quality Planning and Standards; Office of Research and Development, Washington, D.C., December 1997. EPA/452/R-976/003. Available at: http://www.epa.gov/ttn/atw/112nmerc/volume1.pdf.

5. U.S. Environmental Protection Agency. *2002. The Estimations Programs Interface for Windows (EPIWIN) and SMILES*. Updated June 20, 2002. Available at: http://www.epa.gov /oppt/p2framework/docs/epiwin.htm. (Last accessed April, 2004).

6. Stumm, W. and Morgan, J.J. 1996. *Aquatic Chemistry*, 3rd Ed. John Wiley & Sons, Inc., New York.

   Langmuir, D. and Klusman, R.L. 1997. Natural Background Concentrations of Metals That Exceed Drinking Water Standards: Defining Background and the Effect of Sample Filtration. Abstract. *Geol. Soc. Am. Annual Meetings*. Oct. 20-23. Salt Lake City UT: A-435.

   Langmuir, D., Chrostowski, P., Chaney, R., and Vigneault, C. 2003. *Draft Issue Paper on the Environmental Chemistry of Metals.* Submitted to EPA's Risk Assessment Forum by ERG, Lexington, MA. August, 2003.

7. U.S. Environmental Protection Agency. 1999. *Persistent Bioaccumulative Toxic (PBT) Chemicals (40 CFR Part 372*). *Final Rule. Federal Register* 64:58665, October 29, 1999.

   U.S. Environmental Protection Agency. 1999. *Notice of Receipt of Requests for Amendments to Delete Uses in Certain Pesticide Registrations*. *Federal Register* 64:60193, November 4, 1999.

8. Boethling, R.S., P.H. Howard, W. Meylan, W. Stiteler, J. Beauman, and N. Tirado. 1994. Group contribution method for predicting probability and rate of aerobic biodegradation. *Environmental Science and Technology*. 28:459-465.

9. U.S. Environmental Protection Agency. 1995. *Great Lakes Water Quality Initiative Technical Support Document for the Procedure to Determine Bioaccumulation Factors.* Office of Water, Washington, D.C., March 1995. EPA-820-B-95-005.

10. U.S. EPA. 1990. *Interim Final Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions.* Environmental Criteria and Assessment Office, Office of Research and Development, Washington, D.C., January 1990. EPA-600-90-003.

11. U.S. Environmental Protection Agency. 1992. *Classification Criteria for Environmental Toxicity and Fate of Industrial Chemicals*. Office of Pollution Prevention and Toxics, Washington, D.C.

12. U.S. Environmental Protection Agency. 1995. *Technical Support Document for the Hazardous Waste Identification Rule: Risk Assessment for Human and Ecological Receptors. Volume I.* Office of Solid Waste, Washington, DC, August, 1995.

13. U.S. Environmental Protection Agency. 2003. Office of Water Shopping Cart Home Page. Available at: http://yosemite.epa.gov/water/owrccatalog.nsf/.

14. U.S. Environmental Protection Agency. 2003. *AQUatic Information REtrieval (AQUIRE) database, a part of the ECOTOX database. Version 3.0.* Office of Research and Development, National Health and Environmental Effects Laboratory, Mid-Continent Ecology Division, Duluth, MN. Available at: http://www.epa.gov/ecotox/

15. Meylan, W. M., P. H. Howard, D. Aronson, H. Printup, and S. Gouchie. 1997. *Improved Method for Estimating Bioconcentration Factor (BCF) from Octanol-Water Partition Coefficient. Third Update Report - August 1997.* SRC-TR-97-006 (EPA Contract No. 68-D5-0012). Prepared for Robert S. Boethling, U.S. EPA, OPPT, Washington, D.C.; Prepared by SRC, Environmental Science Center, North Syracuse, NY 13212.

16. U.S. Environmental Protection Agency. 1998. *Methodology for Assessing Health Risks Associated with Multiple Pathways of Exposure to Combustor Emissions*. Office of Research and Development, National Center for Environmental Assessment, Cincinnati, OH. EPA 600/R-98/137.

17. U.S. Environmental Protection Agency. 2000. *Methodology for Deriving Ambient Water Quality Criteria for the Protection of Human Health.* Office of Water, Washington, D.C., October 2000. EPA/822/B-00/004. Available at: http://www.epa.gov/waterscience/humanhealth/method/method.html

18. U.S. Environmental Protection Agency. 1998. *Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities, Peer Review Draft.* Office of Solid Waste and Emergency Response, Washington, D.C., July 1998. EOA/30/D-98/001A. Available at: http://www.epa.gov/epaoswer/hazwaste/combust/risk.htm.

19. U.S. Environmental Protection Agency. 2002. *Risk-Screening Environmental Indicators User's Manual RSEI Version 2.1 [1988-2000 TRI Data]*. Office of Pollution Prevention and Toxics, Washington, D.C., December, 2002. Available at: http://www.epa.gov/opptintr/rsei/docs/users_manual.pdf.

20. Mackay, D. 1991. *Multimedia Environmental Models. The Fugacity Approach.* Lewis Publishers, Chelsea, MI.

Mackay D, Paterson S, and WY Shiu. 1992. Generic models for evaluating the regional fate of chemicals. Chemosphere 24(6):695-717.

Mackay D, DiGuardo A, Paterson S, and CE Cowan. 1996. Evaluating the environmental fate of a variety of types of chemicals using the EQC Model. Environmental Toxicology and Chemistry 15(9):1627-1637.

21. Connell, D.W. and D. Emlay. 1989. *Bioaccumulation of Xenobiotic Compounds*. CRC Press.

Chapter 18 Quantification of Exposure: Multimedia Modeling

Table of Contents

18.1 Introduction 1

18.2 Multimedia Fate and Transport Modeling 1
18.2.1 Basis of Multimedia Models 1
18.2.2 Multimedia Exposure Models 2

18.3 Key Parameters/Inputs for Multimedia Models 5

18.4 Examples of Multimedia Modeling 8

References 10

18.1 Introduction

This chapter summarizes the concepts and tools available for multimedia modeling to support a multipathway human health risk assessment. The discussion is divided into three sections:

- Section 18.2 discusses multimedia fate and transport modeling used to estimate chemical concentrations in abiotic and biotic media that indirectly result from air emissions;

- Section 18.3 discusses key parameters used as inputs to multimedia models; and

- Section 18.4 presents examples of the use of multimedia models in air toxics risk assessments.

18.2 Multimedia Fate and Transport Modeling

Although the primary route of exposure to many air toxics is via inhalation, non-inhalation exposure through soil, water, and food pathways can be a potential health concern for those air toxics that persist and which also may bioaccumulate (see Chapter 4 for the list of persistent bioaccumulative hazardous air pollutants (PB-HAP) chemicals). Therefore, risk assessments for these substances often include multimedia modeling to predict the movement of these air toxics in the environment. This section provides an overview of the multimedia fate and transport models commonly used by EPA.

18.2.1 Basis of Multimedia Models

Multimedia fate and transport models take into account various physical and chemical processes to predict the movement of pollutants within and between environmental media. Multimedia models can be grouped into the following basic categories.

- **Linked modeling systems** are composed of several independent single-medium models. These systems typically consist of a "one-way" process through a series of linked single-medium models or algorithms; that is, they calculate fate and transport by running a single-medium model (e.g., an atmospheric model) and using the output as the input for the next single-medium model (e.g., a soil or surface water model). One of the primary advantages of linked modeling systems is that they can incorporate several highly sophisticated single-medium models into a single modeling system. The primary drawbacks of these types of models are (1) they do not always assure conservation of mass; (2) they lack dynamic "feedback" loops; and (3) secondary pollutant transfers are not treated in a fully coupled manner.

- **Fully coupled, mass-conserving models** estimate the fate and transport of pollutants between and within media and are able to fully account for the distribution of pollutant mass within a defined modeling region. In these types of models, each of the included media (e.g., soil, air, biota) are modeled simultaneously (i.e., fully coupled), and thus these models can simulate dynamic "feedback" loops and secondary pollutant transfers. The primary drawback of these types of models is that they typically involve some simplification relative to sophisticated single-medium models due to the computational demands associated with modeling multiple media simultaneously.

18.2.2 Multimedia Exposure Models

To date, EPA has used primarily the Multiple Pathways of Exposure (MPE) model and variations of the MPE approach to conduct multimedia fate and transport modeling for air toxics. More recently, EPA developed the Fate, Transport, and Ecological Exposure (TRIM.FaTE) model as a component of the Total Risk Integrated Methodology (TRIM).[1] This section provides a summary of the MPE model, the variations of the MPE approach, and the TRIM.FaTE model. During the development of the TRIM.FaTE model, EPA conducted a comprehensive review of those multimedia fate and transport models that estimate exposures and risks from emissions of air toxics that EPA and other organizations in the United States use. Exhibit 18-1 provides a summary of the models included in this review, with models grouped into the two basic categories described in the previous section. The TRIM.FaTE documentation provides a description of each of these models. Also presented at the end of this section is a multimedia model developed by the State of California called CalTOX.[2]

Exhibit 18-1. Multimedia Models Reviewed During TRIM.FaTE Development

Linked Modeling Systems	Fully Coupled, Mass-Conserving Models
• Indirect Exposure Methodology (IEM)/Multiple Pathways of Exposure (MPE), developed by EPA's National Center for Environmental Assessment • Multimedia Environmental Pollutant Assessment System (MEPAS), developed by the U.S. Department of Energy	• CalTOX, California Department of Toxic Substance Control's Multimedia Risk Computerized Model • SimpleBOX, developed by the Netherlands National Institute of Public Health and the Environment • Modeling Multimedia Environmental Distribution for Toxics (Mend-Tox)/ISMCM), developed by EPA's Office of Research and Development

<u>Multiple Pathways of Exposure Model (MPE)</u>

The Multiple Pathways of Exposure model, formerly known as the Indirect Exposure Methodology (IEM), primarily consists of a set of multimedia fate and exposure algorithms developed by EPA's Office of Research and Development (ORD).[a] ORD issued an interim document describing this methodology in 1990, a major addendum was issued in 1993, and an updated guidance document was issued in 1999 in response to comments it received during a 1994 Science Advisory Board review of the addendum.[3] The MPE documentation describes fate and transport algorithms, exposure pathways, receptor scenarios, and dose algorithms.

The MPE approach includes procedures for estimating human exposures and health risks resulting from the transfer of emitted pollutants from air to soil and surface water bodies and the

[a] Note that the MPE model and many of its variations are conceptual models used to describe fate and transport, not "ready-to-run" computer models. Typically, users incorporate these conceptual models into spreadsheets or other computer frameworks to create a usable model.

subsequent uptake by vegetation, animals, and humans. The methodology specifically addresses exposures via inhalation; ingestion of food, water, and soil; and dermal contact. The MPE model was designed to predict long-term, steady-state impacts from continuous sources, rather than short-term, time-series estimates. It consists of a "one-way process" through a series of linked models and algorithms, beginning with the modeling of the transport of pollutant emissions in air and the subsequent deposition to soil and surface water and culminating in the uptake of the emitted pollutant(s) into biota. The aspects of the MPE model that address exposure estimation are described in more detail in Section 18.4 below.

EPA designed the MPE model to assess human exposures to air toxics emitted from stationary combustors, although analysts can apply most aspects of the approach to other types of stationary sources. One can apply this model to one or more sources at a single facility simultaneously to estimate exposures within 50 kilometers of the facility. The MPE model will allow modeling of only one chemical at a time, and there is no tracking (i.e., carry through the analysis) of transformation products of the modeled chemical. To apply the MPE approach, users must provide a significant number of site-specific inputs, such as source emission rate, wind speed and direction, soil loss constant, and pollutant degradation rate.

> The *Draft Guidance on the Development, Evaluation, and Application of Regulatory Environmental Models* recommends best practices to help determine when a model, despite its uncertainties, can be appropriately used to inform a decision. The Knowledge Base (KBase) is a web-accessible database of information on some of EPA's most frequently used models. The draft guidance recommends what information about models to document, while the Knowledge Base is the repository where this information is documented. Both products are available at the CREM internet site at http://www.epa.gov/crem.

EPA modified the MPE approach to multimedia fate and transport modeling for use in two additional EPA models and modeling approaches. These models and approaches are as follows.

- **IEM2M**. In 1997, Office of Air Quality Planning and Standards (OAQPS) modified the then-current version of the IEM model to create IEM2M. This revised version of IEM added the functionality necessary to model transformation between the three key species of mercury and track the concentrations throughout the modeled system for each of these species. This model was applied to estimate nationwide exposures to mercury for the *Mercury Study Report to Congress*.[4]

- **Human Health Risk Assessment Protocol (HHRAP)**. EPA's Office of Solid Waste and Emergency Response (OSWER) developed the HHRAP to provide guidance for conducting multipathway exposure and risk assessments of emissions of air toxics from hazardous waste combustion facilities. The suggested protocol for assessing multipathway exposures was adapted from the MPE approach and the documentation of this protocol[5] compiles detailed information on many of MPE's input parameters and algorithms.

Two additional models and approaches used by EPA to assess multipathway exposures to air toxics use many of the same fate and exposure algorithms and methodologies used in the MPE model.

- **Dioxin Reassessment Methodology**. Many of the algorithms used in the MPE model have been used for ongoing EPA efforts to characterize exposure and risks from dioxins, particularly chlorinated dibenzodioxins and dibenzofurans, as part of the Dioxin Reassessment project.[6]

- **Multimedia, Multipathway, Multi-receptor Exposure and Risk Assessment Model (3MRA)**. The 3MRA model is currently being developed by EPA's Office of Solid Waste and Emergency Response to support their Hazardous Waste Identification Rule (HWIR). Many of the fate and exposure algorithms used in 3MRA are similar to those used in MPE.

TRIM.FaTE

EPA developed the TRIM Fate, Transport, and Ecological Exposure (TRIM.FaTE) model[7] to describe the movement and transformation of pollutants over time, through a user-defined, bounded system of environmental compartments (i.e., abiotic media and organisms). The design of the compartment system can encompass spatial interconnections (with some similarities to grid-type Eulerian models) and ecological exposure-related relationships. TRIM.FaTE is designed to generate both media concentrations relevant to human pollutant exposures and exposure estimates relevant to ecological risk assessment primarily for air pollutants for which non-inhalation exposures are important.

In contrast to the IEM/MPE approach, TRIM.FaTE is a fully coupled multimedia model that estimates the flow of pollutant through time among environmental compartments. TRIM.FaTE offers the following important features that are not available using IEM/MPE.

- TRIM.FaTE is able to model mass-balanced "feedback" loops between media as well as secondary emissions (e.g., re-emission of deposited pollutants).

- TRIM.FaTE has the ability to provide detailed time-series estimates of pollutant concentrations in the environmental compartments.

- TRIM.FaTE maintains a full mass balance of the pollutant mass in the system (i.e., all the pollutant introduced into the system is accounted for among all the environmental compartments).

- TRIM.FaTE can model sensitivity of model results to variations in input parameters and perform probabilistic modeling such that uncertainty and variability in model results can be characterized.

- TRIM.FaTE is designed with the flexibility to allow for implementation of nearly limitless configurations (e.g., spatial resolution, types of biota), algorithms, and approaches. Simulations can range from quite simple analyses of pollutant distribution across abiotic media and biota to more complex, spatially-refined assessments, with associated implications with regard to user requirements.

TRIM.FaTE can model multimedia fate and transport of air toxics from any type of stationary source. It can be applied to multiple facilities, sources, and chemicals simultaneously to track the fate and transport of emitted pollutants as well as transformation products of the emitted

pollutants. The amount of input data required by TRIM.FaTE is directly related to the complexity of the user-specified modeling system; however, TRIM.FaTE analyses typically require more input data than similar analyses conducted using the MPE approach. As noted in Exhibit 18-2, TRIM.Expo is the exposure component of the TRIM modeling system (see Section 18.2.2.2).

California Total Exposure Model for Hazardous Waste Sites (CalTOX)

The Department of Toxic Substances Control (DTSC), within the California Environmental Protection Agency, has the responsibility for managing the State's hazardous-waste program. As part of this program, the DTSC funded the development of the CalTOX program.[2] CalTOX has been developed as a set of spreadsheet models and spreadsheet data sets to assist assessing human exposures and defining soil clean-up levels at uncontrolled hazardous wastes sites. More recently, CalTOX has been modified for use in establishing waste classification for landfills and hazardous waste facilities in California. CalTOX addresses contaminated soils and the contamination of adjacent air, surface water, sediments, and ground water. The modeling components of CalTOX include a multimedia transport and transformation model, exposure scenario models, and add-ins to quantify uncertainty and variability. The multimedia transport and transformation model is a dynamic model that can assess time-varying concentrations of pollutants introduced initially to soil layers or for pollutants released continuously to air, soil, or water. This model assists the user in examining how chemical and landscape properties impact both the ultimate route and quantity of human contact. Multimedia, multiple pathway exposure models are used in CalTOX to estimate average daily doses within a human population. The exposure modeling part of CalTOX is described further in Chapter 20.

18.3 Key Parameters/Inputs for Multimedia Models

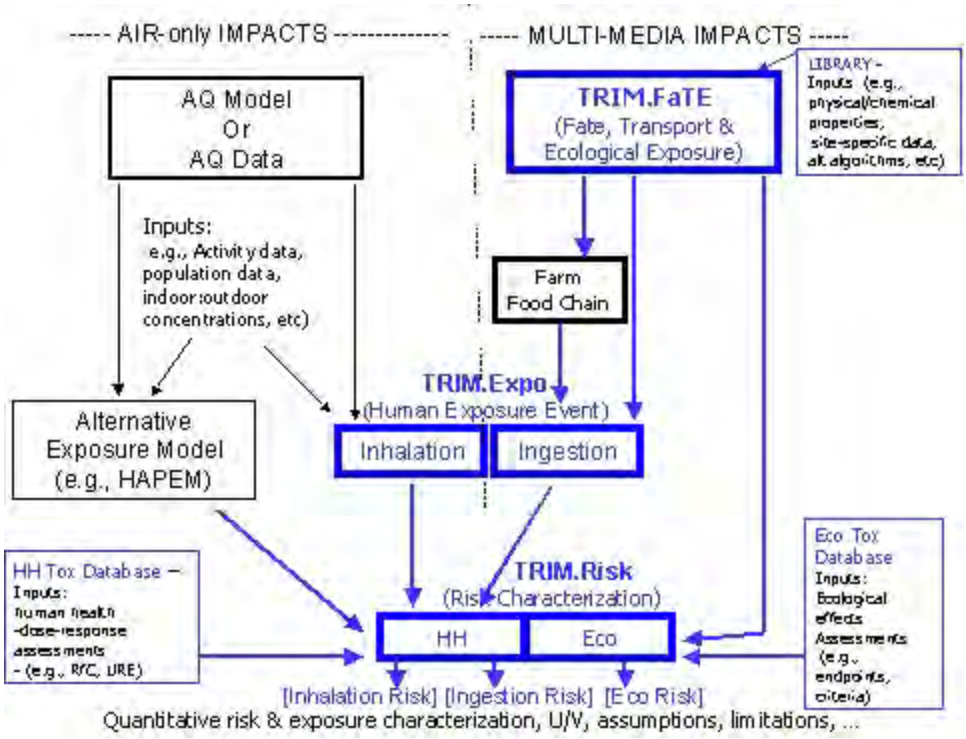
For most air risk applications, multimedia modeling results are strongly dependent on the **emission rate** of pollutants emitted to the air from the facility. For the MPE framework and TRIM.FaTE model, transport of modeled pollutants and accumulation in media of interest result directly from the emission of the chemical into the air from the facility, the dispersion or advection of chemical through the air, and the subsequent deposition of the chemical onto land, water, or other surfaces in the modeled region. In addition to emission rate, several other types of data are often required by multimedia models to characterize the pollutants and site being modeled. Generally, the data requirements for multimedia fate and transport models fall into the following categories.

- **Source characteristics** for the sources that are modeled, such as location, emission rates for the modeled pollutant(s), stack height, exit gas velocity, and exit gas temperature.

- **Environmental setting characteristics** for the abiotic media included in the modeling scenario, such as water body dimensions, surface soil characteristics (e.g., organic carbon content, porosity), and data related to local meteorology and hydrology (e.g., precipitation, erosion, runoff rates).

- **Abiotic chemical/physical data** for the chemicals included in the modeling scenario, such as Henry's law constant and soil-water partition coefficients. EPA's draft HHRAP provides default values for many of these parameters.[5]

Exhibit 18-2. Role of the TRIM Modeling System



The Total Risk Integrated Methodology (TRIM) modeling system can be used to assess human inhalation, human ingestion, and ecological risks. TRIM.FaTE accounts for movement of a chemical through a comprehensive system of discrete compartments (e.g., media and biota) that represent possible locations of the chemical in the physical and biological environments of the modeled ecosystem and provides an inventory, over time, of a chemical throughout the entire system. In addition to providing exposure estimates relevant to ecological risk assessment, TRIM.FaTE generates media concentrations relevant to human ingestion exposures that can be used as input to the ingestion component of the Exposure-Event module, TRIM.Expo. Measured concentrations also can be used as inputs to TRIM.Expo. In the inhalation component of TRIM.Expo, human exposures are evaluated by tracking randomly selected individuals that represent an area's population and their inhalation and ingestion through time and space. $\text{TRIM.Expo}_{\text{Inhalation}}$ can accept ambient air concentration estimates from an external air quality model or monitoring data. In the Risk Characterization module, TRIM.Risk, estimates of human exposures or doses are characterized with regard to potential risk using the corresponding exposure- or dose-response relationships. The TRIM.Risk module is also designed to characterize ecological risks from multimedia exposures. The output from TRIM.Risk is intended to include documentation of the input data, assumptions in the analysis, and measures of uncertainty/variability, as well as the results of risk calculations and exposure analysis. Information on TRIM can be accessed at: http://www.epa.gov/ttn/fera/.

- **Non-chemical-specific characteristics of biota** for any organisms included in the modeling scenario, such as feeding rates, body weight, and population density.

- **Biotic chemical-specific data** for any organisms included in the modeling scenario, such as bioaccumulation and/or bioconcentration factors or assimilation efficiency values.

The Multiple Pathways of Exposure (MPE) model,[8] a commonly used model for multipathway analyses, requires air concentrations, deposition rates, which are typically obtained via Industrial Source Complex (e.g., ISCST3) modeling (see Chapter 9 for descriptions of these models). Risk assessors would execute the ISCST3 modeling for multipathway in a similar fashion to how they executed the modeling for inhalation. Specifically, the sources would be characterized in the same way (e.g., vent height and diameter, release temperature and velocity, flow rate). The user would provide the inputs necessary to calculate the deposition rates properly (e.g., particle size distribution, scavenging coefficient). However, for multipathway analyses, the user should execute the ISCST3 model with the "depletion option" (i.e., telling the model to subtract out the mass of chemical deposited).

The user would need to know the particulate/particle-bound/vapor fractions of the emissions for ISCST3 to calculate wet and dry deposition of vapors and particles. These would probably be considered source-related, since although they are chemical-dependent, they also vary by source (i.e., the industrial process affects the emissions profile).

For dry deposition of particles, the user would supply the following inputs (in addition to the normal ISC inputs), including the:

- Array of particle diameters of the emissions;
- Array of mass fractions corresponding to the different particle diameters; and
- Array of particle densities corresponding to the different particle diameters.

For wet deposition of particles, the user would supply the following inputs (in addition to the normal ISC inputs), including the:

- Particle scavenging coefficients for liquid precipitation corresponding to the different particle diameters; and
- Particle scavenging coefficients for frozen precipitation corresponding to the different particle diameters.

For wet deposition of gases, the user would supply the following inputs (in addition to the normal ISC inputs), including the:

- Gaseous scavenging coefficient for liquid precipitation; and
- Gaseous scavenging coefficient for frozen precipitation.

The ISC user's guide[9] provides more detailed information on the deposition algorithms and required input data. There also is guidance for application of ISC for multipathway assessment in the latest MPE documentation.[8]

The only facility-related/source term data points used by the **TRIM Fate, Transport, and Ecological Exposure (TRIM.FaTE)** model are chemical emission rate, location (lat/long, UTM), and emission height, which are available from the inhalation modeling. TRIM.FaTE calculates all values internally for determining vapor/particle fractions and deposition rates based on chemical-specific (**not** source-specific) properties.

Other multimedia models may require specific source characterization data and other documentation that were not obtained for the inhalation analysis (the various user's guides for these models should be consulted for appropriate inputs).

It is important to note that the number and refinement of inputs to a multimedia model may vary depending on the outputs of interest and level of detail entailed in the modeling.

18.4 Examples of Multimedia Modeling

TRIM.FaTE Test Case Application. As a test case application, the TRIM.FaTE model was used to predict multimedia concentrations of mercury at a chlor-alkali facility in the northeastern United States. Speciated mercury concentrations were calculated for various abiotic media (e.g., surface soil, surface water, lake sediment) and biota (e.g., fish for various trophic levels, birds, mammalian predators) for the ecosystem surrounding the facility. A sensitivity and uncertainty analysis using TRIM.FaTE tools and a model comparison involving the 3MRA modeling system were also performed. The complete report on the test case will be available at the TRIM.FaTE page of EPA's Fate, Exposure, and Risk Analysis (FERA) website: http://www.epa.gov/ttn/fera/trim_fate.html.

Paints Hazardous Waste Listing Determination Analysis. On April 4, 2002, EPA issued a final determination not to list as hazardous certain wastes generated from the production of paint. EPA made this determination pursuant to the Resource Conservation and Recovery Act (RCRA), which directs EPA to determine whether certain wastes from the paint production industry may present a substantial hazard to human health or the environment. EPA proposed concentration-based listings for certain paint waste solids (K179) and liquids (K180) on February 13, 2001 (66 *Federal Register* 10060). However, following a review of the public comments and supplemental analyses based on public comments, EPA determined that the paint wastes identified in the February 13, 2001, proposal did not present a substantial hazard to human health or the environment. EPA conducted a multipathway risk assessment in support of this determination.[10] EPA used a series of models to estimate concentrations of Chemicals of Potential Concern (COPCs) in the environment with which human and ecological receptors may come into contact. The analysis used a source partioning model to estimate environmental releases of each COPC from a waste management unit for each waste stream, as appropriate. These estimated environmental releases provided input to the fate and transport models to estimate media concentrations in air, soil, surface water, and groundwater. A farm food chain model was used to estimate COPC concentrations in produce, beef, and dairy products. Aquatic bioconcentration factors were used to estimate concentrations in fish.

Chlorinated Aliphatics Hazardous Waste Listing Determination. In support of a hazardous waste listing determination for wastewaters and wastewater treatment sludges generated from the production of certain chlorinated aliphatic chemicals, EPA conducted a multipathway human health risk assessment.[11] EPA used the ISCST3 model to estimate dispersion and deposition of vapors emitted from wastewater treatment tanks and landfills, and vapors and particulates emitted from sludge land treatment units. EPA used a series of indirect exposure equations based on the MPE approach to quantify the concentrations of contaminants that pass from contaminated environmental media to the receptor indirectly. For example, EPA examined risks associated with contaminant transport in air; deposition onto plants and soil; accumulation in forage, grain,

silage, and soil; subsequent ingestion by beef cattle and dairy cattle; and human ingestion of contaminated beef and dairy products.

Hazardous Waste Combustor MACT Standard Analysis. A human health and ecological risk assessment was performed in support of developing a Maximum Achievable Control Technology (MACT) standard for hazardous waste combustor facilities.[12] The risk analysis included a multimedia, multipathway assessment that addressed direct exposures to constituents released into the atmosphere by hazardous waste combustor units and indirect exposures due to the movement of air toxics in the food chain. The risk assessment addressed both human health risks (cancer effects and noncancer effects) as well as ecological risks. Constituents assessed were seven congeners of chlorinated dioxin and 10 congeners of chlorinated furan; three species of mercury; 14 metals (antimony, chromium III, chromium VI, arsenic, lead, barium, nickel, beryllium, selenium, cadmium, silver, thallium, cobalt, copper, and manganese); particulate matter; hydrochloric acid; and chlorine gas. To the maximum extent possible, this risk assessment followed the latest risk guidelines adopted by EPA and used the most recent data available.

Columbus Waste-to-Energy Study. A risk assessment study using fate modeling was performed by EPA's NCEA for dioxin emissions at the Columbus, Ohio, Waste-to-Energy incinerator facility.[13] In 1994, EPA headquarters, the Office of Research and Development, and Region 5 conducted a screening assessment of indirect impacts, leading to the conclusion that continued emissions "may pose an imminent endangerment to public health and the environment." Fate modeling used to support EPA's position utilized the air-to-beef model described in the draft Dioxin Exposure document (i.e., based on the principles included in the MPE framework) and assumed a subsistence farming family scenario. Exposure pathways considered beef, milk and vegetable ingestion; soil dermal contact and childhood soil ingestion; and breast milk ingestion. The exposure duration for adults was assumed to be seventy years. Air concentrations used were the average from nine dairy farms located between five and twelve miles from the incinerator. Overall exposure and cancer risk were estimated for each of the exposure pathways, with cancer risk being highest for beef consumption (2×10^{-4}) and lowest for soil dermal contact (9×10^{-9}). Exposure from breast milk ingestion was determined to be higher by one order of magnitude than exposure from beef and milk consumption, and higher by two orders of magnitude than exposure from inhalation. Breast milk exposure near the incinerator site ranged between two and more than seven times the background dioxin levels. A TRIM.FaTE case study has been developed based on this analysis, including a direct model comparison component on the air-soil outputs, and will be available at: http://www.epa.gov/ttn/fera/trim_fate.html.

References

1. A summary of this review can be found in: U.S. Environmental Protection Agency. *TRIM.FaTE Technical Support Document, Volume I (Chapter 2).* Office of Air Quality Planning and Standards, Research Triangle Park, NC, September 2002. EPA/453/R-02/011a. Available at: http://www.epa.gov/ttnatw01/urban/trim/trimpg.html

2. McKone, T.E. 1993. *CalTOX, a Multimedia Total-Exposure Model for Hazardous wastes Sites Part I: Executive Summary*. UCRL-CR-11456, Pt. I. 1993b. *CalTOX, a Multimedia Total-Exposure Model for Hazardous Wastes Sites Part II: the Dynamic Multimedia Transport and Transformation Model*. UCRL-CR-111456, Pt. II. 1993c. *CalTOX, a Multimedia Total-Exposure Model for Hazardous Wastes Sites Part III: The Multiple-Pathway Exposure Model*. UCRL-CR-111456, Pt. III. Livermore, CA: Lawrence Livermore National Laboratory.

3. U.S. Environmental Protection Agency. 1990. *Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions*. *Interim Final*. Office of Health and Environmental Assessment, Washington, D.C., EPA/600/6-90/003.

   U.S. Environmental Protection Agency. 1998. *Methodology for Assessing Health Risks Associated with Multiple Pathways of Exposure to Combustor Emissions*. National Center for Environmental Assessment, Washington, D.C. EPA 600/R-98/137. Both are available at: http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=55525

4. U.S. Environmental Protection Agency. 1997. *Mercury Study Report to Congress*. Office of Air Quality Planning and Standards, Research Triangle Park, NC and Office of Research and Development, Washington, DC. EPA/452/R97/003. Available at: http://www.epa.gov/ttn/atw/112nmerc/mercury.html

5. U.S. Environmental Protection Agency. 1998. Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities, Peer Review Draft. Office of Solid Waste, Washington, D.C. EPA-530-D-98-001A. Available at: http://www.epa.gov/epaoswer/hazwaste/combust/risk.htm.

6. EPA is progressing toward completion of its comprehensive reassessment of dioxin exposure and human health effects entitled, *Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds.* The latest information on the status of the reassessment is available at: http://cfpub.epa.gov/ncea/cfm/dioxreass.cfm?ActType=default.

7. U. S. Environmental Protection Agency. 2002. *Total Risk Integrated Methodology. TRIM.FaTE Technical Support Document. Volume 1: Description of Module*. EPA-453/R-02-011a; *Volume 2: Description of Chemical Transport and Transformation Algorithms*. EPA/453/R-02/011b. *Evaluation of TRIM.FaTE. Volume 1: Approach and Initial Findings*. EPA/453/R-02/012; *TRIM.FaTE User's Guide*. Office of Air Quality Planning and Standards, Research Triangle Park, NC. These documents and information are available at: http://www.epa.gov/ttn/fera/trim_fate.html#current_user.

8. U.S. Environmental Protection Agency. 1998. *Methodology for Assessing Health Risks Associated with Multiple Pathways of Exposure to Combustor Emissions*. Office of Research and Development, National Center for Environmental Assessment, Cincinnati, OH, 1998. EPA/600/R-98/137. Available at: http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=55525.

9. U.S. Environmental Protection Agency. 1995. *User's Guide for the Industrial Source Complex (ISC) Dispersion Models. Volume 1 - User Instructions*. Office of Air Quality Planning and Standards, Research Triangle Park, NC, September 1995. EPA/454/B-95/003a. Available at: http://www.epa.gov/scram001/userg/regmod/isc3v1.pdf

10. U.S. Environmental Protection Agency. 2001. *Risk Assessment Technical Background Document for the Paint and Coatings Hazardous Waste Listing Determination*. Office of Solid Waste, Washington D.C., January 2001. (see also the February 28, 2002 addendum). Available at http://www.epa.gov/epaoswer/hazwaste/id/paint/index.htm.

11. U.S. Environmental Protection Agency. 1999. *Risk Assessment Technical Background Document for the Chlorinated Aliphatics Listing Determination*. Office of Solid Waste, Washington, D.C., July 1999. Available at: http://www.epa.gov/epaoswer/hazwaste/id/chlorali/clordoc1.htm. (see also the September 29, 2000 addendum to the risk assessment. Available at: http://www.epa.gov/epaoswer/hazwaste/id/chlorali/ca_risk.pdf).

12. U.S. Environmental Protection Agency. 1999. *Human Health and Ecological Risk Assessment Support to the Development of Technical Standards for Emissions from Combustion Units Burning Hazardous Wastes: Background Document, Final Report*. Office of Solid Waste, Washington, D.C., July 1999. Available at: http://www.epa.gov/epaoswer/hazwaste/combust/riskdoc1.htm.

13. Lorber, M. 2002. *Comments on the Columbus Waste-to-Energy Site*. Presentation at EPA Regional/ORD Workshop on Air Toxics Exposure Assessment. June 25 - 27, 2002. San Francisco, CA. National Center for Environmental Assessment. U.S. Environmental Protection Agency.

Chapter 19 Quantification of Exposure: Multimedia Monitoring

Table of Contents

19.1 Introduction 1

19.2 Why Monitor? 1

19.3 Planning and Implementing Issues 1

19.4 Monitoring and Sampling Methods, Technologies and Costs 3
19.4.1 Method Selection 3
19.4.2 Available Methods 7

References 12

19.1 Introduction

As noted earlier, modeling is generally the recommended approach for estimating exposure concentrations for air toxics risk assessments (for both inhalation and other pathways). However, there may be circumstances in which monitoring is requested or recommended for a particular multipathway risk assessment. This chapter provides an overview of multimedia monitoring, including the reasons for monitoring (Section 19.2), planning and implementation issues (Section 19.3), and available monitoring methods (Section 19.4).

19.2 Why Monitor?

The reasons for monitoring for a multipathway risk assessment are identical to those noted earlier for inhalation risk assessments (Chapter 10):

- Measuring existing concentrations of air toxics in specific locations (e.g., soils in a schoolyard) and/or food items (e.g., fish from a lake within the study area) for purposes of developing estimates of exposure;
- Developing or refining values for specific parameters needed by multimedia models;
- Evaluating the predictions of a model in specified circumstances (e.g., estimates of sediment concentrations resulting from deposition and runoff);
- Closing gaps that might be present in existing data (e.g., gaps in emissions inventory); and
- Providing compliance/enforcement information as to whether a given facility or set of sources is meeting regulatory or permit requirements.

Monitoring for Evaluation of Multimedia Modeling

For multipathway risk assessments, monitoring is a valuable tool for evaluating model predictions because multimedia modeling is more complicated and involves more uncertainties than does air quality modeling. When using samples to evaluate model predictions, however, it is important to realize that **monitored concentrations may be greater than model predictions** because sources other than those being modeled may have contributed to the contamination.

19.3 Planning and Implementing Issues

The planning and implementation processes for multipathway risk assessment monitoring programs are similar to those for air monitoring programs discussed in Chapter 10. The planning process involves a step-wise integration of data quality and data sampling and analysis processes that are consistent with the study-specific conceptual model (CM), quality assurance project plan (QAPP), and data quality objectives (DQO) process. Many of the general planning and implementation issues for air monitoring programs also apply to multimedia modeling. Some additional considerations arise because the sampling and analysis program might include soils, surface waters, sediments, fish, meat, vegetables, milk, and other human food items. The scale and scope of monitoring could be much greater (e.g., multiple media could be sampled), and issues specific to ingestion need to be considered (e.g., what parts of plants and animals do people eat?).

- **Monitoring or sampling methods should be appropriate for the compounds and environmental media to be measured**. They must have the sensitivity needed to monitor at the levels likely to be of health and/or regulatory concern.

- **Monitoring sites and frequency of monitoring should be appropriate for the spatial and temporal variation of the chemical being measured and the monitoring objective**. Typically, an exposure location (e.g., a water body, a property, an agricultural field) or source (e.g., milk from cows on a specific farm) is defined for the risk assessment. The monitoring program should be adequate to represent the spatial and temporal variation within the location or source, given the particular measure(s) used to support the risk management decision to be made (e.g., average exposure, maximum exposure). However, several aspects of spatial and temporal variation are unique to air toxics that persist and which also may bioaccumulate. For example:
  - The temporal patterns of releases from sources may be less important because the chemicals may slowly accumulate in media and biota over time;
  - Spatial "hot spots" of contamination may occur (for example, if soils erode and collect in low-lying areas);
  - Chemicals generally accumulate in different tissues at different rates; therefore, concentrations may be higher in certain parts of the plant or animal (which may or may not be the parts that people tend to eat, and vice versa);
  - Certain seasonal effects (e.g., growing season for plants, migratory movements in animals) may be important sources of variation; and
  - Age of the plant or animal being sampled may be important if it takes many months or years for contaminants to reach equilibrium in biological tissues (or if equilibrium is never reached). For example, mercury concentrations in fish tend to be higher in older, larger fish.

- **The monitoring effort should consider the relative contributions of the four main sources of variability in measurements**. As noted in Chapter 10, these are analytical, sampling, temporal, and spatial.

- **Standard operating procedures should be defined and followed** both in the field (during sample collection) and in the laboratory (during sample analysis). These include procedures related to sample collection, sample transport and storage (including prevention of sample degradation), sample analysis, "chain of custody," audits, data validation, and data reporting. These procedures may be quite varied due to the range of possible media and biota that could be sampled.

- **Limits of quantitation or detection should be determined and compared** against relevant decision needs, including health benchmarks and likely environmental levels.

- **Measurement processes should be properly calibrated** to ensure the accuracy of the method.

- **Results must be adequately recorded and archived**. The best monitoring program can be compromised by a failure to keep proper records that can be made part of the public record.

A periodic, random check of the archived records (e.g. computer files) should be made against "hard copies" to ensure the integrity of the process of recording the data.

Soil Depth: Issues for Sampling

The depth over which surface soils are sampled should reflect the type of exposure expected in the study area, the type of receptors expected in the study area, the depth of biological activity and the depth of potential contamination. Careful consideration of the size, shape, and orientation of sampling volume is important since they have an effect on the reported measured contaminant concentration values.[1] Selection of sampling design and methods can be accomplished by use of the Data Quality Objectives (DQO) process discussed in Chapter 6. Additional soil sampling guidance that may be consulted includes EPA's *Preparation of Soil Sampling Protocols: Sampling Techniques and Strategies* and *Guidance for Data Usability in Risk Assessment* and *Soil Screening Guidance*, available at: http://www.epa.gov/superfund/programs/risk/tooltrad.htm#dbhh.[2]

19.4 Monitoring and Sampling Methods, Technologies and Costs

19.4.1 Method Selection

Method selection for sample collection and analysis programs that are applicable to multipathway human health risk assessments are dependent on numerous aspects of the project. Factors such as media, sample types, sample program designs, lead regulatory authority, and concentration ranges of concern all can impact the selection of the appropriate methods. While it is not possible for this chapter to review all of the monitoring methods available for this broad range of applications, several of the more important factors that generally influence decisions on methods selected are discussed below.

The primary determining factor in selection of sample collection and analysis methodologies is the sample media to be evaluated. Exhibit 19-1 presents several examples of the types of media that might be sampled for a multipathway human health risk assessment.[a] Other factors that affect selection of sample collection methods are sample type and sample program design. Specific factors in selection of sample collection methods may also be construction material of the sampling devices, its design, decontamination, and proper use, site-specific conditions, relative cost, and data quality limitations.

Sample collection methods may be categorized by sample type as discussed in Chapter 10. However, the distinction is not always clear (e.g., a single fish tissue sample might be considered a grab sample because it is collected at a single location and time; however, because the contaminant concentrations in its tissues accumulate over time, the sample could also be considered a time-integrated sample). The more common types of samples used for non-air sampling are provided below:

- **Grab samples** (also known as discrete samples) are collected at a specific location (and generally instantaneous) time. Any technique where the sampling container is filled to represent a snapshot of the concentration of target contaminants at a single specific time is

[a] Air sampling also may be conducted; however, that is discussed in Chapter 10.

considered a grab sample. Where the population to be represented is demonstrated to be homogenous or consistent, grab samples provide the maximum information.

- **Time-integrated samples** are collected at a single location but over an extended period of time. Similar to grab samples, analysis of time-integrated samples provides a snapshot of that range of time and location as a single value. Only the total pollutant collected is measured, and so only the average concentration during the sampling period can be determined.

Exhibit 19-1. Examples of Environmental Media that May be Sampled for Multipathway Human Health Risk Assessment [(a)]	
Medium	**Comments**
Surface Water	Generally sampled only if used as a drinking water source
Soils	Generally sampled within the top few inches of the surface, where ingestion exposure or erosion may occur, but other considerations may require a different soil sampling depth
Sediments	Generally sampled to support assessments of bioaccumulation in aquatic systems (in more rare instances, sediments might support a dermal assessment of exposure to the sediments themselves)
Fish	Generally focused on species and parts of the fish that people eat (although this may vary regionally; e.g., some native cultures may routinely eat the entire fish)
Vegetables and other crops	Generally focused on the plants and parts of plants that people eat and/or are fed to livestock
Dairy products and other foods	Generally focuses on milk and other dairy products; eggs and meat are also sometimes evaluated
[(a)] Note that this list is not exhaustive; additional types of samples might be appropriate for a given risk assessment.	

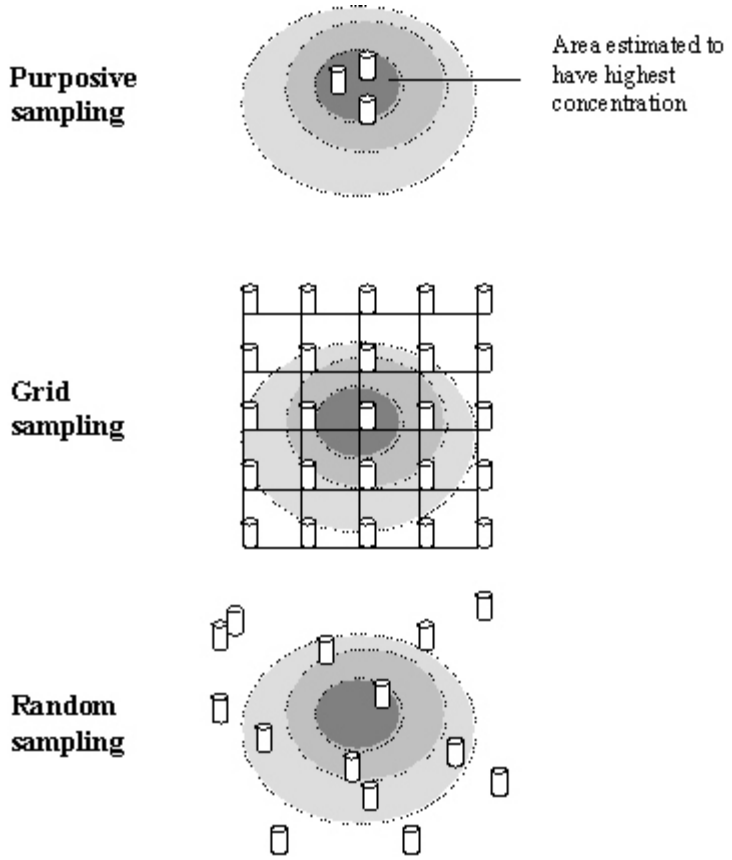
- **Composite samples** represent combinations of discrete samples, which may be collected either at different times or from different locations, that are combined into a single sample for analysis. Composite samples can be helpful when the amount of material that can be obtained from a single sample is very small (e.g., fish tissue), and the analytical quantitation limit can be lowered if the sample mass is increased (e.g., by combining multiple samples into a single composite sample for analysis). Composite samples also can be helpful when resources for laboratory analysis are limited, as they provide an estimate of average concentration across multiple samples, with the analysis cost of only one sample. The greatest drawback to composite samples is that they do not allow for an understanding of the variation in concentration values among the individual samples collected.

- **Continuous samples** provide essentially real-time measurements over time from a single, specific location. Continuous measurements typically involve real-time measurements, because samples cannot be practically collected to provide true continuous data. Continuous monitoring data frequently are evaluated as a function of concentration over the time period

analyzed. Depending on the application, maximum, median, time-weighted average, or distribution curves may be applied to reduce the large amount of results obtained from true continuous data to usable results which can be compared to decision criteria.

Sample collection methods may also be determined by the sample collection design methodology (Exhibit 19-2). Sample design impacts method selection often by determining the number of samples being collected.

Exhibit 19-2. Common Types of Sampling



Purposive sampling focuses the sampling effort in specific locations (in this example, the area estimated to have the highest concentration). Grid sampling consists of regularly-spaced samples in a predetermined grid. Random sampling consists of samples in locations selected by chance.

- **Purposive** sampling involves focused sample collection based on previous knowledge of release event locations. Purposive (also called biased) sampling is named such because the person taking the sample willfully takes that sample at a time or place where, based on prior knowledge, it is expected that concentrations will generally be biased high. Purposive sampling may be desired in programs looking to verify expected model results. Purposive sampling often targets maximum contaminant conditions to evaluate maximally impacted areas. However, it may be used for reasons such as targeting specific species to calibrate bioaccumulation models or defining the spatial extent of contamination.

- **Systematic** sampling consists of collecting samples at locations and times according to specific patterns (e.g., grid sampling). Systematic sampling may use previous knowledge to set frequency, density, or coverage of sampling.

- **Random** sampling involves collecting samples from locations in a manner such that each location has an equal probability of being sampled and analyzed. Random sample collection designs are an important aspect of certain statistical data evaluations.

The factors which primarily affect selection of preparation and analysis methods include target contaminants, required reporting limits (i.e., concentration range of decision criteria), number of samples, data quality limitations, method/instrument portability, previous data comparability, acceptance/approval by regulators and stakeholders, and relative cost and availability.

- **Target contaminants**. The specific contaminants being sampled may have a significant impact on both budget and overall approach. For example, sampling and analytical procedures for metals are different than those for organic chemicals. Careful evaluation before inclusion of unwarranted parameters and establishment of a procedure for identification and removal of chemicals of potential concern (COPCs) is critical to an effective monitoring program.

- **Required reporting limits**. Assessors should select analytical methods so that the reporting limits (usually the estimated quantitation limits) are less than the effects concentrations of interest. If the assessor does not select an adequately sensitive analytical method, the quantitation limit for a given chemical could exceed the chemical's effects benchmark concentration of interest; in that case, monitoring information would not provide meaningful input to the risk assessment.

- **Number of samples**. A sampling program that involves screening-level assessment of a large number of samples may drive selection of certain methods for the bulk of samples in order to allocate limited resources. In the opposite case, determination of low heterogeneity of sample media, and extremely low risk-based concentrations of interest as decision criteria may require fewer samples and more highly sophisticated methodologies.

- **Data quality limitations**. High data quality requirements imposed by high uncertainty or other factors may influence the choice of sampling methods such as procedures that are more stringent and more costly than usual procedures.

- **Method/instrument portability**. In-field or on-site analysis has begun to replace laboratory-based analysis in many monitoring programs. Certain preparation and analysis methodologies are more portable than others, in part because of the sensitivity of the instrumentation. However, considerable expertise in sampling and analysis is needed to decide whether in-field or laboratory-based analysis is appropriate for the study.

- **Previous data comparability**. Previous data sets can affect selection of appropriate methods. All other factors being equal, data comparability goals and objectives are more easily met by use of consistent methods.

- **Stakeholder input**. Stakeholder preferences may influence method selection.

- **Relative Cost/Availability**. The reality of limited resources often impacts method selection. Certain monitoring methods are commonly performed and available at numerous laboratories or by readily available field instrumentation. Other more obscure methods may better meet the needs of the project but are only available from highly specialized laboratories. In addition to cost impact, low availability of some specific monitoring methods can impact data quality due to lack of practice, market competition, appropriate standards, or certifications.

19.4.2 Available Methods

Hundreds of specific sampling, test, analysis, and quality assurance methods and procedures exist for soil, water, sediment, and biota. The list of available methods changes frequently as new methods are introduced and older methods are retired. It is not possible for this chapter to review all of the monitoring methods available. Instead, this section provides an overview of several key EPA resources and provides a listing of web sites that serve as sources of additional information. Key EPA resources include the *EPA Test Methods Index*; the Contract Laboratory Program (CLP); and the Fish and Wildlife Advisories Program.

- ***EPA Test Methods Index*** (http://www.epa.gov/epahome/Standards.html). EPA has developed hundreds of specific sampling, test, analysis, and quality assurance methods and procedures. In response to frequent requests for agency test methods, Region 1 Library staff developed a methods index as a tool to help locate copies. Confirming that there was no single volume containing all agency methods and no comprehensive list of them, the project commenced and in 1988 printed the first *EPA Test Methods Index*.[3] It has been updated periodically to reflect new procedures and revoked methods, and the current edition includes about 1,600 method references. The index includes only EPA methods, and its primary goal remains as a reference tool to identify a source from which the actual method can be obtained, either free or for a fee.

- **EPA Contract Laboratory Program**. The Contract Laboratory Program (CLP) is a national network of EPA personnel, commercial laboratories, and support contractors whose fundamental mission is to provide data of known and documented quality, primarily for the Superfund program (http://www.epa.gov/superfund/programs/clp/about.htm). The Analytical Operations/Data Quality Center (AOC) provides several tools to assist CLP clients, laboratories, and samplers (http://www.epa.gov/superfund/programs/clp/tools.htm). These tools were designed to use the Internet to facilitate many of the essential functions of the CLP.

Available Guidance from EPA's Contract Laboratory Program

Contract Laboratory Program National Functional Guidelines for Low Concentration Organic Data Review EPA-540-R-00-006 June 2001

Contract Laboratory Program National Functional Guidelines for Organic Data Review EPA-540/R-99-008 (PB99-963506) October 1999

Contract Laboratory Program National Functional Guidelines for Inorganic Data Review EPA 540-R-01-008 July 2002

Contract Laboratory Program National Functional Guidelines for Chlorinated Dioxin/Furan Data Review EPA-540-R-02-003 August 2002

Contract Laboratory Program Guidance for Field Samplers (Draft-Final) EPA-540-R-00-003 April 2003

This information, as well as methodology information is available from the CLP at: http://www.epa.gov/superfund/programs/clp/services.htm

- **EPA's Fish and Wildlife Advisories Program** (http://www.epa.gov/waterscience/fish/). EPA's Office of Science and Technology provides technical and outreach material that support efforts by state, local, and tribal (S/L/T) governments to protect their residents from the health risks of consuming contaminated noncommercially caught fish. S/L/T governments do this by issuing consumption advisories for the general population as well as for specific vulnerable sub-populations. These advisories tell the public when high concentrations of chemical contaminants have been found in local fish. They also include recommendations to limit or avoid eating certain fish species from specific water bodies or water body types. The program also provides *Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories* (http://www.epa.gov/waterscience/fish/guidance.html), a set of four volumes that provides guidance for assessing health risks associated with the consumption of chemically contaminated non-commercial fish and wildlife. The set includes Third Editions of Volume 1: *Fish Sampling and Analysis* and Volume 2: *Risk Assessment and Fish Consumption Limits*.

Exhibit 19-3 provides links to information on specific sampling and analysis methods, summarized from key EPA compendia of methods. Methods are divided into four categories (General, Analytical Method Index, Sample Collection, and Quality Assurance). Keywords are added to help readers get to the area they are concerned with. Additional effort may be required to "drill into" each site to view the relevant information. These links generally are limited to government sites. Some non-EPA sites are included (e.g., Occupational Safety and Health Administration (OSHA), National Institute of Standards and Technology (NIST), and National Institute for Occupational Safety and Health (NIOSH)) to help fill specific information gaps.

Exhibit 19-3. Sources for Information on Specific Sampling and Analysis Methods	
Keywords	**Description and URL Link**
General References	
Sample collection, analysis method, criteria, water	General EPA Water page with links to analytical methods, sampling guidance, and criteria for assessment of contamination. http://www.epa.gov/waterscience/
Analysis methods	EPA' s Office of Ground Water and Drinking Water (OGWDW) links to analysis methods. http://www.epa.gov/OGWDW/methods/methods.html
Sample collection, analysis methods, reference	NIOSH pocket guide to chemical hazards contains information by analyte which can support field sample collection, analysis, and determination of relevant criteria. http://www.cdc.gov/niosh/npg/npg.html
Sample collection, analysis methods, reference	NIST web book contains information by analyte which can support field sample collection, analysis, and basic chemical parameters from thermodynamic constants to reference mass spectra. http://webbook.nist.gov/chemistry/
Sample collection, analysis methods, reference	General EPA environmental test methods and guidelines page with numerous links to other areas of information throughout EPA web sites. http://www.epa.gov/epahome/Standards.html
Analysis Method Index	
Analysis methods, sample collection	Region I list of methods available as hardcopy and partial links to analysis methods. http://www.epa.gov/epahome/index/
Analysis methods, sample collection	Searchable online database of analysis methods. NEMI is a project of the National Methods and Data Comparability Board, a partnership of water quality experts from Federal agencies, States, Tribes, municipalities, industry, and private organizations supported by EPA and the U.S. Geological Survey. http://www.nemi.gov
Analysis methods, sample collection	National Exposure Research Laboratory (NERL) formerly EMSL, Manual of Manuals links to information about analysis methods; summaries and ordering information for eight laboratory analytical chemistry methods manuals published by the former Environmental Monitoring Systems Laboratory-Cincinnati (EMSL-Cincinnati) between 1988 and 1995. http://www.epa.gov/nerlcwww/methmans.html
Analysis Methods	
Analysis methods, water	EPA's Office of Water link to analysis methods. Laboratory analytical methods that are used by industries and municipalities to analyze the chemical and biological components of wastewater, drinking water, sediment, and other environmental samples that are required by regulations under the authority of the Clean Water Act (CWA) and the Safe Drinking Water Act (SDWA). http://www.epa.gov/waterscience/methods/
Analysis methods, water, 601, 602, 603, 604, 605, 606, 607, 608, 609, 610, 611, 612, 613, 624, 625, 1624, 1625	Methods for organic chemical analysis under the authority of the Clean Water Act (CWA) and the Safe Drinking Water Act (SDWA). http://www.epa.gov/ostwater/methods/guide/methods.html

Exhibit 19-3. Sources for Information on Specific Sampling and Analysis Methods	
Keywords	**Description and URL Link**
Analysis methods, drinking water	Recent drinking water methods from EPA's Office of Research and Development, National Exposure Research Laboratory (NERL), formerly the Environmental Monitoring Systems Laboratory (EMSL). http://www.epa.gov/nerlcwww/ordmeth.htm
Organic, analysis methods, drinking water	Organic method index with hyperlink to method by analyte in drinking water as maintained by Office of Ground Water and Drinking Water. http://www.epa.gov/OGWDW/methods/orch_tbl.html
Inorganic, metal, analysis methods, drinking water	Inorganic and metal analysis methods in drinking water as maintained by Office of Ground Water and Drinking Water. http://www.epa.gov/OGWDW/methods/inch_tbl.html
Analysis methods, drinking water, radionuclides	Radionuclides in drinking water as maintained by Office of Ground Water and Drinking Water. http://www.epa.gov/OGWDW/methods/rads.html (EPA) http://www.epa.gov/OGWDW/methods/indrads.html (non-EPA)
Analysis methods, drinking water,	Approved methods for unregulated contaminants in drinking water as maintained by Office of Ground Water and Drinking Water. http://www.epa.gov/OGWDW/methods/unregtbl.html
Analysis methods, drinking water,	Secondary contaminants in drinking water as maintained by Office of Ground Water and Drinking Water. http://www.epa.gov/OGWDW/methods/2nd_tbl.html
Analysis methods, immunoassay	Region 1 guidance on immunoassay methods. http://www.epa.gov/region1/measure/ia/iaguide.html
Analysis methods, CLP, organic, dioxin, inorganic, water, soil	Contract Laboratory Program (CLP) methods for organics, inorganics, and dioxins/furans. http://www.epa.gov/superfund/programs/clp/methods.htm
Analysis methods, air	EPA Emissions Measurement Center (EMC) for methods related to determination of airborne pollutants. http://www.epa.gov/ttn/emc/
Analysis methods, pesticide, soil, water	EPA' Office of Pesticide Programs (OPP) database of environmental chemistry, residual, and antimicrobial analysis methods. http://www.epa.gov/oppbead1/methods/
Analysis methods, water, soil, sediment, waste, air	EPA's OSWER provides online updated SW-846 waste sampling and analysis methods manual which is the source of many related methods used in environmental sampling and analysis. http://www.epa.gov/epaoswer/hazwaste/test/main.htm
Sample collection, analysis methods, air	Occupational Safety and Health index of sampling and analysis methods alphabetically by parameter and general information on selection of methods and laboratories. http://www.osha-slc.gov/dts/sltc/methods/index.html
Sample collection, analysis methods, air	EPA's Organic (TO) Compendium of methods for air toxics and EPA's Inorganic (IO) Compendium methods. http://www.epa.gov/ttn/amtic/airtox.html
Sample Collection	
Sample collection, analysis, fish, shellfish, biota	Methods for sampling and analyzing contaminants in fish and shellfish tissue. http://www.epa.gov/waterscience/fishadvice/volume1/index.html

Exhibit 19-3. Sources for Information on Specific Sampling and Analysis Methods	
Keywords	**Description and URL Link**
Sample collection	Current manuals and protocols prepared by NERL-Cincinnati scientists. NERL is the EPA's scientific lead for the following stream and source monitoring indicators: fish, macroinvertebrates, periphyton, zooplankton, functional ecosystem indicators, water and sediment toxicity and fish tissue contaminants. As part of their indicator lead responsibilities NERL-Cincinnati scientists prepare and update field and laboratory protocol and methods manuals for these indicators. http://www.epa.gov/nerleerd/methman.htm
Sample collection, monitoring wells, low stress	Guidance for RCRA/Superfund groundwater sample collection methodologies and the logical process for determining an approach fit to site specifics. http://www.epa.gov/tio/tsp/download/gw_sampling_guide.pdf
Sample collection, monitoring wells, low stress	Generally well accepted low stress (low flow) ground water sample collection guidance from EPA Region I. Several versions exist across EPA regions and within other governmental and State guidelines. http://www.epa.gov/region1/measure/well/wellmon.html
Sample collection, field analysis	EPA Environmental Response Team provides numerous sampling and field analysis Standard Operating Procedures (SOPs) often encountered in environmental responses including otherwise atypical sample collections SOPs such as drum, wipe, and waste pile sampling techniques. http://www.ertresponse.com/sops.asp
Sample collection, field analysis, program design	EPA's Office of Technology Innovation provides a web site with information on proper sampling program design, QA/QC concerns, and use of field methodologies to expedite information collection without loss of data quality. http://clu-in.org
Quality Assurance	
Quality assurance	EPA Agency-wide quality system documents for EPA and non-EPA organizations plus general guidance. Documents are available as PDFs. http://www.epa.gov/quality/qa_docs.html
Quality assurance	Region I guidance includes quality assurance documents. http://www.epa.gov/region1/lab/qa/qualsys.html

References

1. U.S. Environmental Protection Agency. 2000. *Draft Ecological Soil Screening Level Guidance*. Office of Emergency and Remedial Response. July 10, 2000.

2. U.S. Environmental Protection Agency 1992. *Guidance for Data Useability in Risk Assessment (Part A)*. Office of Emergency and Remedial Response, Washington, D.C., April 1992. Publication 9285.7-09A, PB92-963356. Available at: http://www.epa.gov/superfund/programs/risk/datause/parta.htm

   U.S. Environmental Protection Agency 1992. *Preparation of Soil Sampling Protocols: Sampling Techniques and Strategies*. Office of Research and Development. EPA/600/R-92/128. Available at: http://www.epa.gov/superfund/programs/risk/tooltrad.htm#dbh

   U.S. Environmental Protection Agency 1996. *Soil Screening Guidance: User's Guide*. Office of Solid Waste and Emergency Response. Washington, D.C., July 1996. EPA540/R-96/018. See especially Attachment B, *Soil Screening DQOs for Surface Soils and Subsurface Soils.* Available at: http://www.epa.gov/superfund/resources/soil/index.htm#user

   U.S. Environmental Protection Agency. 2002. *Supplemental Guidance For Developing Soil Screening Levels for Superfund Sites*. Office of Solid Waste and Emergency Response, Washington, D.C., December 2002. OSWER 9355.4-24. Available at: http://www.epa.gov/superfund/programs/risk/tooltrad.htm#dbh

3. U.S. Environmental Protection Agency. 1988. *EPA Test Methods Index*. EPA 901/388/001.

Chapter 20 Exposure Metrics for Multimedia Assessment

Table of Contents

20.1 Introduction 1

20.2 Generic Equation for Dietary Intake 3

20.3 Estimating Exposure Concentrations 4

20.4 Calculating Intake Variable Values 6
- 20.4.1 Consumption Rate 7
- 20.4.2 Exposure Frequency 10
- 20.4.3 Exposure Duration 10
- 20.4.4 Body Weight 12

20.5 Calculating Averaging Time Value 12

20.6 Combining Exposure Estimates Across Pathways 13

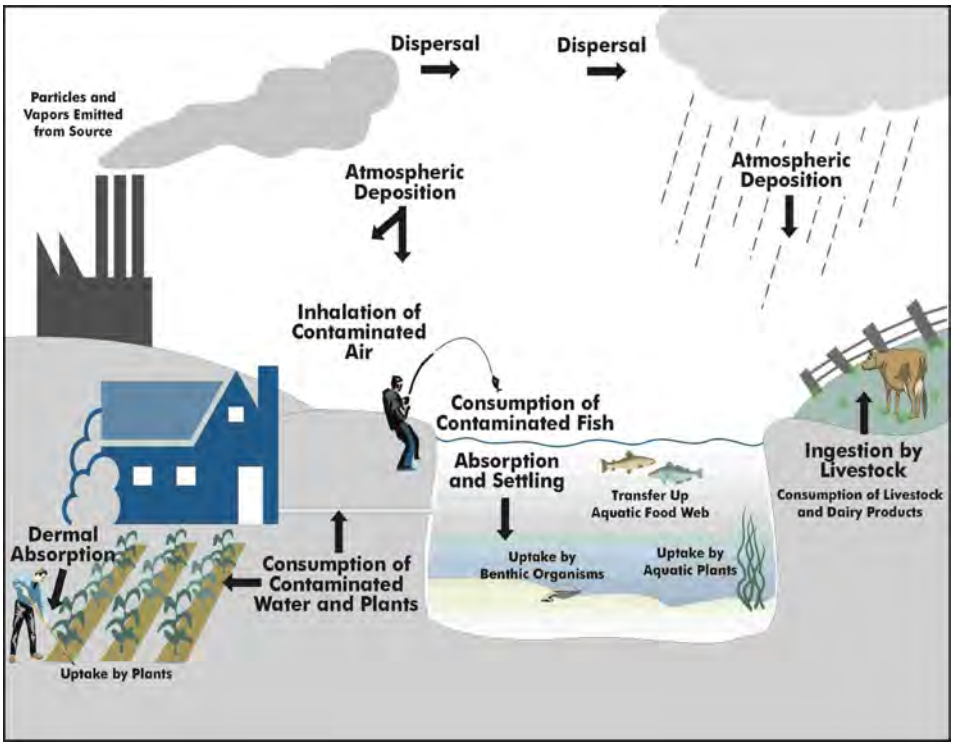
20.7 Exposure Models 14

References 16

20.1 Introduction

This chapter concludes the exposure assessment component of the multipathway risk assessment by describing how to develop estimates of intake (i.e., the metric of exposure) for the ingestion pathways selected for analysis. Estimates of chemical intake via the inhalation pathway were presented in Chapter 11. Exhibits 14-2 and 20-1 provide an overview of the potential multimedia exposure pathways by which air toxics that persist and potentially bioaccumulate may reach ecological and human receptors, respectively. Determination of chemical intake via the ingestion exposure route combines the estimates of chemical of potential concern (COPC) levels in food items and drinking water (discussed in Chapter 7) with estimates of consumption rates (food, water), exposure frequency and duration, averaging time, and body weight to derive estimates of the **chemical intake rate** (expressed generally as mg/kg-day).[1]

Exhibit 20-1. Potential Multimedia Exposure Pathways of Concern



This graphic illustrates many of the potential multimedia pathways of concern for air toxics. Air toxics released from a source disperse through the air and eventually fall to the earth (atmospheric deposition) via settling and/or precipitation. Air toxics deposited to soil may be absorbed by plants that are then harvested for human consumption. Humans may be exposed via ingestion of contaminated plants and soils, or by consuming contaminated terrestrial animals (e.g., beef, for those air toxics that bioaccumulate and transfer up the terrestrial food web). Air toxics deposited to water may be dissolved in the water column and/or may settle and be absorbed into aquatic sediments. Air toxics in sediments and the water column may be absorbed by aquatic plants (uptake). Aquatic organisms (e.g., fish) may be exposed directly to air toxics in the water column and/or by consuming other contaminated aquatic organisms (for those air toxics that bioaccumulate and transfer up the aquatic food web) or sediments. People may be exposed to air toxics by eating contaminated aquatic plants, fish, or shellfish and/or by drinking contaminated water. Note also that, while in the atmosphere, air toxics may also have direct impacts to humans via inhalation.

Chapter 7 described two general approaches for deriving the exposure concentration (EC) for an inhalation risk assessment: (1) use of ambient air concentrations as a surrogate for the EC, and (2) exposure modeling that combines estimates of ambient air concentrations with information about the population of interest, including the types of people present (e.g., ethnicity, age, sex), time spent in different microenvironments, and microenvironment concentrations. **The first approach (i.e., use of ambient concentrations in abiotic media such as soil, water, or sediments) generally is not used for multipathway air toxics risk assessments. Instead, a multipathway exposure assessment must involve some type of exposure modeling** (e.g., at a minimum simple scenarios to characterize persons who are exposed and the amount and duration of their contact with the abiotic and biotic media).

Note that EPA has derived some human health screening-level concentration benchmarks for surface water and soil (i.e., the Office of Water's Ambient Water Quality Criteria for the Protection of Human Health,[2] and the Superfund Program's soil screening levels[3]). However, these human health benchmarks are based on specific scenarios (e.g., how much water a person drinks each day, how much they weigh) that were selected to meet different programmatic goals and statutory requirements. Therefore, the scenarios on which these benchmarks are based may not be appropriate for a specific air toxics risk assessment.

The way a chemical enters the body and eventually reaches the target organ is a complex process (see box below). For most chemicals, however, it is not necessary to quantify anything beyond the chemical **intake rate**, because the dose-response value (e.g., Reference Dose [RfD] or Cancer Slope Factor [CSF]) is also based only on the amount of chemical ingested and not the amount of chemical that has been absorbed into the bloodstream.

Exposure and Intake via Ingestion

The process of a chemical entering the body can be described in two steps: **exposure** (contact), followed by **entry** (crossing the boundary). **Intake** involves physically moving the chemical in question through an opening in the outer boundary (usually the mouth), typically via eating or drinking. Normally the chemical is contained in a medium that comes into contact with the body, such as food or water, and the concentration of the chemical at this point of contact is called the **exposure concentration**. The estimate of how much of the chemical enters into the body is based on how much of the carrier medium enters the body. The **chemical intake rate** is the amount of chemical crossing the outer boundary per unit time, and is the product of the exposure concentration times the ingestion rate. **Ingestion rate** is the amount of the carrier medium crossing the boundary per unit time, such as the number of kilograms of food ingested/day or liters of water consumed/day. Ingestion rates typically are not constant over time (they can vary over time and among individuals) and are usually given (for deterministic analyses) as an average intake rate over some period of time. In addition, the intake rates are usually normalized to body weight. Thus, a common intake rate would take the form of milligrams of pollutant ingested per kilogram of body weight per day (or mg/kg-d). A different ingestion rate would be developed for each type of person in the population under study. For example, one intake rate could be developed to represent the average adult (male and female) while a separate intake rate could be developed to represent children between the ages of birth to four years old.

The remainder of this chapter focuses on how to quantify ingestion exposure (intake) for multipathway air toxics risk assessments. The corresponding chapter for inhalation analyses

(Chapter 11) discusses how to evaluate uncertainty in the exposure assessment and how to present the exposure assessment results; this applies to all exposure evaluations (i.e., inhalation and ingestion).

20.2 Generic Equation for Dietary Intake

Equation 20-1 is the generic equation used to calculate dietary chemical intake:[4]

$$I = \frac{EC \times CR}{BW} \times \frac{EF \times ED}{AT} \qquad \text{(Equation 20-1)}$$

where

I = Chemical intake rate, or the amount of pollutant ingested per unit time per body weight (mass), expressed in units of mg/kg-day. For evaluating exposure to noncarcinogens, the intake is referred to as Average Daily Dose (*ADD*); for evaluating exposure to carcinogenic compounds, the intake is referred to as Lifetime Average Daily Dose (*LADD*).

Chemical-related variable:

EC = Exposure concentration of the chemical in the medium of concern for the time period being analyzed, expressed in units of mg/kg for soil and food or mg/L for surface water or beverages (including milk).

Variables that describe the exposed population (also termed "intake variables"):

CR = Consumption rate, the amount of contaminated medium consumed per unit of time or event (e.g., kg/day for soil and L/day for water).
EF = Exposure frequency (number of days exposed per year).
ED = Exposure duration (number of years exposed).
BW = Average body weight of the receptor over the exposure period (kg).

Assessment-determined variable:

AT = Averaging time, the period over which exposure is averaged (days). For carcinogens, the averaging time is 25,550 days, based on an assumed lifetime exposure of 70 years; for noncarcinogens, averaging time equals *ED* (years) multiplied by 365 days per year.

The values of some exposure factors depend on site conditions as well as the characteristics of the potentially exposed population (e.g., child vs. adult). Because of differences in physiology and behavior, exposures among children are expected to be different than exposures among adults. For example, body weight and consumption rate differ for children and adults. For the evaluation of non-carcinogenic effects, intakes for children generally are estimated separately (often for ages 0-6) than for adults (often from ages 6-beyond). For the evaluation of carcinogenic effects, intake estimates are averaged over the assumed lifetime (70 years).

20.3 Estimating Exposure Concentrations

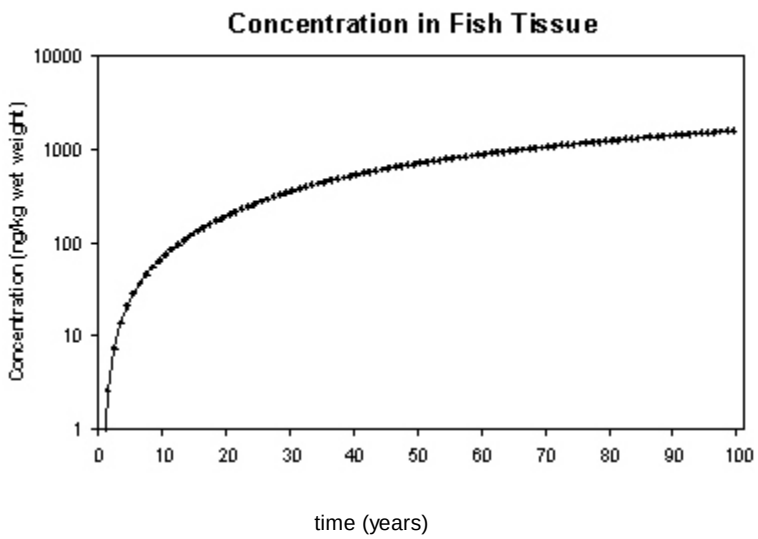
The exposure concentration for a chemical is calculated separately for each food item and environmental medium of concern. The value of these variables may be determined by modeling (Chapter 18), monitoring (Chapter 19), or a combination of both. The specific algorithms for determining these concentrations will depend on the specific models and/or sampling and analysis techniques used. For example, EPA has developed methodologies for estimating EC values in soil, water, sediment, and various food items for releases from hazardous waste combustion facilities (see Appendix L).[5]

For ingestion pathways, the specific media concentration values obtained from a multimedia modeling simulation for use in deriving exposure concentrations depends on several important decisions made during problem formulation, including:

- Choice of modeling duration for a model run;
- Choice of the year or years of the model run on which to base the EC; and
- Choice of a specific ED.

Exhibit 20-2 presents several different examples relevant to different purposes/objectives for an assessment.

Exhibit 20-2. Example Decisions in Assessing Exposures Resulting From Distribution of Air Toxics into Other Media



In this hypothetical example, a modeling analysis was used to predict the concentrations of a persistent bioaccumulative hazardous air pollutant (PB-HAP) in fish tissue during a 100-year emissions scenario (annual average was estimated each year and is plotted here using a logarithmic scale). As discussed below, the exposure scenario assessed will reflect several key choices including:

(1) choice of modeling duration for model run;
(2) choice of year or years of model run on which to base EC (i.e., the model outputs); and
(3) choice of specific ED.

Exhibit 20-2 (continued)

The modeling duration is a separate decision from the ED and is not related to the average human lifespan.

Note that in this example, the analyst assumed that the starting concentration was zero (i.e., the tissue concentrations reflect only the sources being modeled). Some multimedia models (e.g., TRIM.FaTE) can start with an initial concentration.

Modeling Duration. The analyst can choose to run a multimedia model for any period of time. Duration will usually be chosen to reflect the expected duration of emissions from the source(s) being evaluated or, perhaps, that duration expected in order to reach steady-state conditions. A common duration is 30 or 40 years (e.g., the expected lifespan of many facilities or processes). For this example, a 100-year duration was selected.

Selection of Model Outputs. Usually the modeling duration will have been chosen with consideration of the model outputs on which the exposure scenario is to be based and the exposure duration. Some common examples follow:

- **Year of maximum concentration**. Screening-level analyses often use the maximum concentration reached during the modeling period which, for a constant emissions scenario, will usually be the final year of the modeling simulation. For this example (see figure), it would be the 100th year (at such time as the fish concentration was approximately 2,000 ng/kg).
  - **Exposure Duration.** With use of the maximum model result, the analysis presumes no change in fish concentration over the exposure duration (i.e., in this example EC = 2,000 ng/kg throughout the exposure period).

- **Initial years of simulation.** In this case the exposure being assessed is that beginning with initiation of emissions and extending through the duration selected for assessment.
  - **30-year Exposure Duration**. In this case, the analyst is basing the exposure duration near the 95 percentile of how long people live in the same home.[(6)] If the analyst chose to examine changing concentrations over time, the ECs would vary, reflecting the concentration outputs from the first 30 years of the modeling duration.
  - **70-year Exposure Duration.** In this case, the analyst is using a lifetime exposure assumption. The exposure scenario then may be based on the model outputs from the first 70 years of the modeling duration.

- **Last years of simulation.** In this case, the exposure being assessed is that which occurs during the ending years of the simulation, with the number of years involved equal to the exposure duration selected for assessment.
  - **30 -year Exposure Duration.** For this ED, the ECs would vary reflecting the predicted concentrations from the last 30 years of the model simulation.
  - **70-year Exposure Duration.** In this case, the analyst is using a lifetime exposure assumption. The exposure scenario may then employ varying ECs reflecting the predicted concentrations from the last 70 years of the model simulation.

Note: When using varying exposure concentrations for the exposure scenario, other variables included in the calculation of ingestion exposure estimates (pollutant intake, mg/kg-day) for the population(s) of interest may also vary. For example, if the exposure scenario includes exposure for cohorts aging from birth - 30 years, other exposure factors (e.g., body weight, consumption rate) will also vary over time.

20.4 Calculating Intake Variable Values

Each intake variable in Equation 20-1 (e.g., consumption rate, body weight) has a range of potential values. Intake variable values for a given pathway may be selected so that the combination of all intake variables results in an estimate for an individual at the "high-end" of potential exposure levels. Alternatively, the intake variables may be selected to represent a "central tendency" individual expected to receive an average exposure. In doing this, the assessor needs to avoid combinations of parameter values that are inconsistent (e.g., low body weight used in combination with high dietary intake rates), and must keep in mind the ultimate objective of being within the distribution of actual expected exposures and doses, and not beyond it. Commonly, both the central tendency and high end intakes are quantified. In some cases, the distribution of intake rates in the population may be described using probabilistic risk assessment methods (discussed in Part VI).

EPA recommends values for intake variables for the U.S. population in the *Exposure Factors Handbook*,[7] the *Child-Specific Exposure Factors Handbook*,[8] and the *Consolidated Human Activity Database*.[9][a] EPA also recently published draft guidance on selecting the appropriate age groups for assessing childhood exposures.[10] Note, however, that there are likely to be differences between recommended default, and regional and site-specific, exposure parameter values. This may be especially true for consumption rate (see below).

For **central tendency** estimates, risk assessors commonly set all of the exposure factors in the Equation 20-1 at central tendency values. If only limited information on the distribution of the exposure or dose factors is available, risk assessors commonly approach the **high-end** estimates by identifying the most sensitive variables and using high-end values for a subset of these variables, leaving others at their central values. As mentioned earlier, the assessor needs to avoid combinations of parameter values that are inconsistent (e.g., low body weight with high dietary intake rates) and must keep in mind the ultimate objective of being within the distribution of actual expected exposures and doses.

Maximizing all variables will in virtually all cases result in an estimate that is above the actual values seen in the population. When the principal parameters of the dose equation (e.g., concentration [appropriately integrated over time], intake rate, and duration) are broken out into sub-components, it may be necessary to use maximum values for more than two of these sub-component parameters, depending on a sensitivity analysis.

For **probabilistic analyses**, values for exposure factors are commonly allowed to vary according to specific assumed distributions of potential values.

Note that the high-end intake estimate is a plausible estimate of intake for those persons at the upper end of the exposure distribution. This descriptor is intended to estimate the exposures that are expected to occur in small but definable high-end segments of the subject population

---

[a]NCEA recently published a new compilation of consumption data from the 1994-1996 CSFII. This data updates CSFII data in the 1997 Exposure Factors Handbook.
See: http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=56610.

(but not higher than the highest person in the population), but may not be appropriate for estimating exposure for the population as a whole.[1]

20.4.1 Consumption Rate

Consumption rate is the amount of contaminated food or medium consumed per event or unit of time (e.g., amount of fish consumed per meal or per day). The consumption rate is multiplied by a fraction of the total dietary intake for this type of food or medium, representing the amount consumed from the study area. The specific fraction applied depends on the analysis.

- For screening-level analyses, it is common to assume that the person obtains 100 percent of the food type from the study area (e.g., farm, water body) being evaluated. This assumption also might be used for a subsistence-type receptor (e.g., a local fish consumer who only eats fish caught from the study area).

- For higher tiers of analyses, it is common to assume that the person obtains some of the food type from the study area (i.e., the contaminated fraction) and some of the food type from other sources (e.g., at the grocery store). This latter fraction generally is assumed to be uncontaminated by the source(s) under assessment. Thus, if a person is assumed to eat ½ pound of fish per day, but only 25 percent is caught within the study area, the assumed consumption of contaminated fish would be 1/8 pound per day.

The following pathway-specific considerations are important for estimating consumption rate.

- **Food Ingestion**. Plants and animals may accumulate COPCs that were deposited onto soil or water. Humans may be exposed to these compounds via the food chain when they consume these plants (and animals that consume these plants) as a food source. Human intake of COPCs is quantified on the basis of the concentration of COPC in the food (Section 20.3) and:
  - The types of foods consumed, which vary with age (e.g., children and adults often eat different things), geographical region, and sociocultural factors (e.g., ethnicity, cultural factors);
  - The amount of food consumed per day, which can vary with age, sex, and geographic region, and also within these categories;
  - The fraction of the diet contaminated by COPCs (which can vary by food type); and
  - The effect of food preparation techniques on concentrations of COPCs in the food itself.

- **Soil Ingestion**. Children and adults may receive direct exposure to COPCs in soil when they consume soil that has adhered to their hands (called incidental soil ingestion). Factors that influence exposure by soil ingestion include concentration of the COPC in soil, the rate of soil ingestion during the time of exposure, and the length of time spent in the vicinity of contaminated soil. Soil ingestion rates in children are based on studies that measure the quantities of nonabsorbable tracer minerals in the feces of young children. Ingestion rates for adults are based on assumptions about exposed surface area and frequency of hand-to-mouth transfer. Indoor dust and outdoor soil may both contribute to the total daily incidental ingestion of soil (indoor dust is partially made up of outdoor soil that has been tracked inside).

In addition, some young children – referred to as "pica" children – may intentionally eat soil. The typical medical and scientific use of the term "pica" refers to the ingestion of nonfood items, such as soil, chalk, and crayons.[10] Such behavior is considered a temporary part of a child's development. For risk assessment purposes, pica is typically defined as "an abnormally high soil ingestion rate" and is believed to be uncommon in the general population. If available information indicates that there are children exhibiting pica behavior in the assessment area, it may be appropriate to include these children as a separate group in the exposure assessment. EPA's *Exposure Factors Handbook* provides quantitative data on soil ingestion rates related to pica.[11]

Inhalation of soil resulting from dust resuspension by wind erosion generally is not a significant pathway of concern for air toxics.[5] However, it may be an issue for locations at which there is little vegetative cover. Methodologies have been developed to assess the exposure to pollutants resuspended by wind erosion for landfills and Superfund sites.[12] The exposure estimate from resuspended soil would depend on moisture content of the soil, fraction of vegetation cover, wind velocity, soil particle size, COPC concentration in the soil, and size of the contaminated area.

Depth of Contaminated Soils: A Key Variable

When exposures to COPCs in soils are modeled for human health risk assessment, an important factor affecting the exposure estimate is the depth of contaminated soils used to calculate soil concentrations. The same deposition rate will result in different soil concentrations depending on how deeply the COPCs are assumed to mix or migrate into the soil. Mixing depth also may affect exposure estimates via specific pathways. For example, in calculations of exposures resulting from uptake through plant roots, the average concentration of COPCs over the depth of the plant root determines plant uptake. However, calculations that assess soil ingestion through hand-to-mouth activity commonly focus on only the top few centimeters of soil.

COPCs deposited onto undisturbed soils generally are assumed to remain in the shallow, upper soil layer. However, COPCs deposited onto soil surfaces may be moved into lower soil profiles by tilling, whether manually in a garden or mechanically in a large field. Other factors such as soil disturbance by domestic animals (e.g., cattle in an enclosure) also may need to be considered. Some chemicals are also highly soluble in water and may be carried deeper into soil along with infiltrating rainwater. The key questions to ask therefore include:

- Are soils tilled, or is it reasonable to assume they are undisturbed?
- If soils are tilled, what mixing depth is reasonable to assume?
- What other factors might affect how deeply COPCs will be moved into soils?

EPA guidance and other references[5][13] provide a more detailed discussion of depth of contaminated soils, along with recommended values.

- **Ingestion of Drinking Water**. In air toxics assessments, assessors only evaluate the ingestion of drinking water when an affected surface water body or collected precipitation (e.g., a cistern) is used as a drinking water source.[b] Important factors affecting the concentration of COPCs in a surface water body include the location of the surface water body or precipitation collection apparatus relative to emissions sources; concentrations of COPCs in and characteristics of the soils (which affects runoff and leachate concentrations); and the size and location of the watershed. For drinking water, the exposure estimate is affected by:
  - The concentration of the COPC in the water;
  - The daily amount of drinkable water ingested; and
  - The fraction of time that the individual spends in the area serviced by that water supply system. (Note that for screening level analyses, 100% of drinking water may be presumed to come from the contaminated source.)

  Note that in estimated exposures associated with drinking water supplies, risk assessors commonly assume that the drinking water undergoes at least a minimum level of treatment to remove solids (i.e., particles in the water which are PB-HAPs or onto which PB-HAPs may be absorbed). Therefore, the risk assessment commonly focuses on the dissolved concentrations of PB-HAPs in drinking water sources.

Groundwater as a Source of Drinking Water

If site-specific circumstances suggest that groundwater may represent a potential concern (e.g., the presence of extremely shallow aquifers used for drinking water purposes or a karst environment in which the local surface water significantly affects the quality of ground water used as a drinking water source), the TRIM.FaTE library includes a groundwater compartment that can be used to assess the groundwater pathway. EPA's *Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities*[14] and *Draft Technical Background Document for Soil Screening Guidance*[15] discuss the methods for evaluating the groundwater pathway.

- **Ingestion of Fish**. Factors that affect human exposure by ingestion of fish from a surface water body include:
  - Sediment and water COPC concentrations;
  - The types of fish and shellfish consumed;
  - The portion of fish eaten (e.g., fillet only, fillet plus skin, whole body);
  - The effect of food preparation techniques on concentrations of COPCs in the fish;
  - Ingestion rates for the various fish and shellfish groups; and
  - The fraction of dietary fish caught in the surface water body or bodies being evaluated. (Note that for screening level analyses, 100 percent of fish/shellfish is presumed to come from the contaminated water body.)

---

[b] Note that ingestion of contaminated groundwater generally is not a significant pathway of concern for air toxics risk assessments because most air toxics that persist and may bioaccumulate tend to get bound up in soil and, therefore, tend not to move readily into groundwater. However, if the groundwater pathway were a concern for a specific study, it would be evaluated in generally the same way as the ingestion of surface water pathway (i.e., as a drinking water source; however, depending on the circumstances, groundwater may or may not be treated to remove particles prior to consumption).

The types of fish consumed will affect exposure because different types of fish and shellfish accumulate COPCs at different rates. For example, fatty fish tend to accumulate lipophilic organic compounds more readily than lean fish. The amount of fish consumed also affects exposure because people who eat large amounts of fish will tend to have higher exposures. Fish consumption rates and the parts of the fish that are consumed can vary greatly, depending on geographic region and social or cultural factors. Also, because all of a person's dietary fish may not originate from the surface water body near the source of the PB-HAP, the fraction of locally caught fish is also a variable for exposure.

20.4.2 Exposure Frequency

The specific exposure frequency will depend on how the exposure analysis is set up. For example, a scenario-based analysis would specify one or more exposure frequencies for each defined scenario. A typical screening-level exposure frequency is 350 days per year; this number is based on the assumption that all people spend a minimum of two weeks at a location other than the exposure scenario location selected for analysis (e.g., on vacation).[(1) (5)] However, many activities vary on a weekly and/or seasonal basis. For example, recreational fishing is more likely to occur on weekends than on weekdays, and most areas in the U.S. have limited fishing and hunting seasons.

20.4.3 Exposure Duration

Exposure duration is the length of time over which exposure occurs (e.g., a lifetime or a particular residence time). As noted in Section 20.3 above, choice of ED will depend on many factors, including the purpose of the assessment or risk management decision, the tier of analysis, and the particular effect(s) of concern. There are no universally established ED values for risk assessments because different EDs may be appropriate in different situations. Some commonly used EDs include:

- Lifetime (70 years) – generally used for screening-level analyses;
- High-end number of years a person resides in a single location (about 30 years);
- Median number of years a person resides in a single location (about 9-10 years); and
- Seven years (ten percent of an assumed lifetime) – sometimes used for noncancer effects.

Although a source may remain in the same location for more than 70 years, and a person may have a lifetime of exposure to emissions from that source, U.S. Bureau of the Census data on population mobility indicate that many Americans do not always remain in the same area for their assumed 70-year lifetime.[(16)] An estimate of the number of years that a person is likely to spend in one area can be derived from information about mobility rate and median time in a residence.

Analysts may use long EDs when conducting simple screening analyses performed to determine if more complex analyses are necessary. The rationale for use of such EDs is that if risks are not of concern when the exposure duration is long, then they would not be of concern given other, shorter, exposure durations. (Typically analysts also make other conservative or "health-protective" assumptions when conducting this type of screening analysis.) Analysts may use specific EDs particular to the legal framework for the assessment. For example, the residual risk section of the Clean Air Act (CAA) references an Agency rulemaking for which one prominent

risk metric considered a 70-year exposure duration (see CAA section 112(f)(2) and 54 *Federal Register* 38044).

The type of risk metric being derived also influences the consideration of exposure duration. For example, when the analyst wants to describe central tendency risk based on a deterministic analysis, s/he typically will use mean or median exposure assumptions to calculate risk.[c] Similarly, when the analyst wants to describe high-end risk based on a deterministic analysis, s/he may use high-end exposure assumptions or a combination of central tendency and high-end exposure assumptions that provide a reasonable estimate of the individual risk for those persons at the upper end of the risk distribution. As explained in EPA's *Policy on Risk Characterization*:[17] "Conceptually, high-end exposure means exposure above about the 90th percentile of the population distribution, but not higher than the individual in the population who has the highest exposure."[d] When the analyst wants to conduct a probabilistic analysis of risk, s/he typically will use or develop a distribution of exposure durations from the available data (e.g., see EPA's *Exposure Factors Handbook, Part III*; Tables 15-164, 15-166, 15-167, and 15-168).[10]

The areal extent of the impacted area(s) may also be a consideration. If a source of concern occurs in the majority of communities, then it is possible that individuals may be exposed to the source for a longer period of time than one might predict using standard estimates of exposure duration. In this case, the analyst might assume that even though an individual changes residence, the individual still would be exposed to the source of concern, and thus the individual's exposure duration would be greater than typically anticipated. Such an analysis must consider whether the concentration of the pollutant at the multiple locations of exposure would be equivalent. Because location-specific parameters such as meteorological conditions, distance from the source, and the presence of certain pathways of exposure (e.g., surface water, home-grown produce) may vary considerably by geographic area, the analyst likely will have to estimate exposure concentrations for each geographic location or community of interest. Similarly, if a single source impacts a large geographic area, then it is possible that national estimates of population mobility will not adequately capture an individual's potential duration of exposure. That is, an individual may move from one point of exposure associated with a particular source to another point of exposure associated with that same source. For example, data indicate 29 percent of home buyers move less than five miles to a new home (Table 15-171 in EPA's *Exposure Factors Handbook, Part III*[10]). Similar to the caution expressed above, the concentrations of pollutants within an area impacted by a single source may vary considerably. The analysis should reasonably account for such situations.

---

[c]The central tendency estimate of adult exposure duration commonly used in risk assessments is 9 years (Section 15.4.3 and Table 15-174 in EPA's *Exposure Factors Handbook, Part III*).[10] This estimate is a median value based on national residential occupancy data for the general population. This estimate may not be appropriate in certain situations, such as when population-specific data exist or when the analyst is evaluating a specific sub-population that is expected to differ from the general population (e.g., farm families).

[d]As described in Section 20.4, estimation of high-end exposure will sometimes involve setting exposure duration at its high-end value. The high-end estimate of adult exposure duration typically used in risk assessments is 30 years (Section 15.4.3 and Table 15-174 in EPA's *Exposure Factors Handbook, Part III*),[10] although this may vary for specific sub-populations.

The persistence of the source-associated contamination may also be an important consideration in the exposure duration for ingestion pathway exposure assessment. For example, the analyst should not automatically assume that the exposure duration can be no greater than the operating life of the source. Persistent pollutants may remain in the environment (e.g., soils and sediments) for years after the primary source is discontinued. Nevertheless, in certain cases, once the source of exposure stops, the pollutant concentrations in the affected media may diminish. Particularly in more refined assessments, the exposure concentration may reflect any expected variations in media or food concentrations over time.

When evaluating the risk of noncancer health effects from ingestion exposures (i.e., calculating hazard quotients for ingestion exposures), we do not average pollutant dose over the lifetime of an individual as we do when calculating carcinogenic risk. Rather, when calculating hazard quotients for ingestion exposures, we average the dose over an averaging time equivalent to only the period of exposure (i.e., we calculate an average daily dose rather than a lifetime average daily dose). Consequently, the values for exposure duration and averaging time are the same, and mathematically cancel each other out. Nevertheless, when calculating average daily dose, the analyst must still consider exposure duration when selecting and computing food and media intakes for use in the dose equation. EPA typically considers exposures of seven years or greater as chronic exposures. Food and media intakes that represent time-weighted averages over a seven-year period are reasonable for evaluating chronic non-cancer health effects. **Durations as short as one year are also commonly used, particularly in screening assessments, and for childhood evaluations where intake on a per body weight basis may rapidly change from year to year**.

20.4.4 Body Weight

The choice of body weight for use in the exposure assessment depends on the definition of the population group at potential risk. Because children have lower body weights, typical ingestion exposures per unit of body weight, such as for soil, milk, and fruits, tend to be higher for children. If a lifetime exposure duration (or an exposure duration over the childhood and adult years) is being evaluated, it needs to be based on differing values for the different age groups. If a less than a lifetime exposure estimate is being evaluated, it is important to include the children's age group in the specific scenarios or cohorts used. EPA's *Exposure Factors Handbook*[6] and *Child-Specific Exposure Factors Handbook*[7] provide age-specific values for body weight and consumption rate per unit body weight.

20.5 Calculating Averaging Time Value

When evaluating exposure for the purposes of assessing hazard (vs. predicting cancer risk), **intakes** are calculated by averaging intakes over the period of exposure (i.e., subchronic or chronic durations) and result in average daily doses or ADDs for the duration of interest. For evaluation of cancer risks, potential dose is calculated as the average daily dose over a lifetime (i.e., chronic daily intakes, also called lifetime average daily doses or LADDs). The approach for carcinogens is based on the premise that risk is proportional to total lifetime dose (i.e., a high dose received over a short period of time is equivalent to a corresponding low dose spread over a lifetime).[18] The basis for this approach becomes less strong as the exposures in question become more intense but less frequent, especially when there is evidence that the agent has shown age-related variations in carcinogenic potency, or a nonlinear dose-response relationship.

In some cases, therefore, it may be necessary to consult a toxicologist to assess the level of uncertainty associated with the exposure assessment for carcinogens.

Note that, even when the exposure of interest is a full lifetime, chronic hazards are generally calculated separately for chronic exposures to age groups that differ substantially with regard to pertinent exposure factors (e.g., ingestion rate or body weight) and are not combined (i.e., usually the oral route hazards calculated for children are not added to the hazards posed to adults to represent a "lifetime hazard"). Rather, both hazard quotients/indices are presented as chronic hazard metrics relevant to the two groups. When assessing carcinogenic risks for a lifetime exposure, on the other hand, cancer risk estimates are usually added across different age groups, since the risk received over discrete periods of time (e.g., as a child, as a young adult, as an older adult) are each considered to be fractions of the risk associated with a full lifetime of exposure. Note that in calculating LADDs, it is essential to account for differences in the values of different intake variables (e.g., body weight, consumption rate) at different ages.

20.6 Combining Exposure Estimates Across Pathways

A given population may receive exposure to an individual chemical from several different exposure pathways. For example, individuals may receive exposure via inhalation of the chemical in the air and via ingestion of surface water and fish that have become contaminated through deposition. The specific exposure scenarios or cohorts defined for the analysis may include more than one pathway. The corresponding intake variables used in the analysis may need to account for the number of pathways over which exposure will be combined. For example, to develop a high-end estimate for a scenario that includes inhalation, ingestion of soil, and ingestion of fish, it may be necessary to combine high-end exposure assumptions for all pathways. In other cases, it may be more appropriate combine high-end exposure assumptions for particular pathways with more central-tendency assumptions for others. Otherwise, the estimate may represent an extreme situation in which the simulated behavior is assumed to result in high exposures via all pathways.

Two steps are required to determine whether intake estimates should be combined for a single scenario:

- **Identify reasonable exposure pathway combinations**. Identify exposure pathways that have the potential to expose the *same* individual, cohort, or subpopulation at the key exposure areas evaluated in the exposure assessment, making sure to consider *areas of highest exposure* for each pathway. For each pathway, the intake estimates have been developed for a particular exposure area and time period; they do not necessarily apply to other locations or time periods. Hence, if two pathways do not affect the same individual, cohort, or subpopulation, neither pathway's exposure estimate affects the other, and exposures should not be combined.

- **Examine whether it is likely that the *same* individuals would consistently face a reasonable central tendency or high-end exposure by more than one pathway**. Once reasonable exposure pathway combinations have been identified, it is necessary to examine whether it is likely that the *same individuals* would *consistently* face central tendency or high-end exposure conditions. As noted in Section 20.4 above, the exposure estimate for each exposure pathway includes many conservative estimates. Also, some of the exposure

parameters are not completely predictable in space and/or time (e.g., the maximum downwind concentration may shift compass direction). For real-world situations in which contaminant concentrations vary over time and space, the same individual or cohort may or may not experience central-tendency or high-end exposure conditions for more than one pathway over the same period of time. Thus, it is important to clearly explain why the key assumptions chosen for more than one pathway for an individual, subpopulation, or cohort are set at central tendency and/or high-end exposure estimates. (Note that an important goal in the analysis of high-end receptors is to identify exposures that are in the high-end of the range - usually higher than the 90th percentile exposure - but not higher than the highest exposure in the population.)

20.7 Exposure Models

Exposure models have been developed that automate the calculation of chemical intake. They may simply calculate exposure for a set of individual scenarios, or they may draw upon activity pattern and/or dietary survey databases to characterize cohort exposure within a population. Three exposure models are described below.

California Total Exposure Model for Hazardous Waste Sites (CalTOX)

As described previously in Part II, Chapter 9, the California Environmental Protection Agency funded the development of the CalTOX program.[19] CalTOX has been developed as a set of spreadsheet models and spreadsheet data sets to assist in assessing human exposures and defining soil clean-up levels at uncontrolled hazardous wastes sites. CalTOX addresses contaminated soils and the contamination of adjacent air, surface water, sediments, and ground water. The modeling components of CalTOX include exposure scenario models. The exposure models encompass twenty-three exposure pathways. The exposure assessment process consists of relating pollutant concentrations in the multimedia model compartments to pollutant concentrations in the media with which a human population has contact (e.g., personal air, tap water, foods, household dusts, soils). The temporal resolution is either daily for inhalation and dermal exposure or annual for ingestion. The aggregation period is variable, depending on the duration of residence at a single location. The spatial resolution and modeling domain are user-specified, but generally encompass some vicinity around the waste site of interest. Activity data, such as inhalation, ingestion, and dermal contact rates, are derived from EPA's *Exposure Factors Handbook*.[6]

TRIM.Expo

As discussed in Chapter 18, TRIM.Expo is the exposure component of the TRIM modeling system. The ingestion component of TRIM.Expo ($\text{TRIM.Expo}_{\text{Ingestion}}$) is designed to take input values from TRIM.FaTE, but may also be operated independently with inputs from measurement studies or alternative models. $\text{TRIM.Expo}_{\text{Ingestion}}$ will employ a scenario-based approach, based on that used in the 3MRA modeling system, in its initial version. Information about the ingestion component of TRIM.Expo is available on EPA's Fate, Exposure and Risk Analysis (FERA) web site: http://www.epa.gov/ttn/fera.

Stochastic Human Exposure and Dose Simulation Model (SHEDS)

The Stochastic Human Exposure and Dose Simulation (SHEDS) Model[20] is a probabilistic, physically-based model that simulates aggregate exposure and dose for population cohorts and multimedia pollutants of interest. It is being developed by EPA's National Exposure Research Laboratory (http://www.epa.gov/nerlpage/). At present the model is applied to assess children's exposures to pesticides (SHEDS-Pesticides) and population exposures to particulate matter (SHEDS-PM).

SHEDS-Pesticides focuses on children's aggregate population exposure to pesticides. Activity data are selected from daily sequential time/location/activity diaries from surveys contained in EPA's Consolidated Human Activity Database (CHAD).[8] For each individual, SHEDS-Pesticides constructs daily exposure and dose time profiles for the inhalation, dietary and non-dietary ingestion, and dermal contact exposure routes, and then aggregates the dose profiles across routes. A pharmacokinetic component has been incorporated to predict pollutant or metabolite concentrations in the blood compartment or eliminated urine. Exposure and dose metrics of interest (e.g., peak, time-averaged, time-integrated) are extracted from the individual's profiles. Two-stage Monte-Carlo sampling is applied to predict the range and distribution of aggregate doses within the specified population and identify the uncertainties associated with percentiles of interest.

SHEDS-Pesticides is currently being refined to characterize both aggregate and cumulative dose associated with human exposure (i.e., for both adults and children) to a variety of environmental pollutants in addition to pesticides. SHEDS-Pesticides will eventually be expanded to include source-to-concentration (i.e., fate and transport) models and more complete exposure-to-dose models (i.e., pharmacokinetic or dosimetric models).

SHEDS-PM estimates the population distribution of particulate matter (PM) exposure by sampling from distributions of ambient PM concentrations, distributions of emission strengths for indoor sources of PM (e.g., cigarette smoking and cooking), and distributions of mass-balance parameters (e.g., air exchange rate, penetration rate, deposition rate). A steady-state mass balance equation is used to calculate PM concentrations for the residential and other microenvironments. Additional model inputs include demographic and human activity pattern data from the National Human Activity Pattern Survey (NHAPS). Output from the SHEDS-PM model includes distributions of PM exposures in various microenvironments (e.g., in the home, in vehicles, outdoors) and the relative contributions of these various microenvironments to the total exposure.

References

1. U.S. Environmental Protection Agency. 1992. *Guidelines for Exposure Assessment*. 1992. National Center for Environmental Assessment, Risk Assessment Forum. Washington, D.C. EPA 600Z-92/001. 170 pp. Available at: http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=15263.

   U.S. Environmental Protection Agency. 2001. *Risk Assessment Guidance for Superfund: Volume I. Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Assessment, Interim - Review Draft for Public Comment).* Office of Emergency and Remedial Response. Washington, D.C., EPA/540/R/99/005, available at: http://www.epa.gov/superfund/programs/risk/ragse/index.htm.

2. U.S. Environmental Protection Agency. 1999. *National Recommended Water Quality Criteria - Correction*. Office of Water. Washington, D.C. EPA 822-Z-99-001. April.

3. U.S. Environmental Protection Agency. 1996. *Soil Screening Guidance: Technical Background Document.* Office of Superfund Remediation and Technology Innovation, formerly the Office of Office of Emergency & Remedial Response. EPA/540/R-95/128. Available at: http://www.epa.gov/superfund/resources/soil/introtbd.htm.

4. U.S. Environmental Protection Agency. 1989. *Risk Assessment Guidance for Superfund: Volume I. Human Health Evaluation Manual (Part A).* Office of Emergency and Remedial Response. Washington, D.C., EPA/541/1-89/002, available at: http://www.epa.gov/superfund/programs/risk/ragsa/index.htm

5. U.S. Environmental Protection Agency. 1990. *Interim Final Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions.* Environmental Criteria and Assessment Office, Office of Research and Development, January, 1990. EPA-600-90-003.

   U.S. Environmental Protection Agency. 1993. *Review Draft Addendum to the Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions*. OHEA, Office of Research and Development, November 1993. EPA/600-AP-93-003.

   U.S. Environmental Protection Agency. 1998. *Methodology for Assessing Health Risks Associated with Multiple Pathways of Exposure to Combustor Emissions*. Update to EPA/600/6-90/003 *Methodology for Assessing Health Risks Associated With Indirect Exposure to Combustor Emissions*. National Center for Environmental Assessment. EPA-600/R-98-137; available at: http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=55525.

6. U.S. Environmental Protection Agency. 1997. *Exposure Factors Handbook, Volume III. Activity Factors.* Office of Research and Development, Washington, D.C., August 1997. EPA/600/P-95/0002Fc, available at: http://www.epa.gov/ordntrnt/ORD/WebPubs/exposure/.

7. U.S. Environmental Protection Agency. 1989. *Exposure Factors Handbook.* Office of Research and Development, National Center for Environmental Assessment, Washington, D.C., May 1989. EPA/600/8-89/043, available at: http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=12464.

8. U.S. Environmental Protection Agency. 2002. *Child-Specific Exposure Factors Handbook (Interim Report).* Office of Research and Development, National Center for Environmental Assessment, Washington, DC, September 2002. EPA-600-P-00-002B.

9. The Consolidated Human Activity Database (CHAD) is available at: http://www.epa.gov/chadnet1/.

10. United States Environmental Protection Agency. 2003. *Guidance on Selecting the Appropriate Age Groups for Assessing Childhood Exposures to Environmental Contaminants (External Review Draft).* Risk Assessment Forum, Washington, D.C. September, 2003. EPA/630/P-03/003A; available at www.epa.gov/ncea.

11. U.S. Environmental Protection Agency. 1997. *Exposure Factors Handbook, Volume II. Food Ingestion Factors*. Office of Research and Development, Washington, D.C., August 1997. EPA/600/P-95/0002Fb, available at: http://www.epa.gov/ordntrnt/ORD/WebPubs/exposure/.

12. U.S. Environmental Protection Agency. 1985. *Rapid Assessment of Exposure to Particulate Emissions from Surface Contamination Sites*. Office of Health and Environmental Assessment (OHEA). Washington, D.C. EPA/600/8-85/002. NTIS PB 85-192219.

    U.S. Environmental Protection Agency. 1988. *Superfund Exposure Assessment Manual*. Office of Emergency and Remedial Response. Washington, D.C. EPA/540/1-88/001. NTIS PB 89-167985.

    U.S. Environmental Protection Agency. 1994. *Draft Technical Background Document for Soil Screening Guidance*. EPA/540/R-94/106. OSWER. Washington, D.C. December.

13. U.S. Environmental Protection Agency. 1990. *Interim Final Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions*. Environmental Criteria and Assessment Office, January 1990. ORD. EPA-600-90-003.

    U.S. Environmental Protection Agency. 1992. *Estimating Exposures to Dioxin-Like Compounds. Draft Report*. OHEA, Washington, D.C. EPA/600/6-88/005B, August 1992.

    Brzusy, L.P. and Hites, R.A. 1995. Estimating the atmospheric deposition of polychlorinated dibenzo-p-dioxins and dibenzofurans from soils. *Environ. Sci. Technol.* 29: 2090-2098.

14. U.S. Environmental Protection Agency. 1998. *Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities, Peer Review Draft*. Office of Solid Waste and Emergency Response, Washington, D.C., July 1998. EOA/30/D-98/001A. Available at: http://www.epa.gov/epaoswer/hazwaste/combust/risk.htm

15. U.S. Environmental Protection Agency. 1994. *Draft Technical Background Document for Soil Screening Guidance*. Office of Solid Waste and Emergency Response, Washington, D.C., December 1994. EPA/540/R-94/106.

16. U.S. Bureau of the Census. 1986. *Geographical Mobility: March 1983 to March 1984*. Current Population Reports. Series P-20. Number 407. U.S. Government Printing Office. Washington, D.C.

17. U.S. Environmental Protection Agency. 1995. *Policy for Risk Characterization* ("Browner Memorandum"). Science Policy Council, Washington, DC. Available at: http://64.2.134.196/committees/aqph/rcpolicy.pdf

18. U.S. Environmental Protection Agency. 1999. *Guidelines for Carcinogen Risk Assessment (INTERIM)*, Risk Assessment Forum, Washington, DC NCEA-F-0644.

19. McKone, T.E. 1993a. *CalTOX, a Multimedia Total-Exposure Model for Hazardous wastes Sites Part I: Executive Summary*. UCRL-CR-11456, Pt. I. 1993b. *CalTOX, a Multimedia Total-Exposure Model for Hazardous Wastes Sites Part II: the Dynamic Multimedia Transport and Transformation Model*. UCRL-CR-111456, Pt. II. 1993c. *CalTOX, a Multimedia Total-Exposure Model for Hazardous Wastes Sites Part III: The Multiple-Pathway Exposure Model*. UCRL-CR-111456, Pt. III. Livermore, CA: Lawrence Livermore National Laboratory.

20. Zartarian V.G., Ozkaynak H., Burke J.M., Zufall M.J., Rigas M.L., and Furtaw Jr. E.J. 2000. A modeling framework for estimating children's residential exposure and dose to chlorpyrifos via dermal residue contact and non-dietary ingestion. *Environmental Health Perspectives* 108:505-514.

Chapter 21 Ingestion Toxicity Assessment

Table of Contents

21.1 Introduction 1

21.2 Hazard Identification 2

21.3 Predictive Approach for Cancer Effects 2
- 21.3.1 Determining the Point of Departure (POD) 2
- 21.3.2 Deriving the Human Equivalent Dose 2
- 21.3.3 Extrapolating from POD to Derive the Oral Cancer Slope Factor 3

21.4 Dose-response Assessment for Derivation of a Reference Dose 3

21.5 Sources of Human Health Reference Values for Risk Assessment 4

References 5

21.1 Introduction

As described previously in Chapter 12, the purpose of the toxicity assessment is to weigh available evidence regarding the potential for toxicity in exposed individuals (**hazard identification**) and to quantify the toxicity by deriving an appropriate dose-response value (**dose-response assessment**). Toxicity assessment is the second part of the general risk equation. The toxicity assessment is accomplished in two steps: **hazard identification** and **dose-response assessment**. Although the toxicity assessment is an integral and important part of the overall air toxics risk assessment, this is usually accomplished prior to the risk assessment. EPA has completed the toxicity assessment for all HAPs and has made available the resulting toxicity information and dose-response values, which have undergone extensive peer review (see Appendix C).[a]

Risk = *f* (metric of exposure, measure of toxicity)
Toxicity Assessment is a Two-Step Process:

1. **Hazard Identification** –What types of effects does the chemical cause? Under what circumstances?
2. **Dose-response Assessment** – How potent is the chemical as a carcinogen and/or for noncancer effects?

This chapter focuses on toxicity assessment for the ingestion (oral) pathway. Dermal toxicity assessment is described in detail in several EPA guidance documents.[1] The ingestion pathway uses the same general types of studies, hazard and dose-response information, and dose-response methods to assess toxicity as those used for the inhalation pathway (see Chapter 12). The discussion in this chapter focuses on the unique features of toxicity assessment for the oral pathway.

Ingestion Dose-Response Values[a]

Oral Cancer Slope Factor (CSF): An upper bound, approximating a 95 percent confidence limit, on the increased cancer risk from a lifetime exposure to an agent. For ingestion, this estimate is usually expressed in units of amount of risk per amount of intake and is written as risk per mg/kg-day or simply $(mg/kg\text{-}d)^{-1}$.

Reference Dose (RfD): An estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive sub-populations) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Generally used in EPA's noncancer health assessments. RfDs are usually given in units of intake per day on a body weight basis (written as mg/kg-d).

[a]The phrase "dose-response" is used generally here and elsewhere in the document. EPA's values for ingestion, however, are related to oral intake rather than dose. Consideration of the relationship between exposure concentration, dose, and dosimetry (what happens to a chemical in the body once it is ingested) may be considered, depending on data availability in the derivation of these values.

[a]See http://www.epa.gov/ttn/atw/toxsource/summary.html for an up-to-date list of dose-response values.

21.2 Hazard Identification

The hazard identification process for the ingestion pathway is identical to that for the inhalation pathway, although the specific toxic effects of concern and details of the toxicity studies are derived from feeding a chemical to animals (either in food or drinking water) rather than on having the animals inhale the chemical. As with inhalation, the hazard identification step includes consideration of various types of studies (e.g., feeding, in vitro, etc.) and the resulting weight of evidence with regard to potential for carcinogenicity and identification of critical effects. See Part II, Chapter 12, for information on the hazard identification step.

21.3 Predictive Approach for Cancer Effects

The approach to dose-response assessment for cancer effects is identical to that for the inhalation pathway discussed in Chapter 12, including:

- Determination of the **point of departure** (**POD**);
- **Duration adjustment** of the POD to a continuous exposure;
- Extrapolation of an animal study POD into its corresponding **Human Equivalent Dose** (**POD_{HED}**); and
- **Low-dose extrapolation** from the POD_{HED} to lower doses for the purposes of deriving the oral cancer risk estimate.

As with inhalation, the first three steps are also performed in the derivation of reference values for ingestion, such as the oral RfD. In addition to the steps shown above, the derivation of RfDs are followed by the application of uncertainty factors (see Section 21.4). Additionally, the use of tools such as pharmacokinetic modeling, which go beyond these default approaches, may facilitate the accomplishment of several of these steps.

21.3.1 Determining the Point of Departure (POD)

The process for determining the POD for ingestion exposures is identical to that for inhalation exposures. The POD may be the no-observed-adverse-effect level (NOAEL) or lowest-observed-adverse-effect level (LOAEL), or it may be a **benchmark dose** (**BMD**) for noncancer effects.[b]

21.3.2 Deriving the Human Equivalent Dose

The optimal approach for extrapolating from an animal study to a human dose-response relationship is to use Physiologically Based Pharmacokinetic (PBPK)[c] modeling. When such a model us used, the duration adjustment step is incorporated into that model. Otherwise, any duration adjustment, if necessary (e.g., when the exposure is not via daily feed), would be accomplished by deriving an average daily dose for the exposure period (e.g., two years in an animal cancer bioassay).

---

[b]Note that the corresponding value for inhalation exposures is the benchmark concentration (BMC).

[c]A model that estimates the dose to a target tissue or organ by taking into account the rate of absorption into the body, distribution among target organs and tissues, metabolism, and excretion.

For purposes of cancer assessment, an animal to human body weight-based scaling factor is applied to the oral study POD (duration-adjusted if applicable) to extrapolate to a human equivalent oral exposure.[2] The default scaling factor is based on the body mass raised to the 3/4 power of the test animals relative to humans. This step stems from the consideration of various studies of the species differences in toxicity of certain compounds, including data collected on chemotherapeutic agents.[3] These data served as the principal basis for the use of a body surface area or metabolic rate scaling as the default method in cancer risk assessments. Empirically, the best estimate of surface area scaling is $BW^{2/3}$ and for metabolic rate scaling is $BW^{3/4}$.[4] These findings reflect general expectations of more rapid distribution, clearance, and metabolism by smaller animals.

In the case of the RfD, a scaling factor is not currently applied. Instead, the interspecies uncertainty factor is intended to account for potential differences in sensitivity of humans compared to the test animal, including this consideration.[d]

A PBPK model can accommodate adjustments for metabolic rate as well as other species-related dosimetric variables such as liver perfusion rates. The model therefore provides a more accurate estimate of steady-state target site concentrations than use of default methods. EPA's preferred approach for calculating a HED for oral exposures is to use a chemical-specific PBPK model parameterized for the animal species and body regions (e.g., of the gastrointestinal tract) involved in the toxicity.

21.3.3 Extrapolating from POD to Derive the Oral Cancer Slope Factor

As with inhalation, extrapolation from the POD_{HED} to lower doses is usually necessary and, in the absence of a data set rich enough to support a biologically based model (e.g., a PBPK model), is conducted using linear extrapolation or a nonlinear extrapolation using a Reference Dose approach.

The **Cancer Slope Factor** (**CSF**) for oral exposures is derived in a similar way as the unit risk estimate for inhalation (URE) (see Chapter 12). The CSF is derived using the upper bound estimate of risk. In other words, the true risk to humans, while not identifiable, is not likely to exceed the upper-bound estimate (the CSF). The CSF is presented as the risk of cancer per mg of intake of the substance per kg body weight per day ($[mg/kg\text{-}day]^{(-1)}$).

21.4 Dose-response Assessment for Derivation of a Reference Dose

The oral **reference dose** is expressed as a chronic dietary intake level (in units of mg of the substance per kilogram body weight per day, or mg/kg-day) for the human population (including sensitive sub-populations) that is likely to be without an appreciable risk of deleterious effects during a lifetime. In other words, exposures at or below the RfD will probably not cause adverse health effects, even to sensitive sub-populations. While the RfD is routinely employed for

---

[d] At the time of publication, an Agency activity is underway to "harmonize" the cancer assessment and RfD development methods with regard to the method employed for interspecies scaling, which may result in the use of body weight scaling in the development of the RfD.

noncancer effects, it may be inclusive of cancer for those pollutants for which a nonlinear (e.g., threshold) mode of action has been demonstrated consistent with the Cancer Guidelines.

As with the derivation of an inhalation reference concentration, the reference dose is derived by dividing the POD by one or more **uncertainty factors** (**UFs**). EPA includes with each RfD a statement of high, medium, or low confidence based on the completeness of the database for that substance. High confidence RfDs are considered less likely to change substantially with the collection of additional information, while low confidence RfDs may be especially vulnerable to change.[5]

The UFs are applied to account for recognized uncertainties in the extrapolations from the experimental data conditions to an estimate appropriate to the assumed human scenario. As with the derivation of RfCs, a UF of 10, 3, or 1 is applied for each of the following extrapolations:

- Animal to human;
- Human to exposed sensitive human populations;
- Subchronic to chronic;
- LOAEL to NOAEL; and
- Incomplete to complete database.

The UFs are generally an order of magnitude (10), although consideration of available information on the chemical may result in the use of reduced UFs for RfDs (3 or 1). It is noted that as there is currently no default dosimetric adjustment for the oral route. The uncertainty factor for extrapolation from animal to human data is usually the full 10, as compared to the reduced factor of 3, routinely used for RfCs which employs an interspecies dosimetric adjustment. Additional discussion on the application of uncertainty factors is provided in Section 12.4.3.

21.5 Sources of Human Health Reference Values for Risk Assessment

Appendix C provides a current listing of chronic oral dose-response values (i.e., RfDs and CSFs) for HAPs. Chapter 12 describes additional sources of human health reference values for risk assessment for the ingestion route.

References

1. U.S. Environmental Protection Agency. 2001. *Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) Interim Review Draft - For Public Comment,* Office of Emergency and Remedial Response, Washington, D.C. EPA/540/R/99/005, available at: http://www.epa.gov/superfund/programs/risk/ragse/index.htm.

   U.S. Environmental Protection Agency. 1992. Dermal Exposure Assessment: Principles and Applications. Office of Health and Environmental Assessment. EPA/600/6-88/005C.

2. U.S. Environmental Protection Agency. 1999. *Guidelines for Carcinogen Risk Assessment. Review Draft.* Risk Assessment Forum, Washington, DC. NCEA-F-0644.

   U.S. Environmental Protection Agency. 1986. *Guidelines for Carcinogen Risk Assessment. Federal Register* 51(185):33992-43003.

   U.S. Environmental Protection Agency. 2003. *Draft Final Guidelines for Carcinogen Risk Assessment (External Review Draft),* Risk Assessment Forum, Washington, DC, NCEA-F-0644A.

   These documents are available at http://cfpub.epa.gov/ncea/raf/rafguid.htm.

3. Freireich, E.J., Gehan, E.A., Rall, D.P., et al. 1966. "Quantitative comparison of toxicity anticancer agents in mouse, rat, hamster, dog, monkey, and man." Cancer Chemother Report 50:210:244.

4. U.S. Environmental Protection Agency. 1992. *Request for Comments on Draft Report of Cross-species Scaling Factor for Cancer Risk Assessment.* Federal Register 57:24152.

5. U.S. Environmental Protection Agency. 2002. *A Review of the Reference Dose and Reference Concentration Process.* Risk Assessment Forum, Washington, DC. EPA/630/P-02/002F. Available at: http://cfpub.epa.gov/ncea/raf/recordisplay.cfm?deid=55365.

Chapter 22 Multipathway Risk Characterization

Table of Contents

22.1 Introduction 1

22.2 Cancer Risk Estimates 2
- 22.2.1 Characterizing Individual Pollutant Ingestion Risk - Scenario Approach 2
- 22.2.2 Characterizing Risk from Exposure to Multiple Pollutants - Scenario Approach 3
- 22.2.3 Combining Risk Estimates across Multiple Ingestion Pathways - Scenario Approach . . 4
- 22.2.4 Evaluating Risk Estimates from Inhalation and Ingestion Exposures 4

22.3 Noncancer Hazard 4
- 22.3.1 Characterizing Individual Pollutant Hazard - Scenario Approach 5
- 22.3.2 Multiple Pollutant Hazard 6
- 22.3.3 Evaluating Hazard Estimates From Inhalation and Ingestion Exposures 6

22.4 Interpretation and Presentation of Risks/Hazards 7

References 8

22.1 Introduction

The last component of risk assessment, Risk Characterization, integrates the information from the exposure assessment (Chapter 20) and toxicity assessment (Chapter 21), using a combination of qualitative information, quantitative information, and a discussion of uncertainty.[1] Risk assessors should present the risk characterization and its components so that they are transparent, clear, and consistent with EPA guidance and policy, and thus components should support the conclusion that the analysis is reasonably conservative enough for its intended purpose. The risk summary and risk conclusions must be complete, informative, and useful for decision-makers. Major uncertainties associated with determining the nature and extent of the risk should be identified and discussed.

Risk characterization for the multipathway risk assessment is performed using the same approach as described for the inhalation pathway (Chapter 13), except that risks for both inhalation and ingestion are considered. As for inhalation-only analyses, most multipathway risk assessments for air toxics will focus on estimating individual risk and hazard. This chapter focuses on the unique features of risk characterization for multipathway analyses. ***This chapter also assumes that the inhalation risk characterization has been completed, as described in Chapter 13.***

Steps in a Multipathway Risk Characterization

1. Organize outputs of the ingestion exposure and toxicity assessments.
2. Derive cancer risk estimates and noncancer hazard quotients for each pollutant in each pathway.
3. Derive multiple pollutant cancer risk estimates and noncancer hazard indices for each pathway.
4. In consideration of target organ, develop target organ specific hazard indices, if appropriate.
5. As appropriate, combine information on cancer risk and noncancer hazard from the ingestion analysis with appropriate risk information from the inhalation analysis to derive a total estimate of cancer risk and noncancer hazard.
6. Identify key features and assumptions of exposure and toxicity assessments.
7. Assess and characterize key uncertainties associated with the assessment.
8. Consider additional relevant information (e.g., related studies).

The risk characterization should be written consistent with EPA guidance and policy, including a risk summary and risk conclusions that are complete, informative, and useful for decision-makers, and which clearly identify and discuss the major uncertainties associated with determining the nature and extent of the risk.

The general process for characterizing cancer risks and noncancer hazards for multipathway analyses can be thought of as developing information to fill in a matrix similar to that shown in Exhibit 22-1 (which presents cancer risks for a group of chemicals; a similar matrix can be developed to present noncancer hazards [see Exhibit 22-2]). A table like this would be developed for each of the types of receptors being evaluated in the study area (e.g., adult farmer – high-end exposure; adult farmer – central tendency exposures; child resident – high-end exposure). This type of presentation format shows the total risk by chemical, pathway, and across all pathways. In addition, this format allows one to quickly identify both the individual chemicals and pathways that contribute most to the total risk estimate. The following sections describe how to develop the numbers to fill in such a table for both multipathway cancer risk estimates (Section 22.2) and multipathway noncancer hazards (Section 22.3). The focus of this

chapter is on developing risks and hazards for the ingestion pathways; procedures for developing inhalation risk estimates have previously been provided in Chapter 13.

Exhibit 22-1. Example Matrix for Estimating Excess Cancer Risks for Multiple Chemical Exposure through Multiple Ingestion Pathways for a Particular Exposure Scenario

	Pathway 1 (Vegetable Ingestion Risk Estimate)[a]	Pathway 2 (Fish Ingestion Risk Estimate)[a]	Pathway 3 (Egg Ingestion Risk Estimate)[a]	Pathway 4 (Beef Ingestion Risk Estimate)[a]	Aggregate Chemical Ingestion Risk Estimate [a]
Chemical 1	1×10^{-6}	3×10^{-4}	9×10^{-8}	8×10^{-5}	4×10^{-4}
Chemical 2	4×10^{-7}	4×10^{-6}	4×10^{-8}	4×10^{-7}	5×10^{-6}
Chemical 3	4×10^{-9}	7×10^{-7}	3×10^{-8}	9×10^{-9}	8×10^{-7}
Chemical 4	9×10^{-7}	1×10^{-6}	6×10^{-7}	6×10^{-7}	3×10^{-6}
Cumulative Ingestion Pathway Risk Estimate [a]	3×10^{-6}	3×10^{-4}	7×10^{-7}	8×10^{-5}	4×10^{-4}

[a] Standard rules for rounding apply which will commonly lead to an answer of one significant figure in both risk and hazard estimates. For presentation purposes, hazard quotients (and hazard indices) and cancer risk estimates are usually reported as one significant figure.

22.2 Cancer Risk Estimates

As discussed in detail in Chapter 13, estimated individual cancer risk is expressed as the probability that a person will develop cancer as a result of the estimated exposure over a lifetime. This predicted risk is the **incremental risk** of cancer from the exposure being analyzed, which are in addition to other risks due to any other factors (e.g., smoking). Due to default assumptions in their derivation, cancer slope factors (CSFs) are generally considered to be "plausible upper-bound" estimates, regardless of whether they are based on statistical upper bounds or best fits. As noted in Chapter 13, risks may be estimated for both the central tendency (average exposure) case and for the high-end (exposure that is expected to occur in the upper range of the distribution) case, or probabilistic techniques can be used to develop a distribution of estimated risks.

22.2.1 Characterizing Individual Pollutant Ingestion Risk - Scenario Approach

The first step in characterizing individual pollutant risk for an exposure scenario (e.g., a recreational fisher) is to quantify risk for each ingestion exposure pathway being evaluated. In this step, cancer risks for individual pollutants are estimated by multiplying the estimate of the lifetime average daily dose (LADD) for each ingestion exposure pathway by the appropriate CSF to estimate the potential incremental cancer risk:

$$\text{Risk} = \text{LADD} \times \text{CSF} \qquad \text{(Equation 22-1)}$$

where:

Risk	=	Individual cancer risk (expressed as an upper-bound risk of contracting cancer over a lifetime) for each pollutant via the ingestion pathway being evaluated (unitless);
LADD	=	Lifetime Average Daily Dose for the pollutant via the ingestion pathway being evaluated (mg/kg-d); and
CSF	=	Cancer Slope Factor for the pollutant via the ingestion pathway being evaluated [$(mg/kg\text{-}d)^{-1}$]

Estimates of cancer risk are usually expressed as a probability represented in scientific notation as a negative exponent of 10. For example, an additional risk of contracting cancer of 1 chance in 10,000 (or one additional person in 10,000) is written as $1x10^{-4}$. Because CSFs are typically upper-bound estimates, actual risks may be lower than predicted (see Chapter 12) – note that the true value of the risk is unknown and may be as low as zero.[2] These statistical projections of hypothetical risk are intended as screening tools for risk managers and cannot be used to make realistic predictions of biological effects.

Risks are generally evaluated initially for **individuals** within the potentially exposed population. **Population risks** for the exposed population may also be estimated, which may be useful in estimating potential economic costs and benefits from risk reduction. Sensitive subpopulations should also be considered, when possible. Estimates of **incidence** also are possible, although there are some caveats associated with these measures (see Chapter 13).

For carcinogens being assessed based on the assumption of nonlinear dose-response, for which a reference dose (RfD) was derived that considers cancer as well as other effects, the hazard quotient approach will be appropriate for risk characterization (see Section 22.3).

22.2.2 Characterizing Risk from Exposure to Multiple Pollutants - Scenario Approach

For each exposure pathway of a scenario, exposure may be to multiple chemicals at the same time rather than a single chemical; however, CSFs are usually available only for individual compounds within a mixture. Consequently, a component-by-component approach is usually employed.[3] The following equation estimates the predicted cumulative incremental individual cancer risk from multiple substances for a single exposure pathway, assuming additive effects from simultaneous exposures to several carcinogens:

$$Risk_T = Risk_1 + Risk_2 + \ldots + Risk_i \qquad \text{(Equation 22-2)}$$

where:

$Risk_T$	=	Cumulative individual ingestion cancer risk (expressed as an upper-bound risk of contracting cancer over a lifetime); and
$Risk_i$	=	Individual ingestion risk estimate for the ith substance.

In screening-level assessments of carcinogens for which there is an assumption of a linear dose-response relationship, the cancer risks predicted for individual chemicals may be added to estimate cumulative cancer risk for each pathway. This approach is based on an assumption that

the risks associated with individual chemicals in the mixture are additive. In more refined assessments, the chemicals being assessed may be evaluated to determine whether effects from multiple chemicals are synergistic (greater than additive) or antagonistic (less than additive), although sufficient data for this evaluation are usually lacking. In those cases where CSFs are available for a chemical mixture of concern, risk characterization can be conducted on the mixture using the same procedures used for a single compound.

For carcinogens being assessed based on the assumption of nonlinear dose-response, for which an RfD considering cancer as well as other effects has been derived, the hazard quotient approach will be appropriate (see Section 22.3).

Aggregate vs. Cumulative Risk

Aggregate risk refers to risk attributed to a single chemical across multiple pathways/routes

Cumulative risk refers to risk attributed to simultaneous exposure to multiple chemicals via a single or multiple pathways/routes

22.2.3 Combining Risk Estimates across Multiple Ingestion Pathways - Scenario Approach

To evaluate risks associated with the aggregate exposure across multiple pathways of a given scenario, the individual pollutant cancer risk estimates may be summed for each chemical across the multiple ingestion pathways assessed. Additionally, a cumulative multi-pathway risk estimate may be derived by summing cumulative (multiple pollutant) cancer risk estimates across the multiple ingestion pathways.

22.2.4 Evaluating Risk Estimates from Inhalation and Ingestion Exposures

Depending on the ingestion scenario, the inhalation pathway will also have been assessed. In such cases, the inhalation exposures must be presented along with the ingestion exposures to provide an overall estimate of risk across the multiple pathways. When there is a compatibility in the exposure scenarios, inhalation and ingestion risk estimates can be combined. Essentially, an additional column for inhalation can be added to Exhibit 22-1 to achieve this result. Regardless, when both routes are assessed, risk estimates for both routes of exposure should be presented, along with descriptions regarding the populations assessed for all pathways and routes, thereby clarifying any differences in populations.

It is important to note, however, that the methods and assumptions used to derive the inhalation and ingestion risks may not always yield compatible exposure scenarios. This is particularly important when population-level (versus individual) risk estimates are being developed. For example, a scenario-based ingestion exposure assessment will not be easily amenable to producing estimates of numbers of people at different risk levels, while a population-based inhalation assessment may be more appropriate. In addition, it would generally not be appropriate to add an inhalation risk that presumes a 70-year exposure duration with an ingestion pathway that presumes a 30-year exposure duration. Any matching of exposure durations among pathways in a multipathway assessment should be carefully considered.

22.3 Noncancer Hazard

For noncancer effects (as well as carcinogens being assessed based on the assumption of nonlinear dose-response), ingestion exposure concentrations are compared to RfDs, which are estimates (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to

the human population (including sensitive sub-populations) that is likely to be without an appreciable risk of deleterious noncancer effects during a lifetime (see Chapter 21).

As with carcinogens, the development of hazard quotients (HQs) for ingestion typically is performed first for individual air toxics. Then, hazard indices (HIs) may be developed for multiple pollutant exposures and summed across pathways to develop multiple pathway cumulative hazard estimates. An additional step in the multipathway analysis is to evaluate combining both ingestion and inhalation hazard estimates. These steps are described in separate subsections below.

22.3.1 Characterizing Individual Pollutant Hazard - Scenario Approach

The first step in characterizing individual pollutant hazard for an exposure scenario (e.g., a recreational fisher) is to quantify hazard for each pollutant being evaluated. For ingestion exposures, noncancer hazards are estimated by dividing the estimate of the Average Daily Dose (ADD) by the chronic oral RfD to yield an HQ for individual chemicals:

$$HQ = ADD \div RfD \qquad \text{(Equation 22-3)}$$

where:

HQ	=	Hazard Quotient for the pollutant via each ingestion pathway being evaluated (unitless);
ADD	=	Estimate of the Average Daily Dose for the pollutant via the ingestion pathway being evaluated (mg/kg-d); and
RfD	=	Corresponding reference dose for the pollutant via the ingestion pathway being evaluated (mg/kg-d).

In screening assessments, the chronic exposure estimate is commonly based on a simplifying assumption of continued similar conditions for a long-term period (for example, that the maximum annual average modeled concentration remains constant during the full course of the exposure duration). A more refined assessment might consider how concentration changes with time over the exposure duration. In both cases, it is important to match the type of RfD value to the specific exposure scenario. For example, for childhood scenarios (e.g., ages 0-6), risk assessors commonly use chronic RfDs (rather than subchronic). Subchronic RfDs[(a)] are more commonly used to evaluate exposure scenarios that last a year or less (e.g., a construction worker who is exposed for 6 months). For exposure durations of a few years, both chronic and subchronic values may be considered, with chronic values commonly being used, particularly in screening assessments, with explicit recognition of the decision and its basis. Acute toxicity values are for exposures that are much shorter in duration (usually 24 hours or less); however, such exposures generally are not evaluated in a multipathway air toxics risk assessment.

Based on the definition of the RfD, an HQ less than or equal to one indicates that adverse noncancer effects are **not likely to occur**. With exposures increasingly greater than the RfD (i.e.,

---

[a]Although subchronic RfDs are not routinely developed by EPA, ATSDR develops MRLs for "intermediate" exposures and describes them as being relevant to exposure durations on the order of weeks to months (i.e., >14 days to 364 days).

HQs increasingly greater than one), the **potential for adverse effects increases**, but we do not know by how much. An HQ of 100 does not mean that the hazard is 10 times greater than an HQ of 10. Also an HQ of 10 for one substance may not have the same meaning (in terms of hazard) as another substance resulting in the same HQ.

22.3.2 Multiple Pollutant Hazard

Noncancer health effects data are usually available only for individual compounds within a mixture. In these cases, the individual HQs can be summed together to calculate a multi-pollutant HI:

$$HI = HQ_1 + HQ_2 + ...+ HQ_i \qquad \text{(Equation 22-4)}$$

where

HI = Hazard index; and
HQ_i = Hazard quotient for the ith air toxic.

For screening-level assessments, a simple HI may first be calculated for all chemicals of potential concern (COPCs) (Exhibit 22-2). This approach is based on the assumption that even when individual pollutant levels are lower than the corresponding reference levels, some pollutants may work together such that their potential for harm is additive and the combined exposure to the group of chemicals poses greater likelihood of harm. Some groups of chemicals can also behave antagonistically, such that combined exposure poses less likelihood of harm, or synergistically, such that combined exposure poses harm in a greater than additive manner, although information needed to perform such an analysis is generally not available. Where this type of HI exceeds the criterion of interest, a more refined analysis is warranted.

The assumption of dose additivity is most appropriate to compounds that induce the same effect by similar modes of action. Thus, EPA guidance for chemical mixtures[3] suggests subgrouping pollutant-specific HQs by toxicological similarity of the pollutants for subsequent calculations; that is, calculating a **target-organ-specific-hazard index (TOSHI)** for each subgrouping of pollutants. This calculation allows for a more appropriate estimate of overall hazard.

The HI approach encompassing all chemicals in a mixture may be appropriate for a screening-level study. However, it is important to note that applying the HI equation to compounds that may produce different effects, or that act by different mechanisms, could overestimate the potential for effects. Consequently, in a refined assessment, it is more appropriate to calculate a separate HI for each noncancer endpoint of concern when target organs or modes of action are known to be similar. Refined assessments also may employ techniques more complex than the HI derived using RfDs.[4]

22.3.3 Evaluating Hazard Estimates From Inhalation and Ingestion Exposures

As with carcinogenic assessments, inhalation hazards must be combined with ingestion hazards to provide total hazard across all exposure pathways for a receptor. Similar to Exhibit 22-1, inhalation and ingestion risk estimates can be combined either by chemical across pathways or across chemicals within a pathway. Essentially, an additional column for inhalation can be added to Exhibit 22-2 to achieve this result.

Exhibit 22-2. Example Matrix for Characterizing Hazard for Multiple Chemical Exposure through Multiple Ingestion Pathways for a Particular Exposure Scenario					
	Pathway 1 (Vegetable Ingestion HQ Estimate)[a]	Pathway 2 (Fish Ingestion HQ Estimate)[a]	Pathway 3 (Egg Ingestion HQ Estimate)[a]	Pathway 4 (Beef Ingestion HQ Estimate)[a]	Aggregate Chemical Ingestion HQ Estimate[a]
Chemical 1	2×10^{-1}	2×10^{-1}	4×10^{-2}	2×10^{-1}	7×10^{-1}
Chemical 2	3×10^{-1}	7×10^{-1}	3×10^{-2}	2×10^{-1}	1
Chemical 3	1×10^{-1}	4×10^{-1}	2×10^{-1}	4×10^{-1}	1
Chemical 4	9×10^{-2}	1×10^{-2}	1×10^{-1}	2×10^{-2}	3×10^{-1}
Cumulative Ingestion Pathway HI [a]	7×10^{-1}	1	4×10^{-1}	9×10^{-1}	3

[a] Standard rules for rounding apply which will commonly lead to an answer of one significant figure in both risk and hazard estimates. For presentation purposes, hazard quotients (and hazard indices) and cancer risk estimates are usually reported as one significant figure.

22.4 Interpretation and Presentation of Risks/Hazards

In the final part of the risk characterization, estimates of cancer risk and noncancer hazard should be presented in the context of uncertainties and limitations in the data and methodology. Exposure estimates and assumptions, toxicity estimates and assumptions, and the assessment of uncertainty should be discussed. Chapter 13 provides more detailed information and examples. Part VI of this reference manual discusses risk communication and other elements of the risk-based decision-making process.

Estimating Risk for Drinking Water Sources

In evaluating potential risks associated with drinking water supplies, risk assessors commonly assume that the drinking water undergoes at least a minimum level of treatment to remove solids (i.e., particles in the water which are persistent bioaccumulative hazardous air pollutants [PB-HAPs] or onto which PB-HAPs may be absorbed). Therefore, the risk assessment commonly focuses on the dissolved concentrations of PB-HAPs in drinking water sources. In addition, if the drinking water source is part of a public drinking water system, the risk assessment may also assume that the water is treated to meet applicable drinking water standards (i.e., treated to maximum contaminant levels or MCLs, unless study-specific information indicates otherwise) for chemicals regulated under the drinking water program. National Primary Drinking Water Regulations are enforceable standards that apply to public water systems. The MCLs are the highest level of a specific list of contaminants allowed in drinking water (see http://www.epa.gov/safewater/mcl.html).

Note that multipathway air toxics risk assessments are subject to additional sources of uncertainty as compared to inhalation risk assessments. The multimedia modeling effort is both more complex and less certain due to many factors. For example: (1) there are many more chemical-dependent and chemical-independent variables involved as input values to the models;

(2) the models involve analysis of the transfer of air toxics from the air to other media (e.g., soil, sediment, water), the subsequent movement of the air toxics between these media (e.g., soil runoff to surface water), and uptake and metabolism by biota; and (3) many variables affect the ingestion of food, water, and other media by humans and wildlife, and the exposure and risk estimates may differ considerably as a consequence of the assumptions used to derive intake estimates. Sampling of biota and abiotic media also may be more complex. Additional uncertainties are incorporated in the risk assessment when exposure estimates to multiple substances across multiple pathways are summed.

References

1. U.S. Environmental Protection Agency. 2002. *Framework for Cumulative Risk Assessment (External Review Draft).* Risk Assessment Forum, Washington, DC, April 2002, available at: http://oaspub.epa.gov/eims/eimsapi.dispdetail?deid=29570

   U.S. Environmental Protection Agency. 1997. *Guidance on Cumulative Risk Assessment, Part 1, Planning and Scoping*. Science Policy Council, Washington, DC.

   U.S. Environmental Protection Agency. 1984. *Risk Assessment and Management: Framework for Decision Making*, Washington, D.C. EPA 600/9-85-002.

2. U.S. Environmental Protection Agency. 1986. *Guidelines for Mutagenicity Risk Assessment*. Risk Assessment Forum, Washington, DC. EPA/630/R-98/003; published in the Federal Register 51:(1185): 34006-34012, Sept 24, 1986, available at: http://cfpub.epa.gov/ncea/raf/pdfs/mutagen2.pdf.

3. U.S. Environmental Protection Agency. 2000. *Supplementary Guidance for Conducting Health Risk Assessment of Chemical Mixtures*. Risk Assessment Forum, Washington, D.C. EPA/630/R-00/002, available at http://www.epa.gov/ncea/raf/pdfs/chem_mix/chem_mix_08_2001.pdf.

   U.S. Environmental Protection Agency. 1986. *Guidelines for the Health Risk Assessment of Chemical Mixtures*. EPA/630/R-98/002; published in the Federal Register 51 (185):34014-34025, Sept 24, 1986, available at http://www.epa.gov/ncea/raf/pdfs/chem_mix/chem_mix_08_2001.pdf.

4. U.S. Environmental Protection Agency. 2002. *Guidance on Cumulative Risk Assessment for Pesticide Chemicals that have a Common Mechanism of Action.* Office of Pesticides Programs, Washington, D.C., January 2002. Available at: http://www.epa.gov/pesticides/trac/science/.

   U.S. Environmental Protection Agency. 2003. National Center for Environmental Assessment. *Developing Approaches to Estimate Cumulative Risks of Drinking Water Contaminants*. Updated December 30, 2003. Available at: http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=18494. (Last accessed April, 2004.)

PART IV

ECOLOGICAL RISK ASSESSMENT

Chapter 23 Overview and Getting Started: Problem Formulation

Table of Contents

23.1 Introduction 1

23.2 Overview of Air Toxics Ecological Risk Assessment 4
- 23.2.1 Problem Formulation 6
- 23.2.2 Analysis 8
- 23.2.3 Evaluation of Ecological Effects 9
- 23.2.4 Ecological Risk Characterization 9

23.3 Planning and Scoping 9
- 23.3.1 What is the Concern? 10
- 23.3.2 Identifying The Participants 11
- 23.3.3 Determining the Scope of the Risk Assessment 12
- 23.3.4 Study-Specific Conceptual Model 12
  - 23.3.4.1 Identifying Receptors of Concern 13
  - 23.3.4.2 Identifying Assessment Endpoints and Measures of Effects 15
- 23.3.5 Analysis Plan and Quality Assurance Program Plan (QAPP) 16

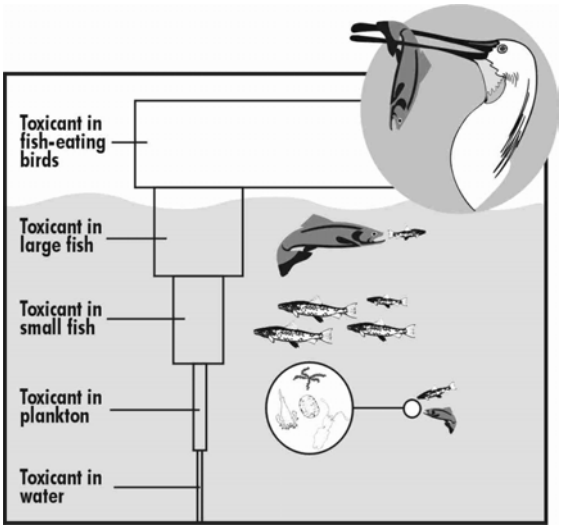
23.4 Tiered Ecological Risk Assessments 21

References 23

23.1 Introduction

Part IV constitutes a snapshot of EPA's current thinking and approach to the adaptation of the evolving methods of ecological risk assessment to the context of Federal and state control of air toxics. While inhalation risk assessment has been increasingly used in regulatory contexts over the last several years, ecological risk assessment tools are less well developed and field tested in a regulatory context. Part IV should be considered a living document for review and input. By publishing Part IV in its current state of development, EPA is soliciting the involvement of persons with experience in this field to help improve these assessment methods for use in a regulatory context. EPA anticipates revisions to this draft section of Part IV on the basis of this input.

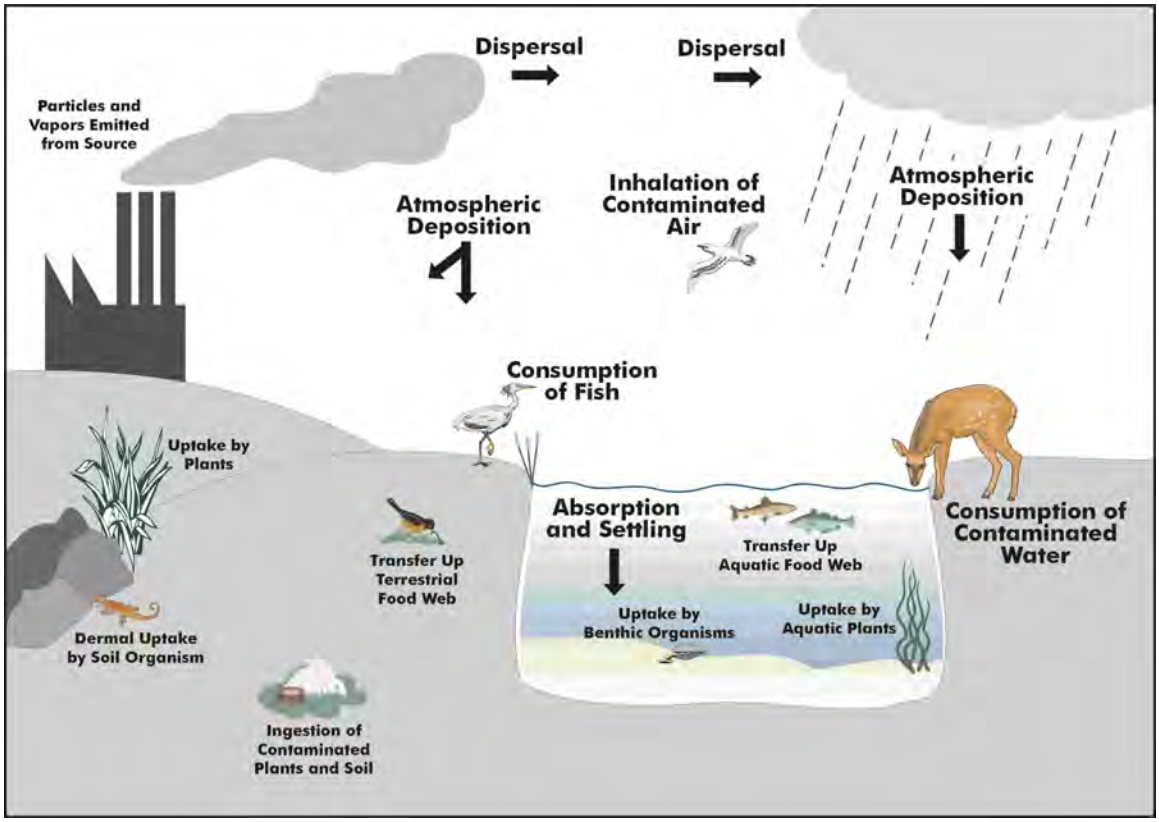
Part III of this Reference Manual discusses how to plan for and conduct a multipathway human health risk assessment when air toxics that persist and may also bioaccumulate (e.g., the persistent bioaccumulative hazardous air pollutant compounds, or PB-HAPs) in media other than air and/or biomagnify in food chains are present in releases. For these compounds, the risk assessment generally will need to include consideration of exposure pathways that involve deposition of air toxics onto soil and plants and into water, subsequent uptake by biota, and potential human exposures via consumption of contaminated soils, sediments, surface waters, and foods. These substances may also pose risks to ecological receptors from direct exposure to contaminated media or through indirect exposure via aquatic and terrestrial food chains (see Exhibit 23-1). The preliminary list of PB-HAPs was derived primarily on the basis of exposure and risk/hazard once HAPs are deposited onto soils, into surface waters, etc. Its derivation did not consider direct exposures of ecological receptors to air toxics while they are in the air (e.g., phytotoxic effects on plants; inhalation by animals). Additional HAPs of potential concern for ecological risk may be identified as EPA gains more familiarity with ecological risk assessments for air toxics. Appendix D describes the process by which EPA identified the PB-HAP compounds.



Trophic Levels and Biomagnification

The trophic level is a way to describe where an organism may be located within an aquatic or terrestrial food web. The lowest trophic level consists of primary producers, the green plants that convert sunlight into carbohydrates via photosynthesis. The next trophic level generally consists of primary consumers, or the organisms that feed directly on green plants. The next level up, often termed secondary consumers, represents animals that feed on primary consumers. The highest trophic level consists of the top predators in the food web. For some chemicals, the concentration in biological tissue can increase as it moves up the food chain, a process called **biomagnification**.

Exhibit 23-1. Air Toxics Exposure Pathways of Potential Concern for Ecological Receptors



This graphic illustrates some of the potential multimedia pathways of concern for air toxics exposure to ecological receptors. Air toxics released from a source disperse through the air and eventually fall to the earth (atmospheric deposition) via settling and/or precipitation. Air toxics deposited to soil may be absorbed or ingested by plants and soil invertebrates (uptake). Terrestrial animals may be exposed to air toxics via ingestion of contaminated plants and soil, or by consuming contaminated terrestrial animals (for those air toxics that bioaccumulate and transfer up the terrestrial food web). Air toxics deposited to water may be dissolved in the water column and/or may settle and be absorbed into aquatic sediments. Air toxics in sediments may be absorbed or ingested by benthic organisms (uptake); those in sediments and the water column may be absorbed by aquatic plants (uptake). Aquatic organisms (e.g., fish) may be exposed directly to air toxics in the water column and/or by consuming contaminated aquatic organisms (for those air toxics that bioaccumulate and transfer up the aquatic food web). Terrestrial animals may be exposed to air toxics by eating contaminated fish or shellfish and/or by drinking contaminated water. Note also that, while in the atmosphere, air toxics may also have direct impacts on plants (direct exposure) and terrestrial animals (inhalation).

This part (Part IV) of this reference manual introduces the basic concepts of ecological risk assessment and describes their application to air toxics. Several differences of particular importance are highlighted in a text box on page 23-3. The discussion of ecological risk assessment follows the same general framework as that presented in Part III since the overall concept is the same; namely that certain air toxics may move from the air into other media where exposures to organisms (in this case, non-human organisms) can occur with potentially adverse outcomes. Readers are strongly encouraged to become familiar with the information provided in Part III before reading this Part. ***However, although there are many similarities between***

multimedia human health risk assessment and ecological risk assessment (e.g., they may use the same multimedia monitoring and modeling tools), professional expertise will always be required to apply the ecological risk assessment principles and tools identified in this document to specific assessment areas or problems. This document is not a substitute for a working familiarity with ecological principles, their application, and the field of ecological risk assessment.

Air toxics may have adverse effects on ecological receptors through direct exposures (e.g., inhalation by animals; direct deposition onto plants). However, EPA does not have sufficient experience with multipathway air toxics risk assessments to identify the circumstances for which these exposures would represent a potential concern. This reference manual therefore does not address these additional exposure pathways. The methods for conducting such an analysis are described in greater detail in EPA's Guidelines for Ecological Risk Assessment.[1]

This chapter presents an overview of ecological risk assessment and discusses the initial planning and scoping activities. The remaining chapters of this part focus on Characterization of Exposure (Chapter 24), Characterization of Ecological Effects (Chapter 25), and Risk Characterization (Chapter 26). The discussion presented here is based largely on EPA's *Guidelines for Ecological Risk Assessment*[1] and the *Residual Risk Report to Congress*.[2] The *Guidelines for Ecological Risk Assessment* were developed especially for evaluating ecological risk. Readers are also strongly encouraged to become familiar with that document for a more complete understanding of EPA's recommended approach to ecological risk assessment. Interested readers are also referred to *EPA's Ecological Risk and Decision Making Workshop* materials which provide detailed information on the definition of ecological risk assessment, how it relates to human health assessment, the ecosystem protection place-based approach, and the bases for ecological protection and risk assessment at EPA.[3]

Key Ecological Risk Assessment Resources

- NCEA's Ecological Risk Assessment webpage http://cfpub.epa.gov/ncea/cfm/ecologic.cfm
- The Oak Ridge National Laboratory Ecological Risk Assessment webpage on tools, guidance, and applications http://www.esd.ornl.gov/programs/ecorisk/ecorisk.html
- The Superfund Ecological Risk Assessment Program http://epa.gov/superfund/programs/risk/ecolgc.htm
- Navy Guidance for Conducting Ecological Risk Assessments http://web.ead.anl.gov/ecorisk/
- EPA's Watershed Ecological Risk Assessment program http://cfpub.epa.gov/ncea/cfm/weracs.cfm?ActType=default

Some Important Differences Between Ecological Risk Assessment and Multipathway Human Health Risk Assessment

- **Planning and scoping**. The ecological risk assessment requires more preliminary analysis and deliberation regarding endpoints to be assessed and toxicity reference values to be used because ecological systems are more complex and are not as well understood biologically as human health systems. The planning and scoping team should include individuals with specific expertise in ecological risk assessment.

- **Assessment area**. It may be necessary to evaluate additional portions of the assessment area that are not of concern from a human health perspective.

- **Potentially exposed populations**. The focus shifts from potentially exposed individual humans to potentially exposed populations and species of ecological receptors of concern. In many cases, the exposure assessment may need to address multiple species and life-stages, many of which have physiological and biochemical processes that differ significantly from humans. (When threatened or endangered species are present, the assessment may also include an evaluation of those organisms as individuals).

- **Exposure pathways and exposure routes**. It may be necessary to assess different exposure pathways and routes that are not of concern for human health.

- **Ecological effects assessment**. Ecological systems have traits and properties that are different from humans and, thus, the ecological effects assessment (comparable to hazard assessment for human health) may consider a wider range of potential causal relationships.

- **Risk characterization**. While risks may be assessed at multiple levels of ecological organization (i.e., organism, population, community, and ecosystem), they generally are assessed at the population level in air toxics assessments. (Nevertheless, when appropriate, consideration should be given to assessments at high levels of ecological organization, such as at the landscape level).

23.2 Overview of Air Toxics Ecological Risk Assessment

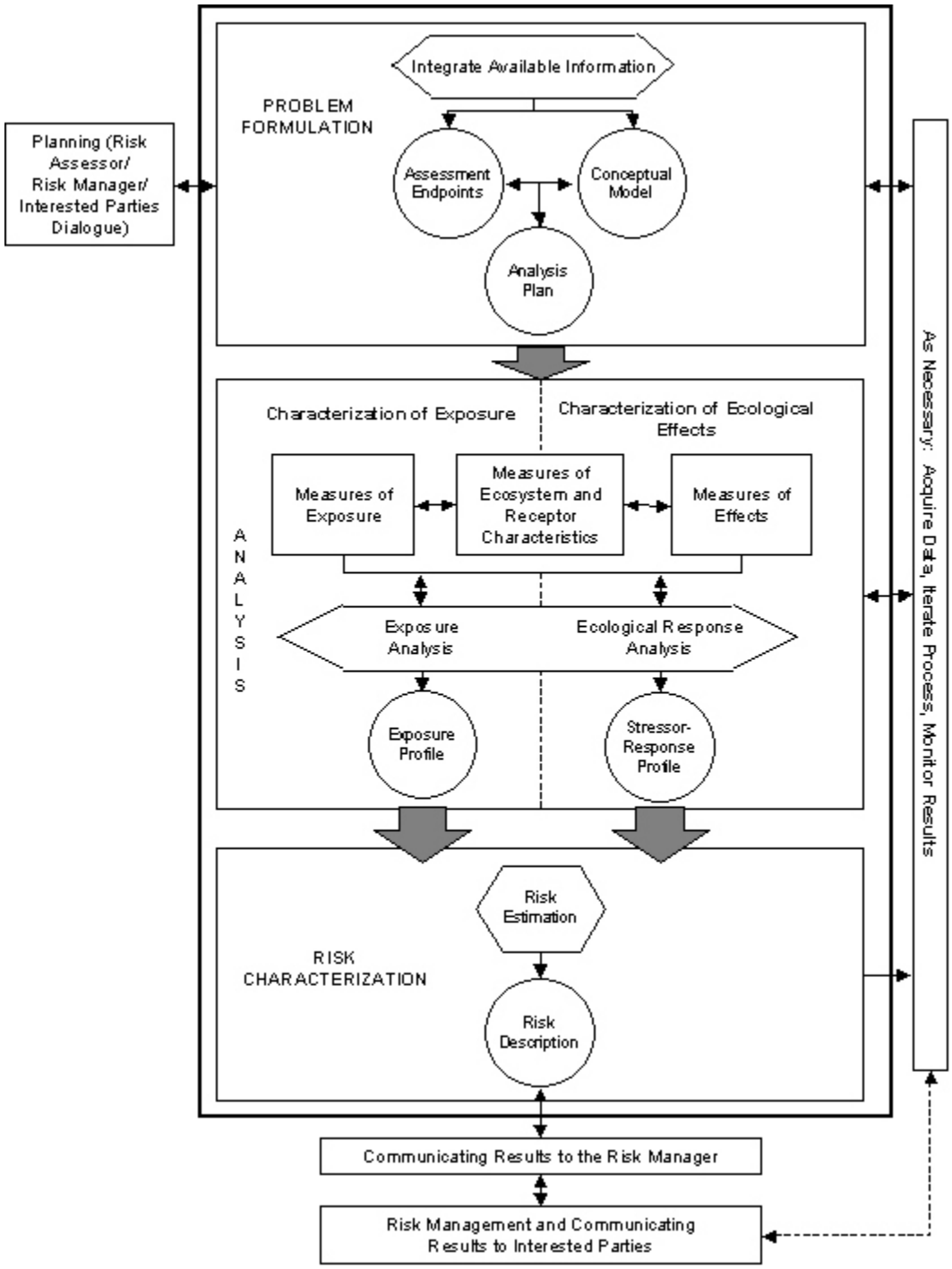
The ecological risk assessment process has three main steps that broadly correspond to the four basic steps in human health risk assessment methodology (Exhibit 23-2):[1]

- **Problem formulation**, which corresponds to the problem formulation step of the human health risk assessment methodology (planning and scoping activities similar to human health risk assessment are also integrated with this step; however, they are discussed separately below to maintain the operational structure of the ecological risk assessment as described in EPA's ecological risk assessment guidelines);

- **Analysis**, which corresponds to the exposure assessment and toxicity assessment steps of the human health risk assessment methodology; and

- **Risk characterization**, which corresponds to the risk characterization step of the human health risk assessment methodology.

Exhibit 23-2. Ecological Risk Assessment Framework



Source: EPA Guidelines for Ecological Risk Assessment[1]

23.2.1 Problem Formulation

Problem formulation provides the foundation for the entire ecological risk assessment. This step includes:

- Identifying risk management goals from an ecological perspective, ecological receptors of concern (e.g., wetlands, fish populations, keystone species that impact the overall ecosystem), and assessment endpoints (explicit expression of the environmental value that is to be protected, operationally defined by an ecological entity and its attributes);

- Developing the ecological risk part of the conceptual model as necessary to account for ecological exposure pathways and receptors; and

- If necessary, developing the Sampling and Analysis Plan and associated Quality Assurance Project Plan to collect data on exposures and measures of effects that are needed to support the ecological risk assessment.

As with human health risk assessments, problem formulation is often an iterative process, in which substantial re-evaluation may occur as new information and data become available. Data collection in subsequent iterations often is triggered by identification of major data gaps and uncertainties in the risk characterization that prevent confident decision-making by risk managers.

The problem formulation process for ecological risk assessment for air toxics focuses on developing a common understanding of what needs to be done to assess *ecological* risks associated with pathways involving deposition; the transfer of compounds to soil, water, sediment, and biota, and subsequent exposure. While the ecological risk assessment may build on the foundation of the human health multipathway assessment (e.g., using the same emissions data and multimedia models), the problem formulation step is particularly critical for the ecological risk assessment because of the effort needed to understand and identify ecological receptors, exposure pathways, endpoints, and management goals. The ecological risk assessment is not simply an "add-on" to the human health multipathway risk assessment. The problem formulation effort will need to consider a wide variety of possible ecological receptors that are not similar to humans. For example:

- Different species (and life stages) may have very different responses to the same exposure. Therefore, knowledge of the exposure-response of many species, including those that may be particularly sensitive to the air toxic, is needed.

- Ecosystems may show adverse effects at lower exposures than most individual species do because species that are important in terms of ecosystem function (e.g., energy flow, nutrient recycling) may also be sensitive to toxic effects. Ecosystem-level metrics such as species diversity indices may be more sensitive indicators of adverse effects than are toxicological studies.

- There may be many different types of ecosystems present in the assessment area, and sensitivity likely varies among them. Therefore, the particular features of the ecosystem(s) that occur in areas where high exposures are predicted may be particularly important.

An Ecological Risk Assessment Case Study: Ozone Risks To Agroecosystems

The case study summarized here provides an example of how EPA has assessed environmental risks from an air pollutant (ozone) as part of EPA's effort to promulgate National Ambient Air Quality Standards (NAAQS) for criteria air pollutants (see Chapter 2). Note that this example is for ozone, a criteria air pollutant; however, the concepts presented here are relevant to air toxics risk assessment. In addition, an agroecosystem, such as the system discussed here, is more of a human construct than a natural ecosystem and is provided here only for illustration of general principles. An actual air toxics ecological risk assessment of a natural system would have to consider site-specific characteristics of the system in question.

Problem Formulation. Pursuant to the Clean Air Act (CAA), EPA is required to set NAAQS for "any pollutant which, if present in the air, may reasonably be anticipated to endanger public health or welfare and whose presence in the air results from numerous or diverse mobile and/or stationary sources." EPA develops public health (primary) and welfare (secondary) NAAQS. According to section 302 of the CAA, the term welfare "includes ... effects on soils, water, crops, vegetation, manmade materials, animals, wildlife, weather, visibility, and climate, damage to and deterioration of property, and hazards to transportation, as well as effects on economic values ...." A secondary standard, as defined in section 109(b)(2) of the CAA, must "specify a level of air quality the attainment and maintenance of which in the judgment of the Administrator, based on such criteria, is requisite to protect the public welfare from any known or anticipated adverse effects associated with the presence of such air pollutant in the ambient air."

This case study focuses on an assessment endpoint for agricultural crops (e.g., the prevention of an economically adverse reduction in crop yields). Yield loss is defined as an impairment of, or decrease in, the value of the intended use of the plant. This concept includes a decrease in the weight of the marketable plant organ, reduction in aesthetic values, changes in crop quality, and/or occurrence of foliar injury when foliage is the marketable part of the plant. These types of yield loss can be directly measured as changes in crop growth, foliar injury, or productivity, so they also serve as the measures of effect for the assessment.

Exposure Analysis. EPA used ambient ozone monitoring data across the U.S. and a Geographic Information System (GIS) model to project national cumulative, seasonal ozone for the maximum three month period during the summer ozone season. This allowed EPA to project ozone concentrations for some rural parts of the country where no monitoring data were available but where crops were grown, and to estimate the attainment of alternative NAAQS scenarios. The U.S. Department of Agriculture's (USDA's) national crop inventory data were used to identify where ozone-sensitive crop species were being grown and in what quantities. This information allowed the Agency to estimate the extent of exposure of ozone-sensitive species under the different scenarios.

Ecological Effects Analysis. Stressor-response profiles describing the relationship between ozone and growth and productivity for 15 crop species representative of major production crops in the U.S. (e.g., crops that are economically valuable to the U.S., of regional importance, and representative of a number of crop types) had already been developed from field studies conducted from 1980 to 1986 under the National Crop Loss Assessment Network (NCLAN) program. The NCLAN studies also included secondary stressors (e.g., low soil moisture and co-exposure with other pollutants like sulfur dioxide), which helped EPA interpret the environmental effects data for ozone.

Risk Characterization. Under the different NAAQS scenarios, the Agency estimated the increased protection from ozone-related effects on vegetation associated with attainment of the different NAAQS scenarios. Monetized estimates of increased protection associated with several alternative standards for economically important crops were also developed. This analysis focused on ozone effects on vegetation since these public welfare effects are of most concern at ozone concentrations typically occurring in the U.S. By affecting commercial crops and natural vegetation, ozone may also indirectly affect natural ecosystem components such as soils, water, animals, and wildlife.

Source: U.S. Environmental Protection Agency. 1999. *Residual Risk Report to Congress*. Office of Air Quality Planning and Standards, Research Triangle, NC, March 1999. EPA-453/R-99-011.

23.2.2 Analysis

Analysis includes two principal steps. **Characterization of exposures** includes identifying the **contaminants of potential ecological concern** (COPECs) that may affect ecological receptors, characterizing the spatial and/or temporal pattern of stressor concentrations in environmental media (including certain body burden levels), and analyzing the level of contact or co-occurrence (exposure) between the stressors and the ecological receptors. This often is done using the multimedia models identified in Chapter 18; however, different models or approaches may be appropriate. **Characterization of ecological effects** includes identifying the types of effects that different stressors may have on ecological receptors, along with characterizing the **stressor-response relationship** (the relationship between the level of exposure to the stressor and the expected biological or ecological response). A common result is the identification of **ecological toxicity reference values (TRVs)**, which are concentrations of chemicals in environmental media (including biota such as fish tissues) below which no significant ecological effects are anticipated. TRVs are similar, in concept, to RfDs (reference doses) and RfCs (reference concentrations) for human health noncancer evaluations. TRVs may be screening level (i.e., conservative, generic values) or more refined values for use in higher levels of analysis. They may be point values, ranges, or developed using more advanced probabilistic methods (such as Monte Carlo techniques). The ecological exposure characterization also is likely to differ significantly from the corresponding multipathway exposure assessment for human health. For example:

- In addition to food chain (ingestion) exposures, many ecological receptors can be exposed to air toxics via direct contact with contaminated soils (e.g., earthworms) or sediments (e.g., sediment-dwelling invertebrates, bottom-feeding fish); direct exposure to surface water (e.g., free-swimming invertebrates and fish); or direct exposure to contaminated air via inhalation (e.g., birds), dermal contact (e.g., amphibians), deposition to plant surfaces, etc.

- Particular geographic areas of concern may differ because ecological receptors may occur in areas rarely used by human populations (e.g., large wetland areas, ponds where people rarely fish).

- Sampling and analysis may involve a wider range of media (e.g., sediment) and different types of biota (e.g., earthworms, aquatic invertebrates). Each type of sampling and analysis has its own methods, protocols, and Quality Assurance/Quality Control (QA/QC) procedures.

- Quantitative metrics of exposure may include both direct and indirect exposures for ecological receptors. Quantification of direct exposure is similar to human health inhalation analyses, in which ambient concentrations of COPECs in soil, water, and/or sediment are compared to corresponding TRVs. Quantification of indirect exposure via ingestion is similar to that for human health ingestion analyses, except that different food items may be involved, and the appropriate ecological **exposure factors** (e.g., diet, body weight) will be different. As with human health analyses, many exposure factors can be treated either as constants or as distributions in the exposure assessment. Ecological exposure assessments for ingestion pathways frequently use bioenergetic models to more explicitly relate intake to adverse effects.[4]

23.2.3 Evaluation of Ecological Effects

The characterization of ecological effects is similar to a toxicity assessment for human health. It considers the types of adverse effects associated with chemical exposures, stressor-response relationships, and related uncertainties. There are two primary differences:

- Adverse effects of concern generally focus at the population, community, or ecosystem level. With rare exceptions (e.g., threatened or endangered species), effects to individual organisms are not the primary concern. Note, however, that ecological risk assessments often use estimates of impacts to individual organisms (e.g., mortality, reproductive effects) to infer impacts at higher levels of organization because exposure-response data for populations, communities, or ecosystems often are lacking. Some approaches are available, however, for incorporating population-level analysis in ecological risk assessments.[5]

- A distinction is made between **assessment endpoints**, which are the environmental values to be protected, and **measures of effects**, which are the specific measures used to evaluate risk to the assessment endpoints (assessment endpoints and measures of effects are defined in Section 23.3.4.2).

23.2.4 Ecological Risk Characterization

Similar to human health risk characterization, ecological risk characterization combines information concerning exposure to chemicals with information regarding effects of chemicals to estimate risks. Human health risk assessments consider health effects in the bodies of individual people. Ecological risk assessments consider various "health" issues that can range from actual health effects in the bodies of individual ecological receptors to something more attuned to the "health" of the ecosystem as measured by species richness and diversity.

23.3 Planning and Scoping

To ensure that the ecological risk assessment will provide information useful to the risk managers who will be making the risk management decisions, EPA's *Guidelines for Ecological Risk Assessment* recommends a planning and scoping dialogue occur between the risk assessors, risk managers, and where appropriate, interested stakeholders at the very start of the risk assessment process. The outcome of the planning and scoping phase is an agreement on the basic goals, scope, and timing of the risk assessment. Important goals of the dialogue are the identification of the risk management goals and risk management options that the risk assessment will be designed to inform (see accompanying text box). This 'kick-off' dialogue sets the stage for the problem formulation phase, when the plans for the ecological risk assessment are finalized.

Planning and Scoping the Ecological Risk Assessment

The planning phase is complete when agreements are reached on:

- The management goals for ecological values;
- The range of management options the risk assessment is to support;
- Objectives for the risk assessment, including criteria for success; and
- The focus and scope of the assessment, and resource availability.

When actually performing the problem formulation phase of an ecological risk assessment, the five-step planning and scoping process identified for human health risk assessments is a helpful tool to get the right people involved and the risk questions, expectations, and plans in place to make the overall assessment go smoothly and in a scientifically responsible manner. Similar to the human health evaluation process, the risk assessment and management team should be assembled to start identifying the concern, identifying who needs to be involved in the risk assessment process, determining the scope of the risk assessment, describing why there may be a problem, and determining how the concern will be evaluated.

23.3.1 What is the Concern?

In human health risk assessment and risk management, the assessors are dealing with a single organism (human beings) and the precedent and rationale for specific risk management goals (such as the $1x10^{-6}$ to $1x10^{-4}$ cancer risk range) are generally well established. The parallel process for ecosystems, however, is not as easy to study or as straightforward to manage. To begin with, it can be difficult to choose which of many organisms in a study area to evaluate. Moreover, there is little agreement on which (if any) organisms or ecosystems are important enough to single out for protection. These factors make planning, evaluation, and management of ecological risks more complicated and time-consuming (and often, more controversial).

EPA's Risk Assessment Forum developed draft guidance[(6)] to help decision-makers work with risk assessors, stakeholders, and other analysts to plan for ecological risk assessments that will effectively inform the decisions they need to make. Planning for ecological risk assessment includes three primary steps:

1. **Defining the risk management decision to be made, the context in which it will be made, and its purpose**. This includes articulating the decision or problem that the risk manager faces, understanding the social and legal context for the decision, placing preliminary boundaries on the scope of the risk assessment, and identifying who needs to be involved. Appropriately framing the context will help ensure that management objectives are relevant to the risk manager's decision and increase the likelihood that the information generated by the risk assessment will be useful.

2. **Developing objectives**. This starts with a clear statement of the problem, issue, or opportunity identified in the first step and ends with a set of specific objectives which will guide all of the remaining steps. An important determination is the "what to protect" (i.e., the assessment endpoint) question for ecological issues and to describe what is at stake. Key questions include:
   - What should be protected? Define the entities, ecological processes, and geographic areas to be considered.
   - How is "protection" defined? Define the ecological objectives.
   - What are the most important objectives and how can they be achieved? Review and structure objectives.

   In some cases, there is a strong consensus on "what to protect" (e.g., if a commercially important resource such as a fishery is potentially exposed). In many other cases, it is not always obvious to a risk manager or the public what features of an ecosystem are of potential concern or what the broader consequences would be from adverse effects to those features.

Developing a consensus on the specific risk management objectives may be a difficult and time-consuming part of the planning and scoping process.

3. **Identifying what information is needed to inform the decision**. When identifying information needs, planners are encouraged to think ahead about everything that will be needed to decide what to do about identified risks. Ecological risk is part of the picture, but issues such as feasibility, practicability, cost, and acceptability also need to be factored into the decision. They should also consider who and what resources are available to perform the ecological risk assessment. The aim of this step is to narrow down which questions the risk assessment should address and identify those that will be addressed elsewhere.

The questions identified at this step will be examined during the remainder of the problem formulation process. Management objectives are by definition closely related to the assessment endpoints evaluated in ecological risk assessment, and it should be possible to characterize them using the measures described below.

Assessment Endpoints

According to EPA's *Guidelines for Ecological Risk Assessment*,[1] an assessment endpoint is an explicit expression of the environmental value that is to be protected, and is operationally defined by an ecological entity and its attributes. For example, a particular area has air toxics releases that may be affecting area salmon populations that are important for location recreation and commercial fishermen as well as an important resource for a local Native American tribe. In the study area, the salmon population is the valued ecological entity; reproduction and age class structure of a salmon population are some of their most important attributes. An appropriate assessment endpoint for this study area might be stated as *salmon reproduction and age class structure.* The ecological risk assessment for this study area would be structured to evaluate whether this specific salmon population is at risk from air toxics with regard to healthy reproductive ability and age class structure.

Given the diversity of species and other ecological attributes in almost any study area, the assessors generally establish at least one assessment endpoint that will, together, provide an assessment of air toxics impacts on the ecosystem as a whole. More than one assessment endpoint may be necessary at the ecosystem level.

23.3.2 Identifying The Participants

The participants for the ecological risk assessment may include some of the same people as those for the human health multipathway risk assessment (e.g., multimedia modelers that understand how to model for both human and ecological receptors). However,

- Additional risk managers may be involved, including natural resource management agencies such as the U.S. Fish and Wildlife Service; state, local, or tribal (S/L/T) fish and game departments; and/or private-sector risk managers.

- The risk assessment technical team will need significantly different experts (e.g., aquatic ecologists, experienced ecological risk assessors).

- The specific set of interested or affected parties may change or be expanded (e.g., different environmental groups may be more concerned/involved; local fishermen may become interested).

EPA's Public Involvement Policy may be helpful in performing this task (see http://www.epa.gov/stakeholders/policy2003/index.htm). Part V of this document provides additional information on community involvement.

23.3.3 Determining the Scope of the Risk Assessment

The scope of the human health multipathway risk assessment may expand to include additional exposure pathways and exposure routes, and to address ecological receptors of concern.

- The specific chemicals that will be the focus of the ecological risk assessment will generally be those that persist, bioaccumulate, and biomagnify (the PB-HAP compounds); however, a different set of PB-HAP compounds may be of more concern for the ecological risk assessment than for human health risk assessment. As with human health risk assessment, additional compounds may need to be added to the analysis, depending on study-area specific considerations.

- The specific sources included in the analysis may be focused on the subset that releases most or all of the identified COPECs.

- The physical boundaries of the study area may need to expand to include geographic areas where COPECs may be transported after deposition (e.g., the COPECs may have the potential to be deposited in a watershed and be carried out of the geographic area defined for the human health multipathway modeling).

23.3.4 Study-Specific Conceptual Model

A study-specific conceptual model for the ecological risk assessment is developed using the fundamental elements of the conceptual model developed for the human health multipathway assessment as a starting point. Steps to develop the study-specific ecological risk conceptual model include the following:

- Determine whether the set of potential sources and chemicals that were identified in the human health multimedia risk assessment are appropriate for the ecological risk assessment.

- Consider expanding the set of potential sources, chemicals, and exposure pathways to include those identified below (potential exposure pathways are listed in Exhibit 23-3).

- Identify ecological receptors of concern (see Section 23.3.4.1).

- Formulate a **risk hypothesis** that describes possible relationships between emissions of a chemical, exposure, and assessment endpoint response, including the information that sets the problem in perspective, as well as an identification of the proposed relationships that need evaluation.

- Identify assessment endpoints and measures of effects (See Section 23.3.4.2).

Exhibit 23-3. Common Exposure Pathways Considered for Ecological Air Toxics Risk Assessments
Direct exposure pathways: air → soil → soil-dwelling biota air → soil → water → aquatic biota air → water → aquatic biota air → water → sediment → aquatic biota air → soil → water → sediment → aquatic biota air → vegetation **Indirect exposure pathways:** air → vegetation → bird/mammal air → soil → vegetation → bird/mammal air → soil → water → aquatic biota → fish air → soil → water → aquatic biota → fish → bird/mammal air → water → aquatic biota → fish air → water → aquatic biota → fish → bird/mammal air → soil → water → sediment → aquatic biota → fish air → soil → water → sediment → aquatic biota → fish → bird/mammal

Conceptual model diagrams, such as the example illustrated in Exhibit 23-4, are used (along with the risk hypothesis) to select the pathways to be evaluated in the analysis phase of the ecological risk assessment, as well as to assist in communication with risk managers.

As with human health risk assessments, the conceptual model for an ecological risk assessment must provide both a graphical representation of the important exposure pathways that are presumed to be occurring along with a written description that outlines each element of the conceptual model. Taken together, these two parts of the conceptual model clearly identify the sources of concern, the COPECs that will be evaluated, the exposure pathways, and the assessment endpoints. Similar to conceptual models for human health analysis, the conceptual model may be modified (perhaps a number of times) as more is learned about the study area.

23.3.4.1 Identifying Receptors of Concern

Ecological receptors of concern are an important part of the conceptual model. These may be plants, animals, habitats, communities, or larger ecosystem elements. Specific receptors may be of concern for a variety of reasons, including:

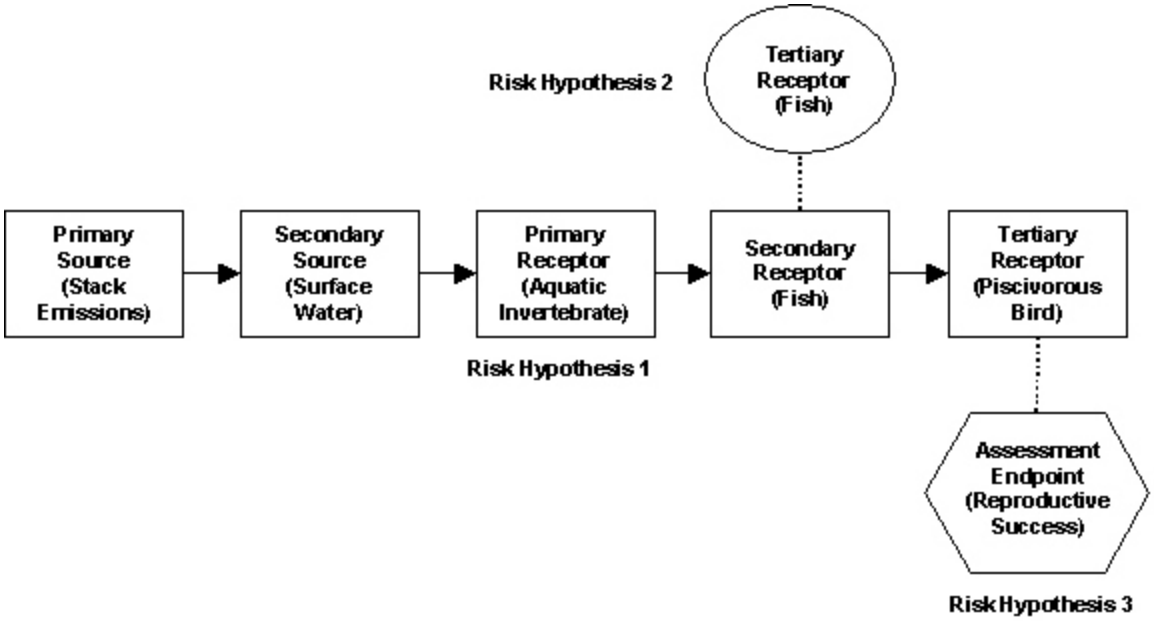
- The receptor (or one of it's life stages) is particularly vulnerable or sensitive to one or more COPECs;

- The receptor (usually a species or a community such as a wetland) is listed as endangered or threatened or is otherwise given special legal protection by the state or federal government;

- The receptor plays an important part in the overall structure or function of the ecological community or ecosystem;

- The receptor is of particular economic or cultural value to stakeholders.

Exhibit 23-4. Conceptual Model Diagram for Exposure of Piscivorous Birds to Air Toxics



Conceptual model diagrams are used, along with the risk hypothesis, to select the pathways to be evaluated in the analysis phase of the ecological risk assessment, as well as to assist in communication with risk managers. The three risk hypotheses in this hypothetical example are:

- **Risk Hypothesis 1**: Concentrations of chemical X in the surface water column are less than a level known to cause adverse effects on survival and reproduction in *Daphnia*
  - Mechanism: Chemical X causes mortality and inhibits larval development

- **Risk Hypothesis 2**: Dietary intake levels of chemical X in lake trout are less than a level known to cause adverse effects on reproductive ability
  - Mechanism: Due to a lack of enzyme A in lake trout, chemical X rapidly accumulates in lipid tissues and damages reproductive organs

- **Risk Hypothesis 3**: Dietary intake levels of chemical X in kingfisher chicks (passed to them by their parents) is less than a level known to adversely affect their survival
  - Mechanism: Chemical X accumulating in egg lipids is a metabolic toxin to the developing embryo

For taxonomic, physiological, and exposure reasons, it is important to consider a broad range of potential ecological receptors during problem formulation. For example, the types of adverse effects that may occur to terrestrial plant communities (e.g., impacts to photosynthesis, nitrogen fixation, nutrient uptake; foliar damage) are very different than the types of adverse effects that may occur to terrestrial mammals. Many ecological receptors (e.g., molds, lichens, many invertebrates) have unique physiological and biochemical features that may make them particularly sensitive to air toxics. Sensitive life stages often are a particular concern. In surface waters and sediments, early life stages (e.g., eggs, larvae) may be particularly sensitive to contaminants due to their small size (e.g., contaminants may readily penetrate cell membranes) and developmental processes (e.g., major metamorphosis from one life stage to another). Many terrestrial organisms (e.g., amphibians, dragonflies) have aquatic-dwelling early life stages. In addition, many invertebrates that can bioaccumulate PB-HAPs (e.g., aquatic dwelling dragonfly larvae) may be sources of food for sensitive life stages of other species (e.g., nestling birds). Often it is important to understand the aquatic and terrestrial food webs in the habitats of concern because these can be important parts of ecological exposure pathways. Top predators are often of special concern for exposure to PB-HAP compounds.

Ecological receptors for each habitat potentially impacted should be identified to ensure (1) plant and animal communities representative of the habitat are represented by the habitat-specific food web, and (2) potentially complete exposure pathways are identified. Screening-level ecological assessments often focus on the most sensitive organisms within an ecosystem or on the most sensitive life stages within a species, if these are known. Ecological receptor identification may need to include species both known and expected to be present in a specific habitat being evaluated, and include resident and migratory populations. Consultation with ecological experts is recommended. Potential sources of information include:

- **Government Organizations**. The U.S. Fish and Wildlife Service has biologists and other ecological experts and also maintains National Wetland Inventory maps.[7] State Natural Heritage Programs provide maps or lists of species based on geographic location, and are very helpful in identifying threatened or endangered species or areas of special concern.

- **Private or Local Organizations**. Private or professional organizations that are examples of sources of information include: National Audubon Society, the Nature Conservancy, local wildlife clubs, and universities.

- **General Literature**. Monographs, field guides, and other literature describing the flora and fauna of America and/or a particular region or state may be useful sources of information.

23.3.4.2 Identifying Assessment Endpoints and Measures of Effects

As previously noted, an **assessment endpoint** is an explicit expression of the environmental value that is to be protected or is of concern. It includes the identification of the ecological entity for the analysis (e.g., a species, ecological resource, habitat type, or community) as well as the attribute of that entity that is potentially at risk and important to protect (e.g., reproductive success, production per unit area, surface area coverage, biodiversity). The **measures of effects** are the measures used to assess these endpoints.[8]

Generally, a manageable subset of the most important assessment endpoints is selected for the risk assessment, and specific measures of effects that address each assessment endpoint are identified. EPA guidance documents discuss additional issues that are important in the identification of assessment endpoints.[9]

Appropriate selection of relevant assessment endpoints is critical so that the risk assessment provides valuable information for the associated risk management decisions. Assessment endpoints that can be measured directly are most effective, although assessment endpoints that cannot be measured directly, but can be represented by measures that are easily monitored or modeled, may also be used. Additional uncertainty is introduced depending on the relationship between the measurement and the assessment endpoints. Exhibit 23-5 provides examples of assessment endpoints, measures of effect, and other elements of the problem formulation phase.

EPA has recently released guidance that describes a set of endpoints, known as Generic Ecological Assessment Endpoints (GEAE), that can be considered and adapted for specific ecological risk assessments.[9] The entities and properties comprising the initial set of GEAEs is presented in Exhibit 23-6. The EPA Guidance defines GEAE further and provides the basis for the terms *assessment community* and *assessment population*, which are used in the definitions. In addition, EPA's Science Advisory Board recently published a *Framework for Assessing and Reporting on Ecological Condition*,[10] which includes a checklist of ecological attributes that should be considered when conducting ecological risk assessments and developing ecological management objectives (Exhibit 23-7). Note that many of these GEAEs and attributes focus at levels of ecological organization higher than organisms (e.g., species richness) or on ecological processes (e.g., nutrient cycling) rather than attributes of organisms (e.g., growth, reproduction).

It often is useful to summarize the results of the problem formulation process in a **problem formulation summary** that lists management objectives, assessment endpoints, and the structure of the risk assessment from exposure scenarios through risk characterization. Exhibit 23-8 provides an example problem formulation summary.

23.3.5 Analysis Plan and Quality Assurance Program Plan (QAPP)

As noted in Parts II and III of this reference manual, the Analysis Plan and QAPP are formulated by considering both the the conceptual model and the data quality required for the risk management decision. The Analysis Plan and QAPP, including data quality objectives, are just as important for the ecological risk assessment as they are for the human health risk assessment, and in some cases may be more complex. The analysis plan for the ecological risk assessment will need to match each of the elements of the conceptual model with the analytical approach that will be used to develop data about the element, including: sources; exposed populations and exposure pathways; exposure concentrations of COPEC; exposure conditions; toxicity of COPECs; risk characterization; QA/QC; documentation; roles and responsibilities; resources; and schedule.

Because the focus is on ecological receptors, additional types of monitoring (sampling and analysis) may need to be conducted. For example, it may be important to measure concentrations of COPECs in the sediments of surface water bodies as part of the analysis of direct exposures for sediment-dwelling invertebrates as well as bioaccumulation from these invertebrates to predatory fish through the aquatic food web.

Exhibit 23-5. Example of Ecological Risk Assessment Problem Formulation: EPA's Water Quality Criteria

A specific example of elements of the problem formulation step in a national-level ecological risk assessment can be found in the development of Ambient Water Quality Criteria by EPA's Office of Water pursuant to the Clean Water Act (CWA).[11] Water quality criteria have been developed for the protection of aquatic life from chemical stressors. The following elements of problem formulation support subsequent analyses in the risk assessments used to establish specific criteria.

Regulatory Goal

- CWA Section 101: Protect the chemical, physical, and biological integrity of the Nation's water.

Program Management Decisions

- Protect 99 percent of individuals in 95 percent of the species in aquatic communities from acute and chronic effects resulting from exposure to a chemical stressor.

Assessment Endpoints

- Survival of fish, aquatic invertebrates, and algal species under acute exposure
- Survival, growth, and reproduction of fish, aquatic invertebrates, and algal species under chronic exposure

Measures of Effect

- Laboratory LC_{50}s for at least eight species meeting certain requirements
- Chronic no-observed-adverse-effect-levels (NOAELs) for at least three species meeting certain requirements

Measures of Ecosystem and Receptor Characteristics

- Water hardness (for some metals)
- pH

The water quality criterion is a TRV derived from a distributional analysis of single-species toxicity data. It is assumed that the species tested (which represent a range of taxonomic groups) adequately represent the composition and sensitivities of species in a natural community.

Exhibit 23-6. Generic Ecological Assessment Endpoints[a]		
Entity	**Attribute**	**Identified EPA Precedents**
Organism-level endpoints		
Organisms (in an assessment population or community)	Kills (mass mortality, conspicuous mortality)	Vertebrates
	Gross anomalies	Vertebrates, shellfish, plants
	Survival, fecundity, growth	**Endangered species, migratory birds, marine mammals, bald and golden eagles**, vertebrates, invertebrates, plants
Population-level endpoints		
Assessment population	Extirpation	Vertebrates
	Abundance	Vertebrates, shellfish
	Production	Vertebrates (game/resource species), harvested plants
Community and ecosystem-level endpoints		
Assessment communities, assemblages, and ecosystems	Taxa richness	Aquatic communities, **coral reefs**
	Abundance	Aquatic communities
	Production	Plant assemblages
	Area	**Wetlands, coral reefs**, endangered/rare ecosystems
	Function	**Wetlands**
	Physical structure	Aquatic ecosystems
Officially designated endpoints		
Critical habitat for endangered or threatened species	Area Quality	
Special places	Ecological properties that relate to the special or legally protected properties	e.g., **National Parks, National Wildlife Refuges, Great Lakes**

[a]Generic ecological assessment endpoints for which EPA has identified existing policies and precedents (in particular, the specific entities listed in the third column). Bold indicates protection by federal statute.
Source: EPA's *Generic Ecological Assessment Endpoints (GEAE) for Ecological Risk Assessment*[9]

Exhibit 23-7. Essential Ecological Attributes and Reporting Categories	
Landscape Condition • Extent of ecological system/habitat types • Landscape composition • Landscape pattern and structure **Biotic Condition** • Ecosystems and communities – Community extent – Community composition – Trophic structure – Community dynamics – Physical structure • Species and populations – Population size – Genetic diversity – Population structure – Population dynamics – Habitat suitability • Organism condition – Physiological status – Symptoms of disease or trauma – Signs of disease **Chemical and Physical Characteristics (Water, Air, Soil, and Sediment)** • Nutrient concentrations – Nitrogen – Phosphorus – Other nutrients • Trace inorganic and organic chemicals – Metals – Other trace elements – Organic compounds • Other chemical parameters – pH – Dissolved oxygen – Salinity – Organic matter – Other • Physical parameters	**Ecological Processes** • Energy flow – Primary production – Net ecosystem production – Growth efficiency • Material flow – Organic carbon cycling – Nitrogen and phosphorus cycling – Other nutrient cycling **Hydrology and Geomporphology** • Surface and groundwater flows – Pattern of surface flows – Hydrodynamics – Pattern of groundwater flow – Salinity patterns – Water storage • Dynamic structural characteristics – Channel/shoreline morphology, complexity – Distribution/extent of connected floodplain – Aquatic physical habitat complexity • Sediment and material transport – Sediment supply/movement – Particle size distribution patterns – Other material flux **Natural Disturbance Regimes** • Frequency • Intensity • Extent • Duration
Source: U.S. EPA. 2002. *A Framework for Assessing and Reporting on Ecological Condition*[10]	

Exhibit 23-8. Example Problem Formulation Summary

1. **Management Objective**
   - Bald eagle (entity), local population size (attribute), should be stable (desired state)

2. **Assessment Endpoints**
   - Bald eagle (entity), reproduction (measurable attribute)
   - Bald eagle (entity), chick survival (measurable attribute)

3. **Exposure Scenario**
   - Sediment –> pore water –> benthic invertebtrates –> forage fish –> bald eagle

4. **Risk Hypothesis**
   - Dose of chemical X to adult bald eagles from consumption of fish is less than a level known to cause adverse effects on reproductive ability
     - Mechanism: Chemical X damages reproductive organs (or interferes with egg shell development)
   - Dose of chemical X to bald eagle chicks (passed to them by their parents) is less than a level known to adversely affect their survival
     - Mechanism: Chemical X accumulating in egg lipids is a metabolic toxin to the developing embryo

5. **Metrics of Exposure**
   - Concentration of chemical X in fish
   - Dose of chemical X received through consumption of fish

6. **Measure of Effect**
   - TRV for chemical X (NOAEL or LOAEL) where adult reproduction was an endpoint
   - TRV for chemical X (NOAEL or LOAEL) where chick survival (mortality) was and endpoint

7. **Measure of Characteristics**
   - Proximity of bald eagle nest site to potentially contaminated foraging areas
   - Proximity of alternative (non-contaminated) foraging areas to the nest site

8. **Risk Characterization**
   - HQ = Oral Intake of chemical X/TRV (separate calculations for adults and chicks)

23.4 Tiered Ecological Risk Assessments

One of the key elements in the ecological risk assessment process is deciding if and when further analysis is warranted. As with human health risk assessment, EPA recommends a tiered approach to ecological risk assessment.[1] Each of these tiers follows the basic three steps (problem formulation, analysis, and risk characterization) but with varying levels of complexity in the assessment and with varying requirements for resources. Examples of the three tiers of ecological risk assessment approaches are described briefly below.

- **Screening-Level** ecological risk assessments provide a general indication of the *potential* for ecological risk (or lack thereof) and may be conducted for several purposes including: (1) to prioritize COPECs based on their relative environmental behavior (e.g., relative potential for bioaccumulation or to exhibit chronic toxicity) or determine their relative contribution to the overall risk estimate; (2) to estimate the likelihood that a particular ecological risk exists; (3) to identify the need for additional data collection efforts; or (4) to focus more detailed ecological risk assessments where warranted. Screening assessments often use simplified conservative assumptions in place of detailed modeling. For example, concentrations in aquatic invertebrates or fish might be estimated from the modeled or measured water concentrations (obtained as part of a multipathway human health risk assessment) and available bioconcentration factors (BCFs) or bioaccumulation factors (BAFs). Another example is the comparison of maximum sediment and water concentrations to screening level TRVs. A screening level assessment, while abbreviated, is nonetheless a complete risk assessment. Therefore, each assessment should include documentation supporting the risk characterization and uncertainty analysis. Some examples of screening level TRVs used in screening level ecological risk assessments are available from EPA's draft Ecological Soil Screening Level Guidance (http://www.epa.gov/superfund/programs/risk/ecorisk/guidance.pdf) and EPA Region 4 (http://www.epa.gov/region4/waste/ots/ecolbul.htm).

- **More Refined** assessments are generally used to: (1) identify and characterize the current and potential threats to the environment from an air toxics release; (2) evaluate the ecological impacts of alternative emissions control or abatement policies; and (3) establish emissions levels that will protect those natural resources at risk. A more refined assessment may contain a more intensive evaluation than a screening level assessment, and usually employs multipathway analysis to estimate if, and to what extent, ecological receptors (e.g., an oyster fishery, a wild duck population, or a unique wetland community) may be exposed. The exposure and potential impact are characterized and evaluated against predetermined assessment endpoints (i.e., edibility of oysters, sustainability of the duck population, maintenance of the integrity of the wetland community). This tier may be iterative. For example, a multipathway analysis using conservative assumptions may first be performed to identify whether any of the COPECs emitted from the sources in an area pose a potentially significant concern to one or more ecological receptors. If so, a more detailed multipathway risk assessment, using more site-specific data, may be performed. From this last stage a detailed characterization of the environmental risks is developed.

- **Probabilistic** assessments are used to increase the strength of the *predictive* evaluation of ecological risks, as well as help better evaluate distributions of observational data for an ecological risk assessment. Screening-level and more refined assessments usually utilize simplified point estimates in the development of a risk characterization, while the

probabilistic tier of assessment uses probability distributions as inputs. Therefore, this tier generally can yield risk estimates that allow for a more complete characterization of variability and uncertainty. Although probabilistic assessments generally are resource-intensive, they may be especially valuable in situations when the risks are close to a policy threshold or if the management decisions, if implemented, would require significant expenditures.

Additional Reference Materials

EPA has developed extensive technical and policy guidance on how ecological risk assessments should be planned and performed. These are available at EPA's "Tools for Ecological Risk Assessment" website http://www.epa.gov/superfund/programs/risk/tooleco.htm.

- EPA's *Guidelines for Ecological Risk Assessment*, April 1998. This document expands upon and replaces the earlier 1992 *Framework for Ecological Risk Assessment.*

- EPA's *Ecological Risk Assessment Guidance for Superfund (ERAGS): Process for Designing and Conducting Ecological Risk Assessments, Interim Final*, June 1997. This document includes processes and steps for use in ecological risk assessments at Superfund sites. This document supersedes the 1989 *RAGS, Volume II, Environmental Evaluation Manual, Interim Final*. Supplements to ERAGS include the *Eco Updates* (Intermittent Bulletin Series, 1991 to present), which provide brief recommendations on common issues for Superfund ecological risk assessments. The approaches and methods outlined in the *Guidelines* and in *ERAGS* are generally consistent with each other.

- *Risk Assessment Guidance for Superfund (RAGS): Volume 1–Human Health Evaluation Manual (Part D, Standardized Planning, Reporting, and Review of Superfund Risk Assessments)*, June 2001. This guidance specifies formats that are required to present data and results in baseline risk assessments at Superfund sites; many of these formats are useful for air toxics ecological risk assessments.

- Policy Memorandum: *Guidance on Risk Characterization for Risk Managers and Risk Assessors*, F. Henry Habicht, Deputy Administrator, Feb. 26, 1992. This policy requires baseline risk assessments to present ranges of risks based on "central tendency" and "high-end" exposures with corresponding risk estimates.

- Policy Memorandum: *Role of the Ecological Risk Assessment in the Baseline Risk Assessment*, Elliott Laws, Assistant Administrator, August 12, 1994. This policy requires the same high level of effort and quality for ecological risk assessments as commonly performed for human health risk assessments at Superfund sites.

- Policy Memorandum: *EPA Risk Characterization Program*, Carol Browner, Administrator, March 21, 1995. This policy clarifies the presentation of hazards and uncertainty in human health and ecological risk assessments, calling for clarity, transparency, reasonableness, and consistency.

- *Issuance of Final Guidance: Ecological Risk Assessment and Risk Management Principles for Superfund Sites.* Stephen D. Luftig for Larry D. Reed, October 7, 1999. This document presents six key principles in ecological risk management and decision-making at Superfund sites; these principles are also useful for air toxics ecological risk assessments.

References

1. U.S. Environmental Protection Agency. 1998. *Guidelines for Ecological Risk Assessment*. Risk Assessment Forum, Washington, D.C., April 1998. EPA/630/R095/002F. Available at: http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=12460.

2. U.S. Environmental Protection Agency. 1999. *Residual Risk Report to Congress*. EPA-453/R-99-001. Available at: http://www.epa.gov/ttn/oarpg/t3/reports/risk_rep.pdf.

3. U.S. Environmental Protection Agency. 1995. *Ecological Risk and Decision Making Workshop*. December 1995. EPA 230/B96/004B.

4. Moore, D.W.J., et al. 1999. A probabilistic risk assessment of the effects of methylmercury and PCBs on mink and kingfishers along East Fork Poplar Creek, Oak Ridge, Tennessee, USA. *Environmental Toxicology and Chemistry* 8:2941-2953.

5. Suter II, G.W., et al. 2000. *Ecological Risk Assessment of Contaminated Sites*. Lewis Publishers, Boca Raton, FL. (see pages 228-231).

6. U.S. Environmental Protection Agency. 2001. *Planning for Ecological Risk Assessment: Developing Management Objectives (External Review Draft).* Risk Assessment Forum, Washington, D.C., June 2001. EPA/630/R01/001A. Available at: http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=20683

7. U.S. Fish and Wildlife Service. 2003. National Wetlands Inventory Maps. Available at: http://nwi.fws.gov.

8. U.S. Environmental Protection Agency. 1992. *Framework for Ecological Risk Assessment.* Risk Assessment Forum, Washington, D.C., February 1992. EPA/630/R92/001.

9. U.S. Environmental Protection Agency. 2003. *Generic Ecological Assessment Endpoints (GEAE) for Ecological Risk Assessment.* Risk Assessment Forum, Washington, D.C., October 2003. Available at: http://cfpub.epa.gov/ncea/raf/recordisplay.cfm?deid=55131.

10. U.S. Environmental Protection Agency. 2002. *A Framework for Assessing and Reporting on Ecological Condition*. Science Advisory Board, Washington, D.C., September 2002. EPA-SAB-EPEC-02-009A. Available at: http://www.epa.gov/sab/pdf/epec02009a.pdf.

11. U.S. Environmental Protection Agency. 1985. *Guidelines for Deriving Natural Numerical Water Quality Criteria for the Protection of Aquatic Organisms and Their Uses.* Office of Water Regulations and Standards, Washington, D.C. EPA/822/R85/100. Available at: http://yosemite.epa.gov/water/owrccatalog.nsf/0/3fab714d53e9ae5385256b0600723bd3?OpenDocument

Chapter 24 Analysis: Characterization of Ecological Exposure

Table of Contents

24.1 Introduction 1

24.2 Characterization of Exposure 1
- 24.2.1 Quantifying Releases 3
- 24.2.2 Estimating Chemical Fate and Transport 3
  - 24.2.2.1 Physical and Chemical Parameters 3
  - 24.2.2.2 Multimedia Modeling 3
  - 24.2.2.3 Multimedia Monitoring 4
- 24.2.3 Quantifying Exposure 5
  - 24.2.3.1 Metrics of Exposure 5
  - 24.2.3.2 Dimensions of Exposure 7
  - 24.2.3.3 Exposure Profile 9
  - 24.2.3.4 Evaluating Variability and Uncertainty 9

References 10

24.1 Introduction

As noted in the previous chapter, the analysis step of ecological risk assessment includes both characterization of exposures and characterization of ecological effects. This chapter describes the approaches and methods used for exposure characterization. Chapter 25 discusses the approaches and measures used for characterization of ecological effects. The discussion in this chapter is based largely on EPA's *Guidelines for Ecological Risk Assessment*.[1] Readers are referred to that document for a more complete discussion of available approaches and methods.

24.2 Characterization of Exposure

Ecological exposure refers to the contact of an ecological receptor with an air toxic through direct or indirect exposure pathways. As with human health risk assessment, characterization of ecological exposure should initially evaluate (in the problem formulation phase) all exposure pathways that are potentially complete. Unlike human health exposure, ecological risk assessments will generally identify a limited number of specific metrics of exposure to actually quantify since it is not usually possible to evaluate all exposure pathways for all the species or other ecosystem attributes present in any given study area. Initially the assessors will generally consider all exposure pathways broadly, but then identify the assessment endpoints which will lead to a specific and narrowly defined set of exposure pathways to actually study in depth.

Ecological exposure pathways that are generally important for air toxics include all pathways where contaminants are taken up directly from environmental media (e.g., air, soil, sediment, and surface or rain water) for lower trophic level organisms (including plants) and ingestion of contaminated plant or animal food items for higher trophic level receptors. Pathways that may be important in specific cases include foliar and root uptake by plants, deposition and dermal exposure pathways, and ingestion via grooming, preening, and food consumption.

Once the specific set of exposure pathways to be studied are determined (and the matching assessment endpoints that are to be assessed are determined), characterization of ecological exposure is based initially on information derived from modeling and/or existing monitoring data. Later, additional modeling and/or site-specific empirical information may be obtained. The objective of the exposure characterization is to produce a summary exposure profile that identifies the exposed ecological entity, describes the course a stressor takes from the source to that entity (i.e., the exposure pathway), and describes the intensity and spatial and temporal extent of co-occurrence or contact (see Section 24.2.4.3). The exposure profile also describes the influence of variability and uncertainty on exposure estimates and reaches a conclusion about the likelihood that exposure will occur. Exhibit 24-1 provides a list of questions that can help define the specific information needed to characterize exposure.

Exposure characterization includes the following steps, each of which is discussed in a separate subsection below:[1]

- Quantifying releases of contaminants of potential ecological concern (COPEC);
- Estimating chemical fate and transport via modeling and/or monitoring;
- Quantifying exposure (e.g., exposure concentrations and dietary intakes);
- Evaluating uncertainty; and
- Preparing documentation.

Exhibit 24-1. Questions to Ask Concerning Source, Stressor, Exposure, and Ecosystem Characteristics

Source and Stressor Characteristics

- What is the nature of the source(s) (e.g., point vs. nonpoint vs. mobile sources)?
- What is the intensity of the stressor (e.g., the dose or concentration of a chemical)?
- What is the chemical form of the stressor and its lability as a function of local physical-chemical conditions?
- What is the mode of action? How does the stressor impact organisms or ecosystem functions?
- How does the stressor come into contact with a receptor (transport)?

Exposure Characteristics

- With what frequency does a stressor release occur (e.g., is it episodic or continuous; is it subject to daily, seasonal, or annual periodicity)?
- What is the duration of release and exposure? How long does the stressor persist in the environment (e.g., what is its half-life)?
- What is the timing of exposure? When does it occur in relation to critical organism life cycles or ecosystem events (e.g., reproduction, lake overturn)?
- What is the spatial scale of exposure? Is the extent or influence of the stressor local, regional, global, habitat-specific, or ecosystem-wide?
- What is the distribution? How does the stressor move through the environment (e.g., fate and transport)?

Ecosystems Potentially at Risk

- What are the geographic boundaries of the study area? How do they relate to functional characteristics of the ecosystem?
- What are the key abiotic factors influencing the ecosystem (e.g., climatic factors, geology, hydrology, soil type, water quality)?
- Where and how are functional characteristics driving the ecosystem (e.g., energy source and processing, nutrient cycling)?
- What are the structural characteristics of the ecosystem (e.g., species number and abundance, trophic relationships)?
- What habitat types are present?
- How do these characteristics influence the susceptibility (sensitivity and likelihood of exposure) of the ecosystem to the stressor(s)? For example, what portion of the receptor's home range is in the area of impact?
- Are there unique features that are particularly valued (e.g., the last representative of an ecosystem type)?
- What is the landscape context within which the ecosystem occurs?

Source: EPA Guidelines for Ecological Risk Assessment[1]

24.2.1 Quantifying Releases

The process used to quantify releases of air toxics for purposes of ecological risk assessment is identical to that for the human health analyses (see Chapter 7).

24.2.2 Estimating Chemical Fate and Transport

The process and methods used to estimate chemical fate and transport generally are similar to those used for multipathway human health risk assessments. Key differences and special considerations are highlighted in the subsections that follow.

24.2.2.1 Physical and Chemical Parameters

The same physical and chemical parameters identified in Chapter 17 affect the persistence of air toxics in the environment and their potential to accumulate in ecological food webs. Additional considerations are specific to ecological risk assessment.

- The bioconcentration factors (BCFs) and bioaccumulation factors (BAFs) used to characterize ecological exposure may be different than corresponding factors used for the human health exposure assessment. For example, wildlife may eat different species of fish/shellfish than humans; these may have different BCFs or BAFs. Also, whole-fish BCFs or BAFs are used for ecological exposure rather than those specific to the parts of the fish people normally eat (e.g., fillets).

- Chemical speciation (e.g., for metals such as mercury) may be an important determinant of exposure and bioavailablity.[(a)]

- Fate and transport analysis may need to examine a wider range of lower-trophic level organisms to assess impacts to the communities and ecosystems of interest as well as to develop exposure estimates for ecological food webs.

24.2.2.2 Multimedia Modeling

As with human health exposure assessment, some combination of multimedia modeling and monitoring is generally used for ecological exposure assessment. The appropriate mix of modeling and monitoring will depend on the level of assessment and the risk management goals.

Modeling is relatively easy and inexpensive to implement and can be used to evaluate not only risks from current levels of contamination, but also how risks might change over time (e.g., concentrations of persistent bioaccumulative hazardous air pollutant [PB-HAP] compounds in fish may slowly increase over time in the presence of a continuous release) or as a result of

---

[a]EPA's Science Policy Council is embarking on the development of an assessment framework for metals. The first step in the process is formulation of an Action Plan that will identify key scientific issues specific to metals and metal compounds that need to be addressed by the framework, potential approaches to consider for inclusion in the framework (including models and methods), an outline of the framework, and the necessary steps to complete the framework.

potential changes in land use (a change in land use might alter a number of habitat factors that influence the number and identity of ecological receptors). The modeling approach, however, has inherent uncertainties, which may lead to either over- or underestimates of exposure.

Model choices range from simple, screening-level procedures that require a minimum of data to more sophisticated methods that describe processes in more detail, but require a considerable amount of data. The same multimedia models used for the multipathway human health exposure assessment generally can be used for at least part of the ecological exposure assessment (e.g., the same models can be used to estimate concentrations in abiotic media at specific locations, whether for human health or ecological exposure assessment). However, choice of specific exposure points or areas may differ due to the focus on ecological receptors, as will the specific food webs being evaluated. Specific models may also be configured in ways that facilitate ecological exposure assessments. For example, TRIM (Total Risk Integrated Methodology) includes a fate, transport, and ecological exposure model (TRIM.FaTE) which simulates multimedia pollutant transfers and ecological receptor exposures in an ecosystem of interest (see Part III).[(2)] However, other approaches (e.g., Multiple Pathways of Exposure) are not specifically designed for ecological exposure assessment).

24.2.2.3 Multimedia Monitoring

The term monitoring in ecological risk assessment can also be more broadly used to mean collection of any type of empirical field data for the assessment (e.g., plant counts and spatial distribution in an assessment area). The use of monitoring in ecological risk assessment can serve a number of purposes. For example, if there is a need to reduce uncertainties in the predictive modeling approach, monitoring can be performed in various media and biota in the study area. As with human health exposure assessment, monitoring can be used to confirm or calibrate predictive modeling estimates of contaminant concentrations in media or biota.

For higher-tier risk assessments, monitoring for ecological exposures also may include site-specific toxicity or bioaccumulation studies, in which test organisms are exposed to the actual mixtures of contaminants from within the study area to develop site-specific and chemical-specific toxicological and/or bioaccumulation relationships (See Chapter 25). However, poorly designed sampling or toxicological evaluations of environmental media from the site may not allow a definitive identification of the cause of adverse response. For example, receptor abundance and diversity as demographic data reflect many factors (e.g., habitat suitability, availability of food, and predator-prey relationships). If these factors are not properly controlled in the experimental design of the study (e.g., through use of a comparison site or a gradient design that examines effects along a two-dimensional gradient downwind of sources), conclusions regarding chemical stressors can be confounded. In addition, monitoring may not provide sufficient information to develop estimates of potential risks should land use or exposure change in the future.

Monitoring techniques for ecological exposure characterization may differ from those used for multipathway human health exposure assessment. In particular, different species or components of the food web may be of concern. For example, large invertebrates such as dragonfly larvae often are a focus for ecological exposure assessments because they are important components of surface water ecosystems as well as key prey items for both aquatic (e.g., fish) and terrestrial (e.g., birds) predators.

Example Consideration in Monitoring: Soil Sampling for Ecological Risk Assessments

The depth over which surface soils are sampled should reflect the type of exposure expected in the study area, the type of receptors expected in the study area, the depth of biological activity, and the depth of potential contamination. For example, if exposures to epigeic (surface dwelling) earthworms are a concern, concentrations in the first few inches of soil are most relevant. On the other hand, if a burrowing mammal is of concern, concentrations at a depth of two or more feet may need to be estimated. Careful consideration of the size, shape, and orientation of sampling volume is important since they have an effect on the reported measured contaminant concentration values.[3] Selection of sampling design and methods can be accomplished by use of the Data Quality Objectives (DQO) process discussed in Chapter 7. Additional soil sampling guidance that may be consulted includes EPA's *Preparation of Soil Sampling Protocols: Sampling Techniques and Strategies* [4] and *Guidance for Data Usability in Risk Assessment.*[5]

24.2.3 Quantifying Exposure

Three elements are important components of quantifying exposure: the specific metrics of exposures that are to be used, the dimensions of exposure, and the exposure profile. Each is described in a separate subsection below. These estimates can be produced by some models such as TRIM.FaTE.[6]

24.2.3.1 Metrics of Exposure

Depending on the specific receptors and pathways of concern, ecological exposure is quantified generally in one of three ways.[1]

- Exposures to abiotic media may be evaluated using contaminant media concentrations as the **exposure concentrations** – that is, concentrations of air toxics in soil, sediment, and/or surface water at the exposure points. This is because the ecological toxicity reference levels (TRVs) used to characterize risk are based on laboratory studies that directly relate environmental concentrations in these media to adverse ecological impacts (e.g., a laboratory study that dissolves known concentrations of a chemical in water and measures adverse responses in the invertebrates or fish living in that water - the resulting concentration in water that shows no effect is then compared to modeled or monitored concentrations of the chemical in study area surface water).

- Exposures via the ingestion route of exposure may be evaluated using the **average daily dose (ADD)**, generally expressed as mg of chemical per kg of body weight per day (mg/kg-d). The general formula[b] for calculating ADD for ecological receptors is similar to that used for human health ingestion exposure:[1]

$$ADD_{pot} = \sum_{k=1}^{m} (C_k \times FR_k \times NIR_k) \qquad \text{(Equation 24-1)}$$

[b]The TRIM.FATE model[6] can output estimates of ingestion intake at user-designated time points in a dynamic simulation, and as an average over a user-designated period, as well as estimates for steady-state simulation.

where

ADD_{pot} = Potential average daily dose, expressed in units of mg/kg-day.

Chemical-related variable:

C_k = Average contaminant concentration in the kth type of food, expressed in units of mg/kg (wet weight)

Variables that describe the exposed ecological receptor population (also termed "wildlife exposure factors"):

FR_k = Fraction of intake of the kth food type that is from the contaminated area (unitless).
NIR_k = Normalized ingestion rate of the kth food type of a wet-weight basis, expressed in kg food/kg body-weight-day.
m = Number of contaminated food types

Exposure factors can be found in the EPA *Wildlife Exposure Factors Handbook*.[7]

Contaminant concentration (C_k) is commonly estimated with the use of multimedia models. In some situations (e.g., a higher tier of analysis), C_k in food has been measured directly at the point of contact where exposure occurs. An example is the use of food collected from the mouths of nestling birds to evaluate exposure to pesticides through contaminated food. Although such measurements can be difficult to obtain, they reduce the need for assumptions about the frequency and magnitude of contact.

- Exposures to some stressors are evaluated using **uptake**. Some stressors must be internally absorbed to exhibit adverse effects. For example, a contaminant that causes liver tumors in fish must be absorbed and reach the target organ to cause the effect. Uptake is evaluated by considering the amount of stressor internally absorbed by an organism and is a function of the following:
  - Chemical form of the contaminant (speciation);
  - Medium (sorptive properties or presence of solvents);
  - Biological membrane (e.g., integrity, permeability); and
  - Organism (e.g., sickness, active uptake).

Because of interactions among these factors, uptake will vary on a study-specific basis. Uptake is usually assessed by modifying an estimate of the exposure concentration indicating the **bioavailable fraction** (i.e., the proportion of the stressor that is available for uptake) actually absorbed (e.g., monomeric aluminum is generally bioavailable to certain aquatic receptors while polymeric aluminum generally is not). Absorption factors and bioavailability measured for the chemical, ecosystem, and organism of interest are preferred. Internal dose can also be evaluated using a physiologically-based pharmacokinetic (PBPK) model or by measuring biomarkers or residues in receptors.

When using a tiered approach, conservative assumptions generally are used at the screening level. Exhibit 24-2 presents examples of conservative assumptions; these are described in more detail in EPA's *Guidelines for Ecological Risk Assessment*.[1]

Exhibit 24-2. Examples of Conservative Assumptions for Ecological Exposure Estimation	
Exposure Factor	**Assumed Value**
Area-use factor (factor related to home range and population density)	100 percent (organism lives completely within area of highest exposure concentrations)
Bioavailability	100 percent
Life stage	most sensitive life stage
Body weight	minimum possible
Food ingestion rate	maximum possible
Dietary composition	100 percent of diet consists of the most contaminated dietary component
The use of conservative assumptions should be informed by study-specific information. For example, assuming 100 percent for area-use factor and diet would not be appropriate if study-specific information indicates otherwise (e.g., the receptor is only present in the assessment area part of the year). Similarly, use of the most sensitive life stage would only be appropriate if that life stage were reasonably expected to be exposed to the chemical.	

24.2.3.2 Dimensions of Exposure

Three dimensions are considered when quantifying exposure: intensity, time, and space.

- **Intensity**. Intensity is generally expressed as the amount of chemical contacted per day. Intensity may be affected by a number of factors, including the concentration of the chemical in various media and biota and chemical form (e.g., speciation), which may affect toxicity, bioavailabilty, and/or bioconcentration.

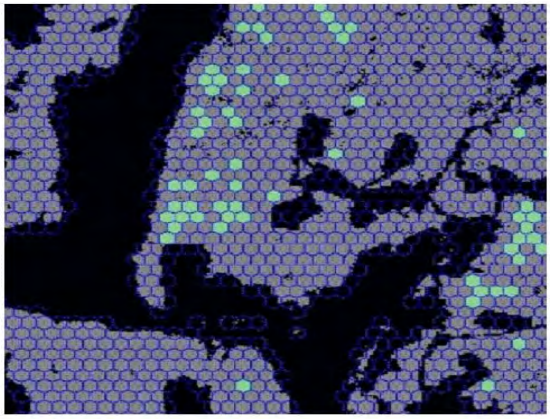
- **Time**. The temporal dimension has aspects of duration, frequency, and timing. For air toxics assessments, intensity and time may sometimes be combined by averaging intensity over time. Due to the emphasis on persistence and bioaccumulation, the focus of the ecological exposure characterization for air toxics is generally on chronic (long-term) exposures. In using predictive modeling to estimate exposure concentrations, an average annual concentration generally is sufficient, at least for screening-level analyses. An exception would include situations where the release and the presence of ecological receptors are both periodic (e.g., releases are much higher in the spring and summer, when ecological receptors are more abundant and active). If using predictive modeling to develop estimates of the average daily dose (ADD), the duration of time modeled generally should be sufficient for concentrations of air toxics in the media and biota of concern to reach equilibrium. If the models indicate that equilibrium is not reached, the duration of time modeled generally should be at least as long as the period of time over which releases are likely to occur (e.g., the design life of a specific facility). Timing is particularly important if the exposure coincides with a sensitive life stage of the receptor organism.

- **Space**. Space is important because ecological risk assessments generally focus at the population level or higher (e.g., community, ecosystem). Therefore, space is a measure of the total fraction of the population, community, or ecosystem that is potentially exposed – a factor that will impact the overall risk characterization. Space is generally expressed in terms of areas (e.g., hectares, acres, square meters) that exceed a particular chemical threshold level. However, another important spatial consideration is the **fraction** of the overall habitat type that is potentially affected. At larger spatial scales, the shape or arrangement of exposure may be an important issue, and area alone may not be the appropriate descriptor. Geographic Information Systems (GIS) have greatly expanded the options for analyzing and presenting the spatial dimension of exposure (see Part VII of this reference manual for more information about GIS). Several recent papers discuss ways to incorporate spatial considerations in ecological risk assessments.[8]

Sometimes, temporal and spacial considerations must both be considered together. For example, in the case of acidic deposition, the andromous fish species in Maryland and other middle-Atlantic states have a special risk scenario. Specifically, their spawning run occurs at the same time when the weather pattern changes in the late winter and early spring from a coastal to a continental pattern. This increases acidic deposition to the headwaters where the spawning occurs and the eggs and hatchlings are at the most vulnerable part of their life cycle.

Using Spatial Information in Ecological Exposure Assessment

Many terrestrial organisms that might be evaluated in an ecological risk assessment are mobile. Where these populations spend their time depends on the locations of habitats necessary to provide food, breeding sites, and protection from predators. Behaviors such as migration also affect locations of receptor populations. Screening-level assessments usually assume that the ecological receptors of interest reside at the locations of the highest exposures modeled. In subsequent tiers of analysis, the assessor may spatially refine the exposure estimate by considering the habitat use and foraging areas of the receptor(s) of interest. GIS land cover and land use information can be used to estimate where an ecological receptor is likely to reside or breed. For example, EPA's Western Ecology Division of the National Health and Environmental Effects Laboratory developed a model called Program to Assist in Tracking Critical Habitat (PATCH), which can be used to generate "patch-by-patch" descriptions of landscapes, assessments of the number, quality, and spatial orientation of breeding sites, and map-based estimates of the occupancy rate. In the example output shown here, the medium grey areas denote significant/acceptable habitat and the lighter gray (or light green) areas denote areas suitable for breeding. This information can be used to identify where the ecological receptors are likely to reside or breed, and the modeled exposure concentrations at those locations can be used in the risk characterization calculations. The PATCH software and user's guides are available at: http://www.epa.gov/wed/pages/models/patch/patchmain.htm.



Example PATCH Output.

24.2.3.3 Exposure Profile

Questions Addressed by the Exposure Profile

- How may exposure occur?
- What may be exposed?
- How much exposure may occur?
- When and where may exposure occur?
- How may exposure vary?
- How uncertain are the exposure estimates?
- What is the likelihood that exposure will occur?

The final product of the ecological exposure assessment is an exposure profile. Exposure is generally described in terms of intensity, space, and time, and in units that can be combined with the ecological effects assessment (see Chapter 25). The exposure profile identifies the receptor and describes each exposure pathway as well as the intensity, spatial extent, and temporal extent of exposure. The exposure profile also describes the impact of variability and uncertainty on exposure estimates and reaches a conclusion about the likelihood that exposure will occur. Depending on the risk assessment, the exposure profile may be a written stand alone document or a module of a larger document. In either case, the objective is to ensure that the information needed for risk characterization has been collected, evaluated, and presented in a clear, concise, and transparent way. The exposure profile also provides an opportunity to verify that all of the important exposure pathways identified in the conceptual model (i.e., those that support an evaluation of the assessment endpoints) were evaluated.

24.2.3.4 Evaluating Variability and Uncertainty

The exposure profile described in the previous section should aid understanding of how exposure can vary depending on receptor attributes (exposure factors) or stressor levels. Variability can be described qualitatively, by using a distribution or by describing where a point estimate is likely to fall on a distribution. EPA policy recommends the use of both central tendency and high-end exposure estimates.[9]

The exposure profile also should summarize important uncertainties (e.g., lack of knowledge), including:

- Identification of key assumptions and how they were addressed;
- Discussion (and quantification, if possible) of the magnitude of modeling, sampling, and/or measurement error;
- Identification of the most sensitive variables influencing the exposure estimate; and
- Identification of which uncertainties can be reduced through additional data collection, modeling, or analysis (e.g., in a subsequent tier of analysis).

Professional judgment often is needed to determine the uncertainty associated with information taken from the literature and any extrapolations used in developing a parameter to estimate exposures. All assumptions used to estimate exposures should be stated, including some description of the degree of bias possible in each. Where literature values are used, an indication of the range of values that could be considered appropriate also should be indicated. The uncertainty and variability associated with ecological effects criteria must also be taken into

consideration. A more thorough description of how to deal with variability and uncertainty in the risk assessment process is provided in Chapter 31.

References

1. U.S. Environmental Protection Agency. 1998. *Guidelines for Ecological Risk Assessment*. Risk Assessment Forum, Washington, D.C., April 1998. EPA/630/R095/002F. Available at: http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=12460.

2. U. S. Environmental Protection Agency. *Total Risk Integrated Methodology* (TRIM). Documentation is available at: http://www.epa.gov/ttn/FERA/urban/trim/trimpg.html.

3. U.S. Environmental Protection Agency. 2000. *Draft Ecological Soil Screening Level Guidance*. Office of Emergency and Remedial Response, Washington, D.C., July 10, 2000.

4. U.S. Environmental Protection Agency. 1992. *Guidance for Data Useability in Risk Assessment (Part A)*. Office of Emergency and Remedial Response Publication 9285.7-09A, PB92-963356, Washington, D.C., April 1992.

5. U.S. Environmental Protection Agency. 1992. *Preparation of Soil Sampling Protocols: Sampling Techniques and Strategies*. Office of Research and Development, Washington, D.C. EPA/600/R92/128.

6. U. S. Environmental Protection Agency. 2002. *Total Risk Integrated Methodology. TRIM.FaTE Technical Support Document. Volume 1: Description of Module*. EPA-453/R-02-011a; *Volume 2: Description of Chemical Transport and Transformation Algorithms*. EPA/453/R-02/011b. *Evaluation of TRIM.FaTE. Volume 1: Approach and Initial Findings*. EPA/453/R-02/012; *TRIM.FaTE User's Guide*. Office of Air Quality Planning and Standards, Research Triangle Park, NC. These documents and information are available at: http://www.epa.gov/ttn/fera/trim_fate.html#current_user.

7. U.S. Environmental Protection Agency. 1993. *Wildlife Exposure Factors Handbook*. Office of Research and Development, Washington, D.C. EPA/600/R93/187. Available at: http://cfpub.epa.gov/ncea/cfm/wefh.cfm?ActType=default.

8. Freshman, J.S., and Menzie, C.A. 1996. Two wildlife exposure models to assess impacts at the individual and population levels and the efficacy of remedial actions. *Human and Ecological Risk Assessment* 2:481-498.

   Hope, B.K. 2001. A case study comparing static and spatially-explicit ecological exposure analysis methods. *Risk Analysis* 21:1001-1010.

   Linkov, I., Burmistrov, D., Cura, J., and Bridges, T.S. 2002. Risk-based management of contaminated sediments: Consideration of spatial and temporal patterns in exposure modeling. *Environmental Science and Technology* 36:238-246.

9. U.S. Environmental Protection Agency. 1992. *Guidance on Risk Characterization for Risk Managers and Risk Assessors*. National Center for Environmental Assessment. Risk Assessment Council, Washington, D.C. February 1992.

Chapter 25 Analysis: Characterization of Ecological Effects

Table of Contents

25.1 Introduction 1

25.2 Ecological Response Analysis 1
25.2.1 Stressor-Response Analysis 1
25.2.1.1 Ecological Effect Levels 1
25.2.1.2 Selection of TRVs for a Particular Assessment 7
25.2.1.3 Stressor-Response Curves 11
25.2.1.4 Species Sensitivity Distribution 11
25.2.2 Linking Measures of Effects to Assessment Endpoints 12

25.3 Stressor-Response Profile 16

25.4 Evaluating Variability and Uncertainty 16

References 16

25.1 Introduction

As noted in the previous chapter, the analysis step of ecological risk assessment includes characterization of exposures and characterization of ecological effects. Chapter 24 described the approaches and methods used for exposure characterization. This chapter describes the approaches and measures used for characterization of ecological effects. The discussion in this chapter is based largely on EPA's *Ecological Risk Assessment Guidelines*.[1] Readers are referred to that document for a more complete discussion of available approaches and methods.

The methodology used to characterize ecological effects is generally similar to that used for human health toxicity assessment. One of the distinctive features of ecological effects characterization relates to the more general management goal of protecting a receptor population or community rather than a single individual. This has led to the development of water, sediment, and soil quality criteria that are designed to protect the communities of organisms that inhabit surface waters and soils. It also provides the option of using a distribution or range of values to characterize chemical toxicity (an option not generally available in human health risk assessment).

Characterization of ecological effects involves describing the potential effects resulting from exposure to a stressor, linking these effect to the assessment endpoints identified during problem formulation, and evaluating the **stressor-response relationship** (i.e., how the effects will change with varying stressor levels). The characterization begins by evaluating effects information to specify the resulting effects, verifying that these effects are consistent with the assessment endpoints, and confirming that the conditions under which the effects occur are consistent with the conceptual model. Once this has been done, the effects characterization involves two additional steps: (1) performing an ecological response analysis, and (2) developing a **stressor-response profile** which also contains an analysis of uncertainty and variability. Each of these additional steps is discussed in a separate section below.

25.2 Ecological Response Analysis

Ecological response analysis examines three primary elements: identifying stressor-response relationships, establishing causality, and determining the linkages between measurable ecological effects and assessment endpoints. Each is described in a separate subsection below.

25.2.1 Stressor-Response Analysis

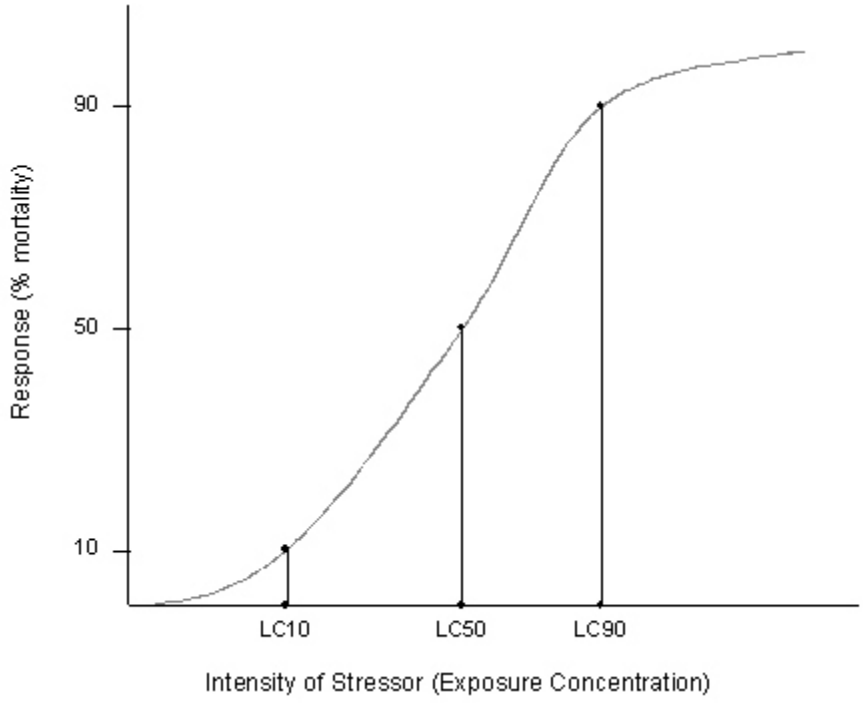
Stressor-response analysis for ecological effects is functionally similar to dose-response analysis for human health effects (e.g., see Chapter 12). The specific stressor-response relationship(s) used in a given risk assessment depend on the scope and nature of the assessment as defined in the problem formulation and reflected in the analysis plan. Three types of stressor-response relationships are commonly used: point estimates, stressor-response curves, and cumulative distribution functions. Each of these is discussed in a separate subsection below.

25.2.1.1 Ecological Effect Levels

Ecological effect levels are point estimates of an exposure associated with a given effect (e.g., a concentration that results in 50 percent mortality in the exposed population, or LC_{50}) used to

compare with an environmental exposure concentration. Data on the toxicity of a chemical is usually obtained from laboratory studies in which groups of organisms (e.g., invertebrates, benthic organisms, plants, earthworms, laboratory mammals, fish) are exposed to varying levels of the chemical, and one or more responses (endpoints such as survival, growth, reproduction) are measured. Various statistical methods are used to establish thresholds for adverse ecological effects associated with acute or chronic exposures. Risk assessors often choose no-effect or low-effect levels as screening values. Stressor-response relationships may be relatively simple (as illustrated in Exhibit 25-1) or may be very complex.

Exhibit 25-1. Hypothetical Simple Stressor-Response Relationship



Hypothetical relationship between intensity of stressor (in this example, concentration of a chemical in water) and ecological response (in this example, percent mortality of an exposed population of minnows). Different points on the curve represent, respectively, the concentration resulting in 10 percent mortality (LC10), 50 percent mortality (LC50), and 90 percent mortality (LC90).

Several specific point estimates are commonly used to characterize ecological effects (Exhibit 25-2):

- **Median effect concentrations or doses** are those levels that result in effects that occur in 50 percent of the test organisms exposed to a stressor. The median effect level is always associated with a time parameter (e.g., 24 hours, 48 hours). Because the tests used to derive median effects levels seldom exceed 96 hours, these values are used primarily to assess acute (short-term) exposures.

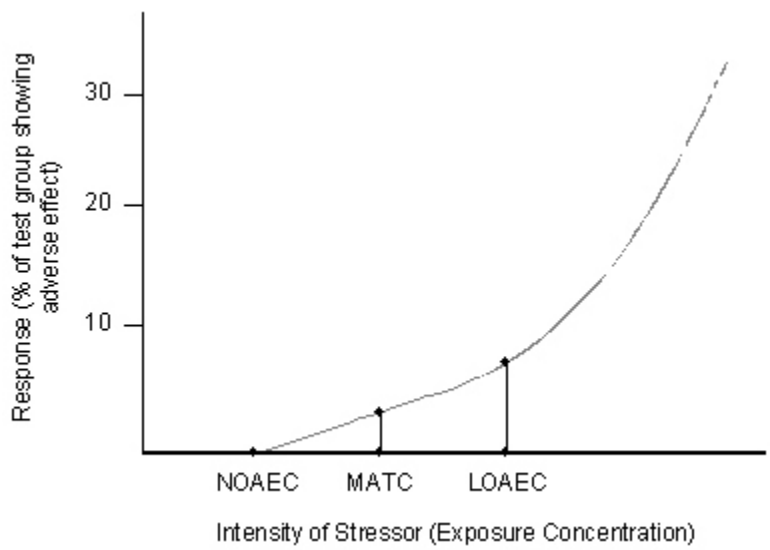
Exhibit 25-2. Commonly Used Point Estimates

Median effect concentrations or doses (acute exposures)

LC_{50} Concentration (food or water) resulting in mortality in 50 percent of the exposed organisms
LD_{50} Dose (usually in dietary studies) resulting in mortality in 50 percent of the exposed organisms
EC_{50} Concentration resulting in a non-lethal effect (e.g., growth, reproduction) in 50 percent of the exposed organisms
ED_{50} Dose resulting in a non-lethal effect (e.g., growth, reproduction) in 50 percent of the exposed organisms

Low- or no-effect concentrations or doses (chronic exposures)

NOAEL no-observed-adverse-effect-level, the highest dose for which adverse effects are not statistically different from controls
LOAEL lowest-observed-adverse-effect level, the lowest dose at which adverse effects are statistically different from controls
NOEC no-observed-effect-concentration, the highest ambient concentration for which adverse effects are not statistically different from controls
LOEC lowest-observed-effect concentration, the lowest ambient concentration at which adverse effects are statistically different from controls
MATC maximum acceptable toxicant concentration, the range of concentrations between the LOEC and NOEC
GMATC geometric mean of the MATC, the geometric mean of the LOEC and NOEC



- **Low- or no-effect concentrations or doses** are derived from experimental data using statistical estimates. The no-effect level is determined by experimental conditions as well as the variability inherent in the experimental data. Thus, depending on experimental conditions (e.g., the range of concentrations tested), two separate tests using the same chemical and the same organism could result in different no-effect levels. Low- or no-effect levels are used primarily to assess chronic (longer-term) exposures.

A variety of different types of studies can be used to develop ecological stressor-response relationships, including field studies, laboratory studies, and microcosm studies (Exhibit 25-3).

For air toxics, stress-response analysis can include both primary and secondary effects.

Point Estimates, TRVs, and Benchmarks

The terms **Toxicity Reference Values (TRVs)** and **Ecological Benchmarks** are used to describe those **Point Estimates** identified or derived for use in ecological risk assessments. These particular point estimates may be derived from a single study (e.g., an NOEC or EC_{50}) or from the integration of multiple studies (e.g., water quality criteria). When TRVs or benchmarks are drawn from a single study, they are usually set in consideration of multiple studies (e.g., from the study most relevant to the purposes and specifics of the assessment has been selected, or the most sensitive result among the relevant studies)

- **Primary effects** (e.g., lethality, reduced growth, neurological/behavioral deficits, impaired reproduction) result from exposure of aquatic and terrestrial organisms to air toxics. An example of a chronic effect would be reduced reproduction in a fish species exposed to air toxics in a surface water body or in a terrestrial bird eating contaminated fish from a small pond. An extreme example of an acute primary effect might be deaths of birds caused by inhalation of a particular toxin. Toxic effects on survival, growth, development, and reproduction might have population-level consequences for a species (e.g., result in local population extinction over time) and are widely accepted as endpoints for characterizing ecological risks. In recent years, more subtle effects have been investigated, including those pertaining to clinical signs of poisoning, immunotoxicity, and even behavioral changes that might influence survival, growth, development, or reproduction.

- **Secondary effects** (e.g., loss of prey species in the community) result from the action of air toxics on supporting components of the ecosystem. These secondary effects occur through biological interaction of one or more species' populations with individuals or populations that have been primarily affected. For example, exposure to an air toxic may adversely affect one or more species of microscopic algae, bacteria, or fungus, which can adversely affect an ecosystem's nutrient cycling and primary production. This can lead to an alteration in the abundance, distribution, and age structure of a species or population dependent on these microscopic organisms, which can then lead to changes in competition and food web interactions in other species. These ecosystem effects can be propagated to still other populations, affecting their presence or representation within the ecosystem. A relatively simple example of secondary effects involves the aerial application of pesticides that dramatically reduced the population of an aquatic insect. This impact to the insect population indirectly affects wild ducklings in the ecosystem, which depend on the insects as a food supply.[(2)] Although it often is possible to identify the potential for secondary effects, developing stressor-response functions for secondary effects (e.g., in a manner analogous to that illustrated in Exhibit 25-2) is not an easy task. A recent paper provides one example of the evaluation of secondary effects in ecological risk assessment.[(3)]

The use of the point estimate approach has some potential limitations. The most important is that the point estimate established by a given study depends on both the range of doses tested and the statistical power of the study (e.g., the ability to detect an effect if it occurs). For example, studies with low power (e.g., those with only a few test animals per dose group) tend to yield NOAEL or NOEC values that are higher than studies with good power (those with many animals

per dose group). In addition, the choice of some point estimates (e.g., NOEC and LOEC) is restricted to concentrations that were tested, which may or may not be close to the environmentally relevant concentrations, and this uncertainty increases as the interval between doses increases. Finally, it is not always easy to interpret the significance of an exposure that exceeds some particular point estimate, since the severity and incidence of response depends on the shape and slope of the exposure response curve (information that is not captured in a point estimate).

Exhibit 25-3. Types of Ecological Stressor-Response Studies
• **Laboratory Studies**. Most information on ecological stressor-response comes from laboratory ecotoxicology studies using a generic set of species to represent different components of terrestrial or aquatic ecosystems. For example, the freshwater crustacean *Daphnia*, is often used as a surrogate for all small invertebrates that inhabit surface waters, and various species of minnows are used as surrogates for fish. Laboratory studies are relatively easy and inexpensive to conduct, and effects can be directly linked to exposure to a single air toxic. There is uncertainty, however, in extrapolating the results from standard laboratory species to the wide array of species in the environment or from the controlled laboratory conditions to the complex conditions that occur in nature. Additionally, in most cases, laboratory studies are not designed to assess effects on populations, communities, and ecosystems. • **Field Studies**. Studies of wildlife, populations, communities, and ecosystems exposed to air toxics in natural settings can provide valuable information on stressor-response effects. Field data can be valuable in demonstrating the presence or absence of a cause-effect relationship that can provide a basis for prioritization or for recognizing the efficacy of a risk reduction action. These studies also can be used to assess stressor-response relationships for the site-specific mixtures of concern. However, the study organisms may be exposed to numerous types of stressors (chemical and non-chemical), and the effects of individual air toxics (and sometimes site-specific mixtures) may be difficult to isolate. In addition, field studies are conducted infrequently due to the significant time and resources required. Comparison of the study area to a control area is necessary to evaluate the potential impact of the chemical release. • **Microcosm Studies**. Microcosm studies use assemblages of several different taxa and environmental media in an enclosed experimental system as a surrogate for natural ecosystems. Such studies can control for some of the uncertainty associated with multiple stressor exposure in field studies. These studies also may provide information about food web dynamics and the interactions of populations or organisms. As with field studies, microcosm studies are time and resource intensive and, therefore, may be relatively uncommon for air toxic studies.

A variety of point estimates are used in ecological risk assessments. Some are developed from acute (short-term) exposures; others are developed from chronic (long-term) exposures. Three general types of point estimates are available for use in ecological risk assessments:

- **Community-level criteria**. EPA has developed ambient water quality criteria (AWQC) and sediment quality criteria for the protection of aquatic communities. These values are based on consideration of a cumulative distribution function (see Section 25.2.1.4). For example, AWQC are designed to protect 95 percent of all aquatic species in freshwater or marine environments. Criteria have been developed for both acute and chronic exposures, although for a limited number of chemicals.

- **Effect levels from laboratory toxicity tests**. A variety of aquatic species are routinely used in ecological toxicity tests, including fathead minnows (a small fish species) and *Daphnia* (a tiny freshwater crustacean). Effects of concern can include acute effects such as mortality (e.g., LD_{50}) as well as chronic effects such as reproduction. Toxicity tests also are available for terrestrial organisms (e.g., earthworms) and occasionally involve vertebrate species of wildlife (e.g., the effects of polychlorinated biphenyls (PCBs) have been studied extensively in mink).

- **Effect levels from field bioassays**. In some cases, ecological effects are evaluated directly by exposing test organisms to ambient conditions. This most often is done where complex mixtures of chemicals are present (e.g., in soils or sediments).

The point estimates employed in ecological risk assessments may be generally termed toxicity reference values (TRVs).[(a)] They may be values taken from individual toxicity studies (e.g., NOECs or EC_{50}s)or the result of integration of multiple studies (e.g., water quality criteria). TRVs may be developed for site-specific ecological receptors, depending on the importance of those receptors to the local ecosystem, or for an endpoint not previously evaluated. For example, while some TRVs may be based on survival, growth, and reproductive success of a population, TRVs protective of a threatened or endangered species, a valuable game species (e.g., trout), or an ecologically key species (e.g., wolf) might be based on an endpoint that is relevant to individual organism health (e.g., a neurological deficit) rather than to population maintenance. On the other hand, TRVs based on higher effect levels (e.g., 20 to 50 percent or higher of the population is affected) might be appropriate for species for which great functional redundancy exists in the ecosystem (e.g., different herbaceous plants).[(4)]

Derivation of TRVs for pathways involving wildlife ingestion would require information on food ingestion rates for sensitive and highly exposed animal species and information on the degree of bioaccumulation in appropriate trophic components. Examples of these derivations for aquatic systems can be found in the Great Lakes Water Quality Initiative (GLWQI) for mercury, dichlorodiphenyltrichloroethane (DDT), PCBs, and dioxin (2,3,7,8-TCDD)[(5)] and for terrestrial systems in the EPA methods of assessing exposures to combustor emissions.[(6)] EPA's *Wildlife Exposure Factors Handbook* [(7)] also provides data, references, and guidance for conducting exposure assessments for wildlife species exposed to toxic chemicals in their environment.

EPA and other organizations have developed a number of types of TRVs based on data for a chemical's toxicity to freshwater or saltwater organisms (see Exhibit 25-4). Toxicity data for longer term or chronic exposures generally will be more useful for an air toxics risk assessment; however, short term or acute toxicity data may be used for chemicals that lack or have incomplete chronic data. EPA has in the past used acute values in conjunction with conversion factors (i.e., acute-to-chronic ratios) to estimate chronic toxicity values, specifically for the derivation of chronic Ambient Water Quality Criteria and Great Lakes Water Quality Initiative criteria for aquatic life.

---

[a]Note that some ecological risk assessment guidance refers to the point estimates of ecological effects selected for a given assessment as Toxicity Reference Values (TRVs), while others use the term ecological benchmarks.

25.2.1.2 Selection of TRVs for a Particular Assessment

In reviewing toxicity studies for potential use in identifying or developing specific TRVs to use in a given assessment, the following questions should be considered:

- What taxa are used in the study?
- Did the study present any significant methodological difficulties?
- Did the study identify a LOAEL?
- Were the adverse effects seen possibly related to growth and survival, or reproduction and development?
- Did the study identify a NOAEL?
- Was the study duration appropriate to assess potential effects of chronic exposure?

If the test species are not within the taxonomic group of the ecological receptors of concern, the study may need to be rejected because the test species are too distantly related to assume similar physiological responses to a toxic agent.

Many studies may be of limited use in selection of TRVs. Potential deficiencies include:

- No control group was analyzed, or there was a high incidence of effects in the control group (applies to laboratory studies);
- No reference area was analyzed, or there was a high incidence of effects in the reference area (applies to field studies);
- No statistical analysis of results was conducted;
- In the case of fish/shellfish, body burdens were estimated, not measured;
- In the case of fish/shellfish, only fillet, carcass (guts, gills, and scales removed), or other body part concentrations were measured, not the whole body;
- In the case of wildlife, insufficient data were provided to calculate the dose to the animal; and
- Multiple contaminants were present in the experimental studies.

Most environmental contamination concerns for air toxics that persist and bioaccumulate will tend to be long-term and relatively low-level. As such, the most appropriate toxicity studies are those evaluating chronic (long-term) toxicity or, if chronic studies are not available, subchronic (medium-term) exposure durations. Although no one definition of "chronic" is accepted by human or ecological toxicologists, the general concept is that the duration encompasses a significant portion of the species life span (e.g., ten weeks for birds and one year for mammals). "Subchronic" is commonly defined as a 90-day or longer study for mammals and 10 weeks or fewer for birds. For aquatic bioassays, chronic tests may span multiple generations and assess sensitive growth or reproductive endpoints. In mammalian and avian tests, the term average daily dietary dose (e.g., expressed as mg/kg-day) generally implies chronic or subchronic exposure.[8]

In order to develop TRVs (sometimes termed benchmarks) for avian and mammalian receptors, Oak Ridge National Laboratory's Toxicological Benchmarks for Wildlife,[11] and some information from EPA's Integrated Risk Information System[9] can be used (in a more limited fashion). Information provided in these sources has to be modified using allometric information available in EPA's *Wildlife Exposure Factors Handbook*[7] to better represent potential wildlife species sensitivity.

Exhibit 25-4. Sources of Ecological TRVs or Benchmarks		
Data Source	**Available Toxicity Reference Value(s)**	**Overview of Data Source and Values**
EPA Office of Water Ambient Water Quality Criteria (AWQC)	• AWQC Chronic Criteria • AWQC Acute Criteria Note: many state water quality standards are based on AWQC	EPA has developed national recommended water quality criteria for the protection of aquatic life for approximately 150 pollutants. These criteria are published pursuant to Section 304(a) of the Clean Water Act (CWA) and provide guidance for States and Tribes to use in adopting water quality standards under Section 303(c) of the CWA. Source: http://www.epa.gov/waterscience/criteria/aqlife.html
Great Lakes Water Quality Initiative (GLWQI) Criteria Documents	• GLWQI Tier I Criteria • Final Chronic Values (FCVs)	GLWQI Tier I criteria and final chronic values (FCVs) are calculated under the same guidelines as the Sediment Quality Criteria (SQC). Draft GLWQI criteria documents were released for public review and were revised as necessary before they were published as "final." • Tier I Criteria are designed to be protective of aquatic communities • FCVs are designed to measure chronic toxicity to aquatic organisms Source: *Final Water Quality Guidance for the Great Lakes System.* Federal Register, Mar. 23, 1995, vol. 60, no. 56, p. 15365-15424
EPA Soil Screening Levels	• Soil screening levels	EPA has developed a methodology and initial soil screening levels protective of ecological receptors. Source: U.S. Environmental Protection Agency. 2000. *Ecological Soil Screening Guidance (Draft).* Office of Emergency and Remedial Response, Washington, D.C., July 2000. http://www.epa.gov/superfund/programs/risk/ecorisk/ecossl.htm.
EPA Region 4 Soil Screening Levels	• Soil screening levels	Source: U.S. Environmental Protection Agency. 1995. Supplemental Guidance to RAGS: Region 4 Bulletins No. 2. Ecological Risk Assessment. Region IV, Waste Management Division. http://www.epa.gov/region04/waste/ots/ecolbul.htm

Exhibit 25-4. Sources of Ecological TRVs or Benchmarks		
Data Source	**Available Toxicity Reference Value(s)**	**Overview of Data Source and Values**
Ecotox Thresholds ECO Update and EPA's Hazardous Waste Identification Rule (HWIR) documents	• GLWQI Tier II Criteria • Secondary Chronic Values (SCVs)	The GLWQI Tier II criteria and SCVs have received some peer review prior to publication, and 12 of them are included in the HWIR, which underwent public comment before promulgation. The GLWQI Tier II methodology calculates SCVs in a similar way to FCVs, but uses statistically derived "adjustment factors" and has less rigorous data requirements. • Tier II Criteria are designed to be protective of aquatic communities • SCVs are designed to measure chronic toxicity to aquatic organisms Source: *Ecotox Thresholds ECO Update* (volume 3, No. 2, January 1996, EPA/540/F-95/038).
ECOTOXicology database (ECOTOX)	• Point Estimates from Chronic Tests (e.g., EC_{50}, EC_{10}, LC_{50}, or GMATC) • Point Estimates from Acute Tests (e.g., LC_{50})	ECOTOX is a source for locating single chemical toxicity data for aquatic life, terrestrial plants, and wildlife. ECOTOX was created and is maintained by EPA's Office of Research and Development and the National Health and Environmental Effects Research Laboratory's Mid-Continent Ecology Division. ECOTOX is a source for locating single chemical toxicity data from three EPA ecological effects databases: AQUIRE, TERRETOX, and PHYTOTOX. AQUIRE and TERRETOX contain information on lethal, sublethal, and residue effects. AQUIRE includes toxic effects data on all aquatic species including plants and animals and freshwater and saltwater species. TERRETOX is the terrestrial animal database. It primarily focuses on wildlife species but the database does include information on domestic species. PHYTOTOX is a terrestrial plant database that includes lethal and sublethal toxic effects data. Source: http://www.epa.gov/ecotox.
Sediment Quality Criteria	• Varies	EPA and other agencies have developed sediment quality criteria for the protection of benthic communities. These criteria are highly specific to regions and bodies of water in the U.S. Regional experts are the recommended source for appropriate site-specific criteria.

Exhibit 25-4. Sources of Ecological TRVs or Benchmarks		
Data Source	**Available Toxicity Reference Value(s)**	**Overview of Data Source and Values**
Ecological Structure Activity Relationships (ECOSAR)	• Estimated Chronic GMATC • Estimated Acute Data (LC_{50} or EC_{50})	ECOSAR is a computer program that uses structure-activity relationships (based on available data) to predict the acute and chronic toxicity of organic chemicals to aquatic organisms. ECOSAR provides quantitative estimates of chronic values (e.g., GMATC), acute LC_{50} values, and acute EC_{50} values for industrial chemicals for several aquatic species (e.g., fish, daphnia, green algae, mysids). When the estimated aquatic toxicity value exceeds the water solubility of the compound, the estimated value is flagged; this situation generally is interpreted to mean that the chemical has no toxic effects in a saturated solution. Source: http://www.epa.gov/oppt/newchems/21ecosar.htm
Exposure-Related Effects Database (ERED)	Tissue-based effects values for fish and benthic invertebrates	The U.S. Army Corps of Engineers Exposure-Related Effects Database (ERED) lists toxicity information for a large number and wide taxonomic range of fish and shellfish. ERED is constantly being updated. Source: http://www.wes.army.mil/el/ered/
Jarvinen and Ankley database	Fish and shellfish exposure and effects information	The authors assembled a database of fish and shellfish exposure and effect information. Source: Jarvinen and Ankley (1999)[9]
Oak Ridge National Laboratory (ORNL) Soil Invertebrate toxicity database	Acute and chronic TRVs for soil invertebrates and microbial processes	This report focuses on chemicals found at U.S. Department of Energy (DOE) sites; however there are overlaps with air toxics (metals and organics). Source: Efroymson et al. (1997);[8] http://www.esd.ornl.gov/programs/ecorisk/documents/tm126r21.pdf
ORNL Plant toxicity database	Acute and chronic TRVs for terrestrial plants	This report presents a standard method for deriving TRVs, a set of data concerning effects of chemicals in soil or soil solution on plants, and a set of phytotoxicity TRVs for 38 chemicals potentially associated with DOE sites. Source: Efroymson et al. (1997)[8]
ORNL Wildlife toxicity database	Wildlife NOAEL and LOAELs	This report presents both NOAEL- and LOAEL-based TRVs for assessment of effects of 85 chemicals on 9 representative mammalian wildlife species and 11 avian wildlife species. Source: Sample et al. (1996)[10]

25.2.1.3 Stressor-Response Curves

One way to resolve some of the limitations in the TRV approach is to fit a mathematical equation to the available exposure-response data and describe the entire **stressor-response curve**. Data from individual experiments may be used to develop curves and point estimates both with and without associated uncertainty estimates. The advantages of curve-fitting approaches include using all of the available experimental data, the ability to interpolate to values other than the data points measured, and an improved ability to extrapolate to values outside the range of experimental data (e.g., for a low- or no-effect level). Curve-fitting often is used to extrapolate from observed effects levels to develop estimates of NOAELs, NOECs, and/or GMATCs. Stressor-response curves can be developed using any convenient data fitting software, but EPA has developed a software package specifically designed for this type of effort. This software is referred to as the Benchmark Dose Software (BMDS). More information on this software can be found on the National Center for Environmental Assessment's webpage.[11] A disadvantage of curve fitting is that the number of data points required may not always be available (e.g., especially for toxicity tests with wildlife species)

25.2.1.4 Species Sensitivity Distribution

In some cases, risk management decisions may also consider community-level effects as well as population-level or sub-population effects (one example is the Ambient Water Quality Criteria for the protection of aquatic life discussed in Section 25.2.1.1). That is, a stressor might be considered to be below a level of concern for the sustainability of a community if only a small fraction of the total number of exposed species are affected. In this case, toxicological responses may be best characterized by the distribution of toxicity values across species. This is called a **Species Sensitivity Distribution (SSD)**. The SSD approach is generally used for communities of aquatic receptors, since all of the different species that make up the community (e.g., all fish, benthic invertebrates, aquatic plants, and amphibians that reside in a stream) will be exposed to approximately the same concentration of contaminant in the water.

The process for generating an SSD consists of the following steps:

(1) Select an appropriate type of endpoint (e.g., lethality, growth, reproduction), and select an appropriate type of point estimate from the exposure-response curve for each species. For example, the TRV might be the LC_{50} for lethality or the EC_{20} for growth. The key requirement is that the SSD be composed of TRVs that are all of the same type, not a mixture.

(2) Collect all reliable values for that type of TRV from the literature for as many relevant species as possible. When more than one value is available for a particular species, either select the value that is judged to be of highest quality and/or highest relevance, or combine the values across studies to derive a single composite value for each species. It is important to have only one value per species to maintain equal weighting across species.

(3) Characterize the distribution of values across species with an appropriate SSD. Note that there is no *a priori* reason to expect that an SSD will be well characterized by a parametric distribution, so both parametric and empirical distributions should be considered.

Once an SSD has been developed, the fraction of species in the exposed community that may be affected at some specified concentration may be determined either from the empirical distribution or from the fitted distribution. These distributions can help identify stressor levels that affect a minority or majority of species.

A limiting factor in the use of SSDs is the amount of data needed as inputs. SSDs also can be derived from models that use Monte Carlo or other methods to generate distributions based on measured or estimated variation in input parameters for the models.

25.2.2 Linking Measures of Effects to Assessment Endpoints

As noted in Chapter 23, assessment endpoints express the environmental values of concern for the risk assessment; however they cannot always be measured directly. For example, the assessment endpoint may be maintaining a healthy population of trout in a lake, but measures of effect (e.g., toxicity tests) were conducted on different species (e.g., fathead minnows). Where there is a lack of time, monetary resources, or practical means to acquire more data, extrapolations may be the only way to bridge the gap in available data. Two general approaches are used for such extrapolations:

Examples of Extrapolations

- Between taxa (e.g., minnow to rainbow trout)
- Between responses (e.g., mortality to growth or reproduction)
- From laboratory to field
- Between geographic areas
- Between spatial scales
- From data collected over a short time frame to longer-term effects

- **Empirical extrapolations or process models**. Empirical extrapolations use experimental or observational data; process-based approaches rely on some level of understanding of the underlying operations of the system of interest.

- **Professional judgment**. This is not as desirable as empirical or process-based approaches, but it is the only option when data are lacking. However, professional judgment can be credible, provided it has a sound scientific basis.

One of the most common types of extrapolations is that of effects observed in the laboratory (e.g., toxicity tests) to those observed in the field. Exhibit 25-5 highlights the general questions to consider when performing such an extrapolation.

When conducting field sampling or other monitoring studies, it sometimes is difficult to identify exposure-response relationships. However, there are a number of reasons why a relationship between a chemical and a toxic response in a natural system may not be apparent (Exhibit 25-6). Therefore, the lack of an observed exposure-response relationship does not disprove that one or more air toxics caused an apparent toxic effect. These sources of variation should be considered during planning and scoping, but may not become apparent until field studies have begun.

Exhibit 25-5. Questions to Consider When Extrapolating from Effects Observed in the Laboratory to Potential Effects in Natural Systems
Exposure Factors • How will environmental fate and transformation of the air toxic affect exposure in the field? • How comparable are exposure conditions and the timing of exposure? • How comparable are the routes of exposure? • How do abiotic factors influence bioavailability and exposure? • How likely are preference and avoidance behaviors in the receptors of concern? • How does life-stage affect exposure? **Effects factors** • What is known about the biotic and abiotic factors controlling populations of the receptors of concern? • To what degree are critical life-stage data available? • How may exposure to the same or other stressors in the field have altered organism sensitivity?

Empirical approaches are derived from experimental data or observations. They commonly are used when adequate effects data are available, but the understanding of the underlying mechanisms, action, or ecological principles is limited. Two types of empirical approaches are generally used:

- **Uncertainty factors** are derived numbers that are divided into measure of effects values to derive an estimated level of stressor that should not cause adverse effects to the assessment endpoint. An example might be an uncertainty factor of 10 to convert an acute LC_{50} value into a presumed NOAEL. Uncertainty factors should be used with caution, especially when used in an overly conservative fashion, as when chains of factors are multiplied together without sufficient justification.

- **Allometric scaling** is used to extrapolate the effects of a chemical stressor on one species to another species. Allometry is the study of change in the proportions of various parts of an organism as a consequence of growth and development. Processes that influence toxicokinetics (e.g., renal clearance, basal metabolic rate, food consumption) tend to vary across species according to allometric scaling factors that can be expressed as a nonlinear function of body weight. Allometric scaling factors are commonly used for human health toxicity assessments (see for example Chapter 12), but have not been applied as extensively to ecological effects.

When sufficient information on stressors and receptors is available, process-based approaches such as population or ecosystem process models may be used. Process models allow information on individual effects (e.g., mortality, growth, reproduction) to be extrapolated to potential alterations in specific populations, communities, or ecosystems. Such models are particularly useful in evaluating hypotheses about the duration and severity of impacts from a stressor on an assessment endpoint (e.g., species diversity) that cannot be tested readily in a laboratory. Two types of process-based models are commonly used:

Exhibit 25-6. Reasons Why Contaminant Concentrations in Ambient Media May Not Be Correlated with Toxicity of Those Media
Variation in bioavailability • Due to variance in medium characteristics • Due to variance in contaminant age among locations (contaminants deposited to soil and sediments may become less bioavailable over time due to sequestration) • Due to variance in transformation or sequestration rates among locations **Variation in the form** of the chemical (e.g., ionization state) **Variation in the concentration** over time or space (i.e., samples for analysis may not be the same as those tested) • Spatial heterogeneity • Temporal variability (e.g., aqueous toxicity tests last for several days but typically water from only one day is analyzed) **Variation in the composition of releases** (concentrations of components of releases other than the individual air toxic that is believed to be the principal toxicant may vary over space and time, thereby obscuring the relationship) **Variation in co-occurring contaminants** (concentrations of contaminants from upgradient [background] sources may vary over time) **Inadequate detection limits** (if detection limits are too high, gradients of toxic effect may be observed even when the chemicals are at the "not detected" levels) **Variation in toxicity tests** • Inherent variation • Variation due to variance in medium characteristics (e.g., hardness, organic matter content, pH) *Source: Guidelines for Ecological Risk Assessment*[1]

- **Single-species population models** describe the dynamics of a finite group of individuals through time. They have been used extensively in ecology and fisheries management to assess the impacts of power plants and toxic chemicals on specific fish populations.

- **Community and ecosystem models** are particularly useful when the assessment endpoint involves structural (e.g., community composition) or functional (e.g., primary productivity) elements or when secondary effects are of concern.

Exhibit 25-7 provides further discussion of process-based models, highlighting a few models that have been applied in ecological risk assessment.

Exhibit 25-7. Process-based Model Applications in Ecological Risk Assessment

Process-based models can help the assessor understand the potential significance of toxicant effects to the population structure, and ecosystem models can help determine whether the effect may result in secondary effects on other species in the system that are linked in the food web or on overall ecosystem functions. Pastorok et al.[12] review a number of population, and community and ecosystem models, as well as software that implement these models.

Population models typically deal with the dynamics of the abundance or distribution of a single species, sometimes with explicit descriptions of endpoints in time and space. These models can be categorized as scalar abundance, life history, individual-based, and metapopulation models. The first two types of models are highlighted here:

- *Scalar abundance models*, which represent populations as a single scalar dimension without a breakdown of population age structure, are frequently used in screening assessments. These models include Malthusian population growth models and logistic population growth models.
- *Life history models* estimate population characteristics such as survival rates and fecundity as a function of age or size/morphological state. These models are important because toxicants can have a differential impact on different demographic sections of the same species. These models include deterministic and stochastic age- or stage-based models, which are implemented in software by programs such as *RAMAS*-Age®, *-Stage*®, *-Metapop*®, *or -Ecotoxicology*®; and *ULM*®.

Community and Ecosystem models are intended to describe ecological systems composed of interacting species. These models incorporate species dynamics and specific biological interactions (predator-prey, competition, dependence) to predict ecosystem endpoints such as species richness or the productivity of a multi-species assemblage. Pastorok et al. categorize these models as food web, aquatic, and terrestrial models.

- *Food web models* capture feeding relationships between all or some species in an ecological community, thus determining population dynamics as well as identifying key exposure pathways for bioaccumulative chemicals. These models include predator-prey models and population-dynamic food chain models, which are implemented in software such as *RAMAS Ecosystem*®, *Populus*®, and *Ecotox*.
- *Aquatic ecosystem models* are spatially aggregated models that represent biotic and abiotic structures in combination with physical, chemical, biological, and ecological processes in rivers, lakes, reservoirs, estuaries, or coastal ecosystems. A number of models exist for each type of aquatic ecosystem. The standard water column model or *SWACOM*® requires the use of laboratory data to predict changes in the parameters of an entire ecosystem. The extrapolation is accomplished with knowledge of toxicological modes of action, and by simulation of the effects of a toxic substance across different trophic levels according to the relationship between nutrients, phytoplankton, zooplankton, and fish. *AQUATOX* (http://www.epa.gov/ost/models/aquatox/) predicts the fate of various pollutants, such as nutrients and organic chemicals, and their effects on the aquatic ecosystem, including fish, invertebrates, and aquatic plants. The Comprehensive Aquatic Simulation Model (*CASM*) is a bioenergetics-based food web model that includes phytoplankton, periphyton, macrophytes, zooplankton, benthic invertebrates, fish, bacteria, and cyanobacteria.
- *Terrestrial ecosystem models* represent biotic and abiotic components in deserts, forests, grasslands, or other terrestrial environments, and often include physical, chemical, biological, and ecological processes. The primary endpoints of these models include the abundance of individuals within species or guilds, biomass, productivity, and food-web endpoints such as species richness or trophic structure.

25.3 Stressor-Response Profile

The final product of an ecological response analysis is a summary profile in the form of a written document or a component of a larger process model. The stressor-response profile should address the following questions:

- What ecological entities are affected? These may include single species, populations, general trophic levels, communities, ecosystems, or landscapes.

- What are the nature of the effects? The nature of effects should be germane to the assessment endpoints. For example, if a single species is affected, the effects should represent parameters (e.g., growth, reproduction) appropriate for that level of organization.

- Where appropriate, what is the time scale for recovery? Short- and long-term effects should be reported as appropriate.

- How do changes in measures of effects relate to changes in assessment endpoints (see Section 25.2.2 above)?

- What is the uncertainty associated with the analysis (see Section 25.4)?

25.4 Evaluating Variability and Uncertainty

The stressor-response profile described in the previous section should include an explicit description of any uncertainties associated with the ecological response analysis. If it was necessary to extrapolate from measures of effect to the assessment endpoint, both the extrapolation and its basis should be described. Similarly, if a TRV was calculated, the extrapolations, assumptions, and uncertainties associated with its development should be described. The discussion also should include any information about known or potential variability in a stressor-response profile (e.g., among different species or taxa).

Professional judgment often is needed to determine the uncertainty associated with information taken from the literature and any extrapolations used in developing a parameter to estimate stressor-response. All assumptions used to develop stressor-response relationships and TRVs should be stated, including some description of the degree of bias possible in each. Where literature values are used, an indication of the range of values that could be considered appropriate also should be indicated. A more thorough description of how to deal with variability and uncertainty in the risk assessment process is provided in Chapter 31.

References

1. U.S. Environmental Protection Agency. 1998. *Guidelines for Ecological Risk Assessment*. Risk Assessment Forum, Washington, D.C., April 1998. EPA/630/R095/002F. Available at: http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=12460.

2. Sheehan, P.J., Baril, A., Mineau, P., Smith, D.K., Harfenist, A., and Marshall,W.K. 1987. *The Impact of Pesticides on the Ecology of Prairie-nesting Ducks*. Technical Report Series, No.19. Canadian Wildlife Service, Ottawa.

3. Lohman, K. et. al. 2000. A probabilistic analysis of regional mercury impacts on wildlife. *Human and Ecological Risk Assessment* 6:103-130.

4. Lawton, J.H. and Brown, V.K. 1994. *Redundancy in Ecosystems: Biodiversity and Ecosystem Function.* Springer-Verlag, Berlin Heidelberg, Germany. pp. 255-270.

5. U.S. Environmental Protection Agency. 1995. *Great Lakes Water Quality Initiative Criteria Documents for the Protection of Wildlife: DDT, Mercury, 2,3,6,8-TCDD, PCBs*. Office of Water, Washington, D.C. EPA/820/B95/008.

   U.S. Environmental Protection Agency. 1995. *Final Water Quality Guidance for the Great Lakes System. Final Rule. Federal Register* 60:15366, March 23, 1995.

6. U.S. Environmental Protection Agency. 1993. *Addendum to the Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions*. Office of Research and Development, Washington, DC. EPA/600/AP93/003.

7. U.S. Environmental Protection Agency. 1993. *Wildlife Exposure Factors Handbook.* Office of Research and Development, Washington, D.C. EPA/600/R93/187. Available at: http://cfpub.epa.gov/ncea/cfm/wefh.cfm?ActType=default.

8. Efroymson, R.A., Will, M.E., and Suter II, G.W. 1997. *Toxicological Benchmarks for Contaminants of Potential Concern for Effects on Soil and Litter Invertebrates and Heterotrophic Processes: 1997 Revision.* Oak Ridge National Laboratory, Oak Ridge TN. ES/ER/TM-126/R2.

9. U.S. Environmental Protection Agency. 2003. *Integrated Risk Information System.* Office of Research and Development, National Center for Environmental Assessment. Available at: http://www.epa.gov/iris/.

10. Sample, B.E., Opresko, D.M., and Suter II, G.W. 1996. *Toxicological Benchmarks for Wildlife: 1996 Revision.* June 1996. ES/ER/TM-86/R3. Available at: http://www.hsrd.ornl.gov/ecorisk/tm86r3.pdf .

12. U.S. Environmental Protection Agency. 2003. *Benchmark Dose Software.* National Center for Environmental Assessment. Available at: www.epa.gov/ncea/bmds.htm.

13. Pastorok, R.A., Bartell, S.M., Ferson, S., Ginzburg, L.R.(eds). 2002. *Ecological Modeling in Risk Assessment: Chemical Effects on Populations, Ecosystems and Landscapes.* Lewis Publishers, Boca Raton, FL.

Chapter 26 Ecological Risk Characterization

Table of Contents

26.1 Introduction 1

26.2 Risk Estimation 2
- 26.2.1 Single-Point Exposure and Effects Comparisons 2
- 26.2.2 Comparisons Involving the Entire Stressor-Response Relationship 3
- 26.2.3 Comparisons Involving Variability 4
- 26.2.4 Process Models 4

26.3 Risk Description 5
- 26.3.1 Lines of Evidence 5
- 26.3.2 Significance of the Effects 6

26.4 Risk Characterization Report 7

26.5 Evaluating Variability and Uncertainty 9

References 9

26.1 Introduction

Similar to human health risk characterization, ecological risk characterization combines information concerning exposure to chemicals with information regarding effects of chemicals to estimate risks. The major difference in ecological risk characterization is the necessity for estimating risks based on individual lines of evidence and then combining them through a process of weighing the evidence.[a] Another difference is that in human health assessment, we primarily consider health effects in the bodies of individual people. In ecological assessment, we consider various "health" issues that can range from actual health effects in the bodies of individual ecological receptors to something more attuned to the "health" of the ecosystem as measured by species richness and diversity. This chapter provides an overview of the approaches and methods used for ecological risk characterization. As before, additional information is provided in EPA's *Guidelines for Ecological Risk Assessment*,[(1)] and readers are referred to that document for a more complete discussion of available approaches and methods.

Risk characterization is the final phase of ecological risk assessment and is the culmination of the planning and scoping, problem formulation, and analysis of predicted or observed adverse ecological effects related to the assessment endpoints. It is also based on metrics of exposure and ecosystem and receptor characteristics that are used to analyze air toxics sources, their distribution in the environment, and the extent and pattern of contact. Risk characterization is used to clarify the relationships between stressors, effects, and ecological entities, and to reach conclusions regarding the occurrence of exposure and the likelihood of anticipated effects. The results of the analysis phase are used to develop an estimate of the risk posed to the ecologically valued entities that are the focus of the assessment endpoints.[(2)] After estimating the risk, the risk estimate is described in the context of the significance of any adverse effects and lines of evidence supporting their likelihood. Finally, the uncertainties, assumptions, and qualifiers in the risk assessment are identified and summarized, and the conclusions are reported to risk managers.

Conclusions presented in the risk characterization should provide clear information to risk managers in order to be useful for environmental decision making. If the risks are not sufficiently defined to support a management decision, risk managers may elect to proceed with another iteration of one or more phases of the risk assessment process. Re-evaluating the conceptual model (and associated risk hypotheses) or conducting additional studies may improve the risk estimate.

Characterization of ecological risk includes risk estimation, (usually a quantitative risk estimate; see Section 26.2), risk description (Section 26.3), and documentation of results (Section 26.4).

---

[a]Consistent with EPA's *Guidelines for Ecological Risk Assessment*,[(1)] the term "lines of evidence" includes a "weight of evidence" in order to emphasize that both qualitative evaluation and quantitative weighting may be used.

26.2 Risk Estimation

Several general techniques are available for characterizing ecological risks associated with air toxics that persist and bioaccumulate. These are divided broadly into single-point comparisons, comparisons incorporating the entire stressor-response relationship, comparisons involving variability in exposure and/or effects, and process models. Each is described in a separate subsection below. EPA's *Guidelines for Ecological Risk Assessment*[1] provides additional discussion and examples of these techniques.

26.2.1 Single-Point Exposure and Effects Comparisons

The simplest approach for comparing exposure and effects estimates for air toxics ecological risk assessments is the Hazard Quotient (HQ) approach (also referred to as the "quotient method"), which is similar to that used for human noncancer health risk assessments (see Chapter 13). In this approach, modeled or measured concentrations of the chemical in each environmental medium are divided by the appropriate point estimate for ecological effects to yield a HQ for an individual chemical.

$$HQ = \frac{Oral\ Intake}{TRV} \quad or \quad HQ = \frac{EEC}{TRV} \quad or \quad HQ = \frac{BB}{TRV} \qquad \text{(Equation 26-1)}$$

where:

HQ	=	hazard quotient
Oral Intake	=	estimated or measured contaminant intake relevant to the oral intake-based TRV (usually expressed as mg/kg-day)
TRV	=	Toxicity reference value. This may be in terms of oral intake, media concentration, or body burden. As described elsewhere, it may be a result of a single study (e.g., NOAEL) or the result of integration of multiple studies (e.g., water quality criterion).
EEC	=	estimated or measured environmental media concentration at the exposure point (usually expressed as mg/L for water and mg/kg for soil and sediment)
BB	=	estimated or measured body burden (usually expressed as mg/kg wet weight)

As with human health assessments, the measure of oral intake, EEC, or BB must be in the same units as the TRV to which the measure is being compared.

As chronic risk will usually "drive" an ecological assessment, the HQ approach will usually be employed for chronic exposure scenarios using chronic duration TRVs. For initial screening, conservative exposure factors may be used (see Exhibit 24-2). As in human health risk assessment, an HQ greater than one indicates the potential for adverse ecological effects to occur, but does not predict their occurrence (see Chapter 13).

When ecological toxicity data for complex mixtures are unavailable, the hazard index (HI) approach[b] may sometimes be used in screening assessments, as scientifically appropriate, to assess potential ecological risks associated with simultaneous exposure to multiple air toxics.[1]

If the HI approach is used, the assumptions and associated limitations should be clearly documented. It may often be the case that a single chemical is responsible for the HI exceeding one, and the assessment can then focus on the HQ for that chemical. In more refined assessments, an alternative approach may be necessary.

As with human health assessments, a number of limitations restrict application of the HQ approach. While a quotient can be useful in answering whether adverse effects are likely to occur or not, it may not be helpful to a risk manager who needs to make a decision requiring an incremental quantification of ecological hazard. For example, it is seldom useful to say that a mitigation approach will reduce the value of a quotient from 25 to 12, since this reduction cannot, by itself, be clearly interpreted in terms of effects on an assessment endpoint. Quotients also may not be the most appropriate methods for predicting secondary effects (e.g., bioaccumulation, loss of prey species). Finally, in most cases the quotient does not explicitly consider uncertainty, such as extrapolation from the test species to the species or community of concern. Some uncertainties, however, can be incorporated into single-point estimates to provide a statement of likelihood that the effects point estimate exceeds the exposure point estimate (see Exhibit 26-1).[1]

State Water Quality Standards

Pursuant to Section 303 of the Clean Water Act, States have developed numerical water quality standards for the protection of aquatic ecosystems. These standards generally are considered regulatory requirements that must be met, and often are based on EPA's Ambient Water Quality Criteria (see Chapter 25). If persistent, bioaccumulative hazardous air pollutants (PB-HAPs) enter surface waters, one way to assess risk is to compare the EEC to a water quality standard using the HQ approach. State water quality standards can be accessed via EPA's national water quality standards database at http://www.epa.gov/ost/wqs/.

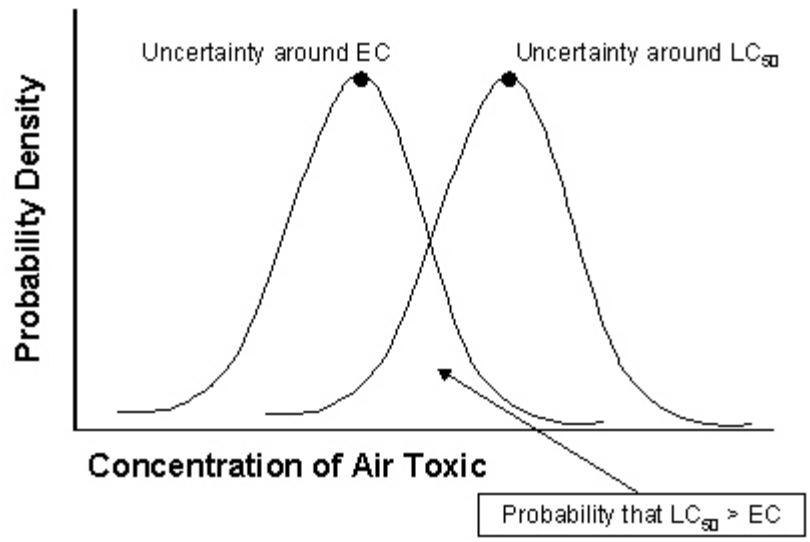
26.2.2 Comparisons Involving the Entire Stressor-Response Relationship

If a curve relating the intensity or level of the stressor to the magnitude of response is available (for example, see Exhibit 25-1), the risk characterization can examine risks associated with many different levels of exposure. These estimates are particularly useful when the risk management decision is not based on exceeding a pre-determined reference value or regulatory standard (e.g., a state water quality standard). This approach provides a predictive ability lacking in the hazard quotient approach, and it may be used in screening level assessments or subsequent more refined risk analyses. Because the slope of the effects curve relates the magnitude of change in effects to incremental changes in exposure, the ability to predict changes in the magnitude and likelihood of effects for different exposure scenarios can be used to compare different risk management options. Also, uncertainty can be incorporated by calculating uncertainty bounds on the stressor-response or exposure estimates. Limitations to this approach may include: lack of consideration

[b] The HI approach is termed the "quotient addition approach" in EPA's *Guidelines for Ecological Risk Assessment*[1]

for secondary effects, assuming the exposure pattern used to derive the stressor-response curve is comparable to the environmental exposure pattern; and failing to consider uncertainties such as extrapolations from tests species to the species or communities of concern.

Exhibit 26-1. Example Comparison of Point Estimates with Associated Uncertainties



26.2.3 Comparisons Involving Variability

If the exposure or stressor-response profiles describe the variability in exposure or effects, then many different risk estimates can be developed. Variability in exposure can be used to estimate risks to moderately or highly exposed members of a population being investigated, while variability in effects can be used to estimate risks to average or sensitive members of populations. As an example, exposure can vary by life-stage (e.g., exposure may be greater during spawning or migration). Likewise, effect may also vary by life-cycle (e.g., hatchlings may be more sensitive to a chemical than are adults). A major advantage of this approach is its ability to predict changes in the magnitude and likelihood of effects for different exposure scenarios and thus provide a means for comparing different risk management options. Limitations include the increased data requirements compared with previously described techniques and the implicit assumption that the full range of variability in the exposure and effects data is adequately represented. In addition, secondary effects are not readily evaluated with this approach. This risk estimation technique would likely be used in more refined risk assessments. (A discussion of probabilistic techniques, including Monte Carlo Simulation, is provided in Chapter 31.)

26.2.4 Process Models

Process models are mathematical expressions that represent understanding of the mechanistic operation of a system under evaluation. They can be useful tools in both analysis and risk characterization (process models are discussed briefly in Chapter 25). A major advantage of using process models is the ability to consider “what if” scenarios and to forecast beyond the limits of observed data that constrain approaches based solely on empirical data. Process models

also can consider secondary effects, and in some cases, the combined effects of multiple stressors. Process model outputs may be point estimates, distributions, or correlations. However, since process models are only as good as the assumptions on which they are based, the outputs from these models should be interpreted with care. The lack of knowledge on basic life histories for many species, and incomplete knowledge about the structure and function of natural ecosystems are some of the many uncertainties that need to be considered. These models are complex and, are usually reserved for more refined risk assessments.

Risk Assessment Frontiers: Integrating Human Health and Ecological Risk Assessment

Many tribal cultures view ecological and human health in an integrated way such that they cannot be easily separated. Similarly, there is some effort (especially in Canada) toward an integration of human health and ecological assessment, as well as decision-making, in a field known as **strategic environmental assessment**.[3] This approach has not been applied widely in the United States, and it remains to be seen how it will develop in the next few years.

The World Health Organization has published approaches to integrating human health and ecological risk assessments to improve data quality and understanding of cumulative risks for decision making.[4] This approach includes an integrated framework (modified from EPA's guidance)[1] and case studies.

EPA, in its *Framework for Cumulative Risk Assessment*,[5] offers a flexible structure for conducting and evaluating cumulative risk assessment. By "cumulative risk," EPA means "the combined risks from aggregate exposures to multiple agents or stressors." Agents or stressors may be chemicals, but they may also be biological agents or physical agents, or an activity that, directly or indirectly, alters or causes the loss of a necessity such as habitat.

26.3 Risk Description

The results of the risk characterization should be documented in the **risk description**, which includes an evaluation of the lines of evidence supporting or refuting the risk estimate(s) and an interpretation of the significance of the observed and/or predicted effects.

26.3.1 Lines of Evidence

The development of lines of evidence provides both a process and a framework for reaching a conclusion regarding confidence in the risk estimate. Confidence in the conclusions of a risk assessment may be increased by using several lines of evidence to interpret and compare risk estimates. These lines of evidence may be derived from different sources or by different techniques relevant to adverse effects on the assessment endpoints (e.g., hazard quotients, modeling results, or field observational studies). There are three principal categories of factors to consider when evaluating lines of evidence:

1. **Data adequacy and quality**. Data quality directly influences confidence in the results of a risk assessment and the conclusions that can be drawn from the study. Specific concerns include: whether the experimental design was appropriate for the questions being evaluated in the risk assessment; whether data quality objectives were clear and adhered to; and whether the analyses were sufficiently sensitive and robust to identify stressor-caused effects in light of natural variability of the attributes of the ecological receptors of concern.

2. **Relative uncertainty**. One major source of uncertainty comes from extrapolations (e.g., from one species to another; from one temporal scale to another; from laboratory to field effects). In general, the greater the number of extrapolations, the greater the uncertainty.

3. **Relationship to the risk hypothesis**. Finally, the relative importance of each line of evidence may be determined by how directly they relate to the risk hypothesis developed during planning and scoping. For example, lines of evidence based on a definitive mechanism rather than associations alone are likely to be relatively important.

The evaluation of lines of evidence involves more than just listing the evidence that supports or refutes the risk estimate. Each factor should be examined carefully, and its contribution in the context of the risk assessment should be evaluated. For example, data or study results are often not reported or carried through the risk assessment because they are of insufficient quality. If such data or results are eliminated from the evaluation process, however, valuable information may be lost with respect to needed improvements in methodologies or recommendations for further studies.

When lines of evidence do not point toward the same conclusion, it is important to investigate possible reasons for the disagreements. A starting point is to distinguish between true inconsistencies and those related to methodology (e.g., statistical powers of detection). For example, if a model predicts adverse effects that were not observed in the field, it is important to determine whether the model predictions were unrealistic, or the experimental design of the field study was inadequate to detect the predicted effects, or both.

26.3.2 Significance of the Effects

In this step, the significance of the observed or estimated changes in the assessment endpoints is interpreted in light of the lines of evidence evaluated above. In this context, significance refers to a conclusion as to whether the observed or estimated changes are considered "adverse." Adverse ecological effects represent changes that are undesirable because they alter valued structural or functional attributes of the ecological receptors of concern (e.g., the loss of a keystone species). This determination is difficult and is frequently based on professional judgment. The assessment of degree of adversity, along with other factors such as the economic, legal, or social consequences of the ecological change, may be considered in the risk management decision. Unless an endangered or threatened species is at issue, society is generally not concerned with the death of individual plants or animals, and therefore significance is generally assessed at the population, community, or ecosystem level(s). The following factors may be used to evaluate the degree of adversity (see also Exhibit 26-2):

- **Nature and intensity of effects**. This focuses on distinguishing adverse changes from those that are within the normal pattern of ecosystem variability or that result in little or no significant alteration of biota. For example, if survival of offspring will be affected, by what percentage will it diminish, and is that likely to have a major impact on population dynamics? It is important to consider both ecological and statistical information in evaluating the nature and intensity of effects. For example, a small change in a growth rate may not be statistically distinguishable from natural variation; however, its impact may be more significant for a population of slowly reproducing fish than for rapidly reproducing algae. When performing a more refined assessment, it is necessary to compare the potentially impacted ecosystem to a

non-impacted ecosystem (i.e., a "control" site) so there is a basis for statistical comparisons between the two systems.

Exhibit 26-2. Examples of Considerations for Determining Ecological Significance
• How large is the area where ecological criteria have been exceeded? • What proportion of the habitat is affected at local, county, State, and national levels? • Are the exposure concentrations and ecological criteria above background levels for the area of interest? • What types of ecological impacts have been associated with this pollutant or similar pollutants in the past? • Is the criterion or stressor-responsive curve based on high quality data (i.e., is there a high degree of confidence in the criterion)?

- **Spatial and temporal scale**. The spatial dimension encompasses both the extent and pattern of effect as well as the context of the effect within the broader ecosystem or landscape. Factors to consider include the absolute area affected, the percentage of area affected compared with a larger area of interest, and the relative importance of the affected area(s) to the ecological receptors of concern (e.g., are they critical breeding or overwintering areas?). For air toxics that persist and bioaccumulate, the temporal dimension of concern generally will be in the years to decades range, although effects in other time frames may be important in specific cases. Temporal responses for ecosystems may involve intrinsic time lags, so responses to a stressor (or risk mitigation effort) may be delayed.

- **Potential for recovery**. Recovery refers to the rate and extent of return of a population or community to some aspect of its condition prior to exposure to the stressor(s) of concern. Because ecosystems are dynamic, even under natural conditions, it is unrealistic to expect that a system will remain static at some level or return to exactly the same state that it was before it was disturbed. Thus, the "attributes" of a recovered population, community, or ecosystem should be carefully defined. In general, changes that preclude recovery or result in long recovery times are more significant than changes that allow rapid recovery. Note that different components of a community or ecosystem may recover at different rates. For example, stream chemistry may recover relatively rapidly after removal of a stressor, but re-establishment of predatory fish populations may take several years or more.

26.4 Risk Characterization Report

The information on estimates of ecological risk, the overall degree of confidence in the risk estimates, lines of evidence, and the interpretation of the significance of ecological effects generally is included in a **risk assessment or risk characterization report**. Exhibit 26-3 lists the elements that generally are considered in the risk characterization report. A risk characterization report may be brief or extensive, depending on the nature of and resources available for the assessment. The report need not be overly complex or lengthy; it is most important that the information required to support the risk management decision be presented clearly and concisely. To facilitate mutual understanding, EPA policy[6] requires that risk characterizations be prepared "in a manner that is clear, transparent, reasonable, and consistent with other risk characterizations of similar scope prepared across programs in the Agency." It describes a philosophy of transparency, clarity, consistency, and reasonableness (TCCR), and

provides detailed approaches to achieving TCRR. Exhibit 26-4 provides an overview of the TCRR principles (these are the same principles listed in Chapter 13).

Exhibit 26-3. Possible Risk Characterization Report Elements
• Describe risk assessor/risk manager planning results. • Describe the scope of the assessment. • Review the conceptual model and the assessment endpoints. • Describe the measures of effect. • Discuss the major data sources and analytical procedures used. • Review the stressor-response and exposure profiles. • Assign risks to the assessment endpoints, including risk estimates and adversity evaluations. • Review and summarize major areas of uncertainty (as well as their direction) and the approaches used to address them: – Discuss the degree of scientific consensus in key areas of uncertainty; – Identify major data gaps and, where appropriate, indicate whether gathering additional data would add significantly to the overall confidence in the assessment results; – Discuss science policy judgments or default assumptions used to bridge information gaps and the basis for these assumptions; and – Discuss how the elements of quantitative uncertainty analysis are embedded in the estimate of risk.

Exhibit 26-4. Transparency, Clarity, Consistency, and Reasonableness Principles

Principle	Definition	Criteria for a Good Risk Characterization
Transparency	Explicitness in the risk assessment process	• Describe assessment approach, assumptions, extrapolations, and use of models • Describe plausible alternative assumptions • Identify data gaps • Distinguish science from policy • Describe uncertainty • Describe relative strength of assessment
Clarity	The assessment itself is free from obscure language and is easy to understand	• Employ brevity • Use plain English • Avoid technical terms • Use simple tables, graphics, and equations
Consistency	The conclusions of the risk assessment are characterized in harmony with EPA actions	• Follow statutes • Follow Agency guidance • Use Agency information systems • Place assessment in context with similar risks • Define level of effort • Use review by peers
Reasonableness	The risk assessment is based on sound judgment	• Use review by peers • Use best available scientific information • Use good judgment • Use plausible alternatives

26.5 Evaluating Variability and Uncertainty

An important part of the Risk Characterization Report is a discussion and assessment of variability and uncertainty in all aspects of the ecological risk assessment. Note that ecological risk assessments are subject to additional sources of uncertainty and variability as compared to multipathway human health risk assessments. In addition to the uncertainties associated with multimedia modeling and sampling, the ecological risk assessment involves many decisions regarding choice of ecological receptors of concern and associated assessment and measures of effect. Some of these may be at levels of organization above individual species (e.g., communities, ecosystems), where stressor-response relationships are poorly understood. Because many different species and higher taxonomic groups may be included in the assessment, selection of many parameter values such as bioconcentration factors, dose-response values, and dietary intake is more complex and uncertain for the ecological risk assessment as compared to the human health multipathway risk assessment.

References

1. U.S. Environmental Protection Agency. 1998. *Guidelines for Ecological Risk Assessment*. Risk Assessment Forum, Washington, D.C., April 1998. EPA/630/R095/002F. Available at: http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=12460.

2. U.S. Environmental Protection Agency. 1992. *Framework for Ecological Risk Assessment*. Risk Assessment Forum, Washington, D.C., February 1992. EPA/630/R92/001.

3. Bonnell, S., and Storey, K. 2000. Addressing Cumulative Effects Through Strategic Environmental Assessment: a Case Study of Small Hydro Development in Newfoundland, Canada. *Journal of Environmental Assessment Policy and Management* 2: 477-499.

4. World Health Organization (WHO). 2001. *Approaches to Integrated Risk Assessment*. International Programme on Chemical Safety, Geneva. WHO/IPCS/IRA/01/12. Available at: http://www.who.int/pcs/emerg_main.html

5. U.S. Environmental Protection Agency. 2002. *Framework for Cumulative Risk Assessment*. Office of Research and Development, Washington, D.C., October 2002. EPA/630/P-02/001A.

6. U.S. Environmental Protection Agency. 2000. *Supplementary Guidance for Conducting Health Risk Assessment of Chemical Mixtures*. Risk Assessment Forum, Washington, DC. EPA/630/R-00/002. Available at: http://cfpub.epa.gov/ncea/raf/recordisplay.cfm?deid=20533.

PART V

RISK-BASED DECISION MAKING

Introduction to Part V

Part V of this Reference Manual provides an overview of three components of risk-based decision making.

- Risk Management (Chapter 27) refers to the regulatory and other actions taken to limit or control exposures to air toxics, including the role of risk management in regulating hazards.

- Community Involvement (Chapter 28) is an integral part of many risk management strategies because good community involvement helps ensure that the strategy selected will have the highest likelihood of success. Various levels of community involvement are also required by many laws.

- Risk Communication (Chapter 29) describes the process of planning the risk assessment (during scoping) and conveying the results of the risk assessment in a way that meets the information requirements for the risk management decisions. This chapter discusses the importance of risk communication, and planning and implementing a risk communication strategy.

Chapter 27 Risk Management

Table of Contents

27.1 Introduction 1

27.2 Role of Risk Management in Regulating Hazards 1

27.3 Types of Risk Management Decisions Related to Air Toxics 4

27.4 Use of Risk Estimates in Decision-Making 5

27.5 Process for Making Risk Management Decisions 9
- 27.5.1 Define the Problem and Put it in Context 9
- 27.5.2 Analyze the Risks Associated with the Problem in Context 9
- 27.5.3 Examine Options for Addressing the Risks 11
- 27.5.4 Make Decisions about Which Options to Implement 12
- 27.5.5 Take Actions to Implement the Decisions 13
- 27.5.6 Conduct an Evaluation of the Action's Results 14

27.6 Information Dissemination 14

References 15

27.1 Introduction

This chapter introduces risk management, focusing on its role in addressing the risks that air toxics pose. It provides an overview of the types of risk management decisions related to air toxics, a discussion of how risks to individuals and populations are presented to the public, and options for implementing decisions (e.g., regulation, voluntary risk reduction activities).

Specifically, **risk management** refers to the regulatory and other actions taken to limit or control exposures to a chemical. **Risk assessment**, on the other hand, is a tool used to support risk management decisions by providing quantitative and qualitative expressions of risk, along with attendant uncertainties. Specifically, the risk assessment conveys a quantitative and qualitative description of the types of impacts that may occur from exposure to an air toxic, the likelihood that these impacts will occur given existing conditions, and the uncertainties surrounding the analysis. Risk management considers these principle factors along with a variety of additional information (which may include the cost of reducing emissions or exposures, the statutory authority to take regulatory actions, and the acceptability of control options) to reach a final decision.

27.2 Role of Risk Management in Regulating Hazards

Risk management may include implicit or explicit policy and value judgments. Therefore, one would expect there to be differences of opinion concerning what represents an appropriate risk management action. Even the most basic risk management decision can be highly controversial. A classic example is the decision(s) needed to answer the question **how clean is clean?** This question refers to a risk management decision that must establish a target level to which existing levels of contamination/pollution should be reduced. Establishing this level is not a trivial matter. Working through these issues can be complicated by the different values of the stakeholders and debates over individual perceptions about risk. As discussed below, many authors and organizations stress the importance of understanding risk management mandates, options, and concerns throughout the risk assessment process, from the initial problem formulation steps to the final risk characterization and risk communication. Many of the critical decisions in structuring the technical risk assessment depend on risk management concerns (e.g., what risk management options are feasible, what level of certainty in the risk estimate is acceptable).

Although the National Academy of Sciences and others stress the **distinction** between risk assessment and risk management, they also stress the **integration** of the two efforts (see Exhibit 27-1). Risk assessments are often designed and conducted with awareness of the risk management options available to decision-makers and the social, economic, and political context in which those decisions are to be made. Likewise, periodically reviewing the risk management options during the risk assessment effort ensures that the results of the risk assessment will provide meaningful input into the decision-making process. The National Research Council (NRC) of the National Academy of Sciences (NAS), in their 1983 study entitled *Risk Assessment in the Federal Government: Managing the Process* (the "Red Book"),[1] advocated a clear conceptual distinction between risk assessment and risk management, noting, for example, that maintaining the distinction between the two would help to prevent the tailoring of risk assessments to the political feasibility of regulating a chemical substance. However, the NRC also recognized that the choice of risk assessment techniques could not be isolated from society's

risk management goals. Ultimately, the risk assessors should be aware of risk management goals; however, the fundamental science performed in the risk assessment should be impartial and based on the factual base of information, to the extent possible.

Use of the Term "Safe"

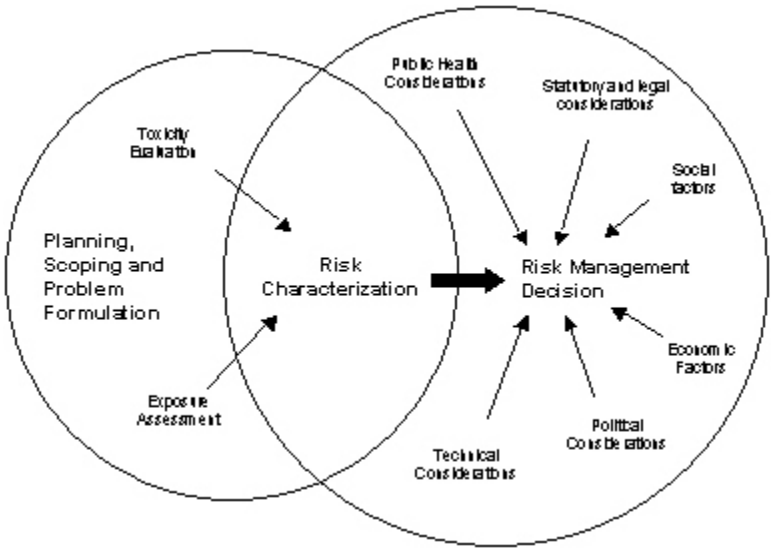
Safe: Condition of exposure under which there is a practical certainty that no harm will result to exposed individuals (as defined in EPA's *Terms of Environment*).

Safe: Free from harm or risk (as defined in the Merriam-Webster Collegiate Dictionary).

During government and community interactions and risk communication, it is important to be sensitive to perceived meanings of the term "safe." Regulators and scientists are often reluctant to use the term "safe," because many people understand "safe" to mean "zero risk." Ideally, one would like to eliminate all risks, but this is usually not a realistic expectation. Regulators commonly work to address the most important risks and decrease them to the level at which they believe the risks are smaller than the benefits of the activity causing the problem (in this case, risk from exposure to air toxics). They commonly refer to this level as "acceptably low risk."

However, community members may become frustrated with regulators who are reluctant to use the term "safe," potentially perceiving the regulators' choice of words as a dodge of the issue. Therefore, it is important for government representatives to address perceptions of the meaning of safe during risk communication and, as appropriate, use risk comparisons to help in communicating the concepts of safe versus acceptably low risk. Information on risk communication is provided in Chapter 29, and Section 29.4 provides specific information about risk comparisons.

Exhibit 27-1. Illustration of the Integration Between Risk Assessment and Risk Management

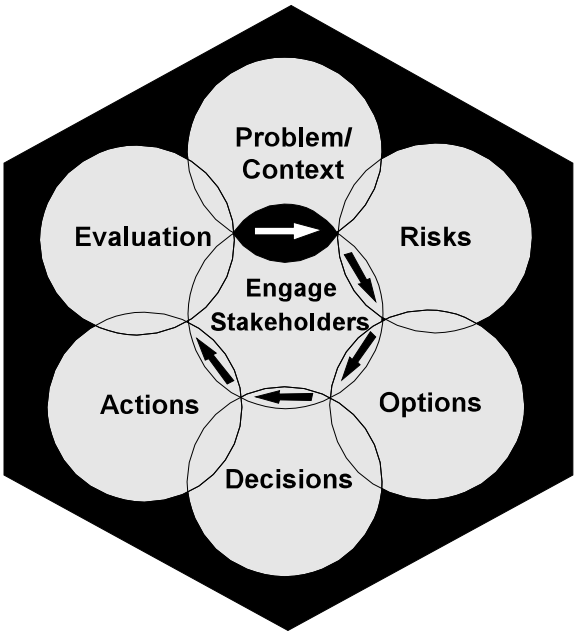


The NRC, in their 1994 report, *Science and Judgment in Risk Assessment* (the "Blue Book"),[2] noted that, while the Red Book emphasized the distinction between risk assessment and risk management, the purpose of separation was not to prevent any exercise of policy judgment when evaluating science or to prevent risk managers from influencing the type of information that assessors would collect, analyze, or present. The Blue Book concluded further that the science-policy judgments that EPA makes in the course of a risk assessment would be improved if there were more clearly informed by the Agency's priorities and goals in risk management. Protecting the integrity of the risk assessment, while building more productive linkages to make risk assessment more accurate and relevant to risk management, is essential.

The integration between risk assessment and risk management also has been emphasized by Presidential/Congressional Commission on Risk Assessment and Risk Management. In their Reports *Framework for Environmental Health Risk Management* and *Risk Assessment and Risk Management In Regulatory Decision-Making* (the two-volume "White Book"),[3] the Commission developed a six-stage integrated framework for environmental health risk management that can be applied to most situations (Exhibit 27-2):

1. Define the problem and put it in context;
2. Analyze the risks associated with the problem in context;
3. Examine options for addressing the risks;
4. Make decisions about which options to implement;
5. Take actions to implement the decisions; and
6. Conduct an evaluation of the action's results.

Exhibit 27-2. The CRARM Framework for Risk Management



The Commission noted that the process of examining risk management options does not have to wait until the risk analysis is completed, although a risk analysis often will provide important information for identifying and evaluating risk management options. In some cases, examining risk management options may help refine a risk analysis. The Commission also recommended that all of these steps involve stakeholders (see Chapter 28).

When discussing risk management, it is important to consider **where** and **how** changes or interventions may occur in the causal sequence of environmental impacts since interventions may reduce pollutants a number of ways along the critical path of environmental impacts. For example, interventions such as changing manufacturing processes, implementing emissions controls, or influencing worker behaviors that actively reduce exposure may have a positive mitigating effect on environmental impacts. In the discussion of risk management that follows, it is critical to keep in mind the range of ways in which environmental risks can be mitigated; it is up to the risk managers to determine the most feasible and critical "points of entry" along the path when developing a risk management strategy.

27.3 Types of Risk Management Decisions Related to Air Toxics

Two general categories of risk management decisions are relevant to air toxics: emissions control and siting.

- **Emissions control**. Emissions control decisions may involve "command-and-control" decisions (e.g., emissions limits) or incentives (e.g., tax credits for reduced emissions). EPA's preference is to encourage pollution prevention whenever feasible (see Exhibit 27-3). Emissions control decisions are most likely to involve formal risk assessments.

- **Siting/locating**. These decisions involve where to locate industrial facilities, businesses, waste disposal facilities, and transportation routes. Siting decisions are typically made by S/L/T governments through mechanisms such as zoning, deed restrictions and other property controls, and in some cases regulation. Many of these decision-making processes include public involvement in which citizens may seek to influence the final decision. Siting decisions may involve assessment of environmental impacts pursuant to the National Environmental Policy Act, other federal statutes, or similar state statutes. Siting decisions may increasingly involve air toxics risk assessments.

Not All Risk Management Decisions are Regulatory

Some risk management decisions are made by EPA or state, local and tribal (S/L/T) regulators pursuant to specific statutory criteria. However, government agencies may have limited authority to impact many other decisions. For example, some decisions are made by the individuals who own or operate the facilities that release air toxics, while others are made by citizens who are being impacted by emissions. Risk management decisions may need to consider looking beyond technological solutions.

Exhibit 27-3. Pollution Prevention Hierarchy

In the Pollution Prevention Act of 1990, Congress established a hierarchy for the handling of pollution (see graphic). The Act established as United States policy that pollution should be prevented or reduced at the source whenever feasible, that pollution that cannot be prevented should be recycled in an environmentally safe manner whenever feasible, and that pollution that cannot be prevented or recycled should be treated in an environmentally safe manner whenever feasible. Disposal or other release into the environment should be employed only as a last resort and should be conducted in an environmentally safe manner.

Pollution prevention is the reduction or elimination of pollutants at the source. As defined in the Pollution Prevention Act, "source reduction" means any practice which (1) reduces the amount of any hazardous substance, pollutant, or contaminant entering any waste stream or otherwise released into the environment (including fugitive emissions) prior to recycling, treatment, or disposal, and (2) reduces the hazards to public health and the environment associated with the release of such substances, pollutants, or contaminants. It includes equipment or technology modifications, process or procedure modifications, reformulation or redesign of products, substitution of raw materials, and improvements in housekeeping, maintenance, training, or inventory control. Examples of the value of pollution prevention for reducing environmental risks at the community level are demonstrated by EPA's Environmental Justice through Pollution Prevention (EJP2) grant program. EPA encouraged community groups, tribes, and local governments to identify environmental problems and generate potential pollution prevention solutions for their communities.

Source: U.S. Environmental Protection Agency. 2002. *Environmental Justice Through Pollution Prevention Program.* Updated July 9, 2002. Available at: http://www.epa.gov/opptintr/ejp2/. (Last accessed April, 2004.)

27.4 Use of Risk Estimates in Decision-Making

Decision-makers have a number of options when deciding what types of risk estimates to consider as inputs to risk management decisions. Estimates of human health risk generally fall into two categories, estimated **cancer risk** and the estimated **noncancer hazard** magnitude of exposure concentration or dietary intake greater than a pre-established reference exposure level), as described in more detail in Chapters 13 and 22. Non-cancer hazard may be considered for both acute (short-term) and chronic (longer-term) exposures. In some cases, **ecological risk** may be a factor in decision-making.

In some situations, risk managers may choose to consider EPA's approach for assessing an "ample margin of safety." For cancer risks, EPA generally considers incremental risk (or probability) of cancer for an individual potentially exposed to one or more air toxics. In protecting public health with an ample margin of safety, EPA strives to provide maximum feasible protection against risks to health from HAPs by (1) protecting the greatest number of

persons possible to an individual lifetime risk level no higher than 1×10^{-6} (one in one million) and (2) limiting to no higher than approximately 1×10^{-4} (one in ten thousand) the estimated risk that a person living near a source would have if exposed to the maximum pollutant concentrations for 70 years. These goals are described in the preamble to the benzene National Emissions Standards for Hazardous Air Pollutants (NESHAP) rulemaking (54 *Federal Register* 38044, September 14, 1989) and are the goals incorporated by Congress for EPA's residual risk program under Clean Air Act (CAA) section 112(f). Exhibit 27-4 describes some of the key steps in the development of the 1×10^{-4} to 1×10^{-6} carcinogenic risk range.

For non-carcinogenic substances, on the other hand, risk managers may consider a reference level that is developed based on data from laboratory animal or human epidemiology studies (see Chapter 12), and to which uncertainty factors are applied. The reference level is usually an exposure level below which there are not likely to be any adverse effects from exposure to the chemical. Exposures above the reference level may have some potential for causing adverse effects. This concept may also be applied generally to ecological risks.

Risk estimate options generally revolve around estimates of individual risk, the number of people at different risk levels (population risk), and occasionally include the expected incidence of disease in the entire population. Risk estimates can be derived for the current population as currently distributed in an area or for a population size and geographic distribution that might occur in the future; similarly, they may focus on risk estimates for persons currently exposed or possible risks calculated for a hypothetical individual located where exposures are expected to be relatively high. It is important to note that risk estimates should strive to take into account both **indoor** and **outdoor** exposure to toxics, when possible.

- **Risk to a specified individual**. Most risk assessments focus on estimating individual risk rather than the incidence of adverse effects (e.g., numbers of predicted cancer cases per year) in a population. There are two general estimates of individual risk:

  – **High-end** risk estimates seek to determine a "plausible worst case" situation among all of the individual risks in the population. This estimate is meant to describe an individual who, as a result of where they live and what they do, experiences the highest level of exposure within some reasonable bounds. Reasonable maximum risk estimates are often defined conceptually as "above the 90th percentile of the population"[(4)] but not at a higher exposure level than the person exposed at the highest level in the population. When calculated using deterministic methods, the high-end individual is calculated by combining upper-bound and mid-range exposure factors (e.g., an average body weight, but high-end ingestion rate) so that the result represents an exposure scenario that is both protective and reasonable, but not higher than the worst possible case.

  – **Central-tendency** risk estimates seek to determine a reasonable "average" or "mid-range" situation among all of the individual risks in the population. Many risk management decisions related to exposure to radioactive substances (e.g., in nuclear power plants) are based on central-tendency risk estimates.

Exhibit 27-4. Development of the 1×10^{-4} to 1×10^{-6} Carcinogenic Risk Range

The 1970 CAA established Section 112 to deal with hazardous air pollutants. Once the EPA Administrator had identified such a pollutant and "listed" it, he/she was directed to set emission standards for sources emitting it at levels that would "provide an ample margin of safety to protect the public health." The regulation of benzene pursuant to Section 112 illustrates the evolution of risk-based decision-making for carcinogens and the consideration of the "ample margin of safety."

- EPA listed benzene as a HAP in June 1977 and indicated that the "relative risk to the public" would be considered in judging "the degree of control which can and should be required."

- In 1980, the first round of benzene standards followed the proposed procedures in EPA's 1979 draft airborne carcinogen policy, which reflected a technology-based approach to emission standard development with a limited role for quantitative risk assessment in establishing priorities and ensuring that the residual risks following the application of "best available technology" (BAT) were not unreasonable.

- In 1984, after "weighing all factors," EPA made several changes to the proposed benzene rules, arguing that the risks were "too small to warrant Federal regulatory action." These decisions were promptly challenged by the Natural Resources Defense Council, who argued about the uncertainties in the risk estimates and the inappropriate consideration of cost in regulatory decisions made under Section 112. The issues raised were similar to litigation already pending on amendments to the original vinyl chloride standards.

- On July 28, 1987, Judge Robert Bork, writing for the D.C. Circuit Court of Appeals, remanded the vinyl chloride amendments to EPA, finding that the Agency had placed too great an emphasis on technical feasibility and cost rather than the provision of an "ample margin of safety" as required by the statute. The opinion also laid out a process for making decisions, consistent with the requirements of the law. The Bork opinion held that EPA must first determine a "safe" or "acceptable" level considering only the potential health impacts of the pollutant. Once an acceptable level was identified, the level could be reduced further, as appropriate and in consideration of other factors, including cost and technical feasibility to provide the required ample margin of safety. The Court also held, however, that "safe" did not require a finding of "risk-free" and that EPA should recognize that activities such as "driving a car or breathing city air" may not be considered "unsafe."

- In September of 1989, after proposing several options and receiving considerable public comment, EPA promulgated emission standards for several categories of benzene sources. EPA argued for the consideration of all relevant health information and established "presumptive benchmarks" for risks that would be deemed "acceptable." The goal, which came to be known as the "fuzzy bright line," is to protect the greatest number of persons possible to an individual lifetime risk no higher than one in 1,000,000 and to limit to no higher than approximately one in 10,000 the estimated maximum individual risk. The selection of even "fuzzy" risk targets placed greater emphasis on the development and communication of risk characterization results.

Source: National Academy of Sciences' *Science and Judgment in Risk Assessment* (The Blue Book).[2]

Note that, when calculating deterministic risk estimates, both a high end and central tendency estimate of risk give the risk manager some sense of the range of risks in the population. When risks to a population are developed using probabilistic methods, this becomes a moot point, since the result is a distribution of risks across the population, which necessarily includes information about the full variability of risk across the population – including both high and central tendency risks. See Chapter 31 for more information on probabilistic approaches to risk assessment.

- **Risk to the total population**. Whether or not risk to the total population is considered by EPA may depend on the regulatory authority provided by the CAA. For example, Section 112(k) of the CAA requires EPA to develop an Urban Air Toxics Strategy to reduce HAPs from area sources to achieve a 75 percent reduction in cancer incidences attributable to such sources. Two general types of descriptors are used for population risk. One, sometimes termed **population at risk** is derived by determining the number of people in a population with a particular individual risk level (e.g., "1,340,000 people are exposed at the 1×10^{-6} level, and 320 people are exposed at the 1×10^{-4} level"). This is a useful estimate of the variability of risk in a population.

- **Incidence**, another descriptor used for population risk, is an estimate of the total number (incidence) of adverse effects in a population over a specified time period (e.g., a period of 70 years). A screening approach to deriving this estimate for a 70-year period involves multiplying the estimate of individual risk (central tendency and/or reasonable maximum) by the number of persons for which that risk estimate was predicted. For example, in a population of 200 million persons, an individual cancer risk of 1×10^{-4} (i.e., one in ten thousand) for everyone in the population would translate to an incidence of hundreds or thousands of excess cancer cases over a 70-year period (depending on the exposure assumptions). However, in a small population (e.g., a town of 200 persons), the same individual cancer risk to everyone would translate to an excess incidence of cancer of less than one over a 70-year period.

- **Present versus future scenarios**. Risks may be characterized using present or future scenarios. Use of present scenarios involves predicting risks associated with the current exposures to individuals (or populations) that currently reside in areas where exposures are predicted to occur. For example, a current population risk estimate would use the existing population within some specified area. The resultant risk estimates are associated with the presumption that the current exposure conditions exist for the current population over the period of time associated with the assessment (e.g., into the future). Use of future population scenarios involves estimating risks associated with exposure conditions to individuals that might reside, at some future point, in areas where potential exposures may occur (e.g., if a housing development were built on currently vacant land).

- **Potential risk**. Risks may be sometimes be characterized for hypothetical exposures. For example, in a screening air toxics modeling application, a potential risk estimate may be derived using the location where the maximum modeled exposure concentration occurs, regardless of whether there is a person there or not. This estimate may be considered along with the predicted individual risk associated with a currently populated area, such as the MIR, which reflects risk associated with the maximum exposure concentration at an actual residence or in a census block with a non-zero population (see Chapter 11).

27.5 Process for Making Risk Management Decisions

A number of different authors and organizations have identified key steps or factors to consider in making risk management decisions. The discussion in this section is taken largely from the risk management framework developed by the Presidential/Congressional Commission on Risk Assessment and Risk Management.[3] The Commissions's framework has six stages, each of which is briefly described below. The Commission also noted that the framework is conducted:

- In collaboration with stakeholders; and
- Using iterations if new information is developed that changes the need for or nature of risk management.

27.5.1 Define the Problem and Put it in Context

The problem/context stage is the most important step in the Risk Management Framework. It involves:

- Identifying and characterizing an environmental health problem, or a potential problem, caused by chemicals or other hazardous agents or situations;
- Putting the problem into its public health and ecological context;
- Determining risk management goals;
- Identifying risk managers with the authority or responsibility to take the necessary actions; and
- Implementing a process for engaging stakeholders.

These steps are all important, but may be conducted in different orders, depending on the particular situation. For example, when a federal or S/L/T regulatory agency is mandated by law to take the lead on an air toxics issue, the steps they take often will proceed in the order listed above, with the identity of the risk managers already clear, since the agency will have assumed that role from the start. On the other hand, in a community based effort to characterize the cumulative risk posed by multiple sources of air toxics in a neighborhood, stakeholders might have to engage in a collaborative stakeholder process first to identify resources as well as risk managers with the needed authority to act before the other steps can take place.

27.5.2 Analyze the Risks Associated with the Problem in Context

The nature, extent, and focus of a risk assessment should be guided by the risk management goals. The results of a risk assessment – along with information about public values, statutory requirements, court decisions, equity considerations, benefits, and costs – all can influence whether and how to manage the risks.

Risk assessment can be controversial, reflecting the important role that both science and judgment play in drawing conclusions about the likelihood of effects on human health and the

environment. Often, the controversy arises from what we do not know and from what risk assessments cannot tell us, because our knowledge of human vulnerability and of environmental impacts is incomplete, especially at the relatively low levels of chemical exposure commonly encountered in the general community.

Some Factors to Consider in Defining the Problem for an Air Toxics Risk Assessment

- **Risk**. The specific estimates of risk to be used as inputs to the decision should be defined as explicitly as possible. Are acute risks (e.g., short-term exposures) the primary concern, or are exposures over the longer-term more important? Are ecological risks a concern? How certain are we that our risk estimates are an accurate reflection of true exposure and risk?

- **Air toxics of concern**. What are the primary air toxics of concern? Are they more prevalent in indoor or outdoor environments? How many individual chemicals contribute to the risks that need to be managed? Do these chemicals exert their effects independently, or are some acting in a synergistic (or antagonistic) manner? Are all equally important, or will reducing exposures to a subset of these air toxics result in adequate risk reduction? How important is it to manage every chemical of concern versus only those that pose the greatest risk?

- **Sources**. What are the primary sources of the air toxics that need to be managed? Where are these sources located? How many are there? Are they all equally important, or will controlling a subset result in adequate risk reduction?

- **Exposure pathway considerations**. What exposure pathways/routes are most important? Are all equally important, or does a subset represent the greatest risk? Does control of each pathway require controls over all components of the pathway (e.g., emissions, exposure), or can the pathway be controlled by controlling a subset of these components?

- **Amount of emissions reduction desired/achievable**. What is the overall target for emissions/exposure reduction? How does this relate to risk reduction by the estimates identified above? Will partial reductions result in significant risk reduction, or is it more of an all-or-none situation? What technologies are available to achieve the desired level of risk reduction? How much do the various options cost?

- **Spatial and temporal factors**. Are releases of concern limited to a relatively brief period of time, or do data support the emissions being relatively continuous over a longer period of time? Are the released toxics specific to a single location or are there several wide-spread emission points? What is the fate and transport of the released chemicals? How does background risk relate to the risk reduction strategy?

- **Data gaps and uncertainties**. What are the main sources of uncertainty in the data used in the risk assessment? How do these uncertainties affect the risk management decision? Will more information reduce these uncertainties and can the uncertainty be addressed with available time and resources? Approaches for identifying and managing uncertainties associated with risk assessment are discussed in Chapters 13 and Part VII.

27.5.3 Examine Options for Addressing the Risks

This stage of the risk management process involves identifying potential risk management options and evaluating their effectiveness, feasibility, costs, benefits, unintended consequences, and cultural or social impacts. This process can begin whenever appropriate after defining the problem and considering the context. It does not have to wait until the risk analysis is completed, although a risk analysis often will provide important information for identifying and evaluating risk management options. In some cases, examining risk management options may help refine a risk analysis. Risk management goals may be redefined after risk managers and stakeholders gain some appreciation for what is feasible, what the costs and benefits are, and what contribution reducing exposures and risks can make toward improving human and ecological health.

The Commission noted that stakeholders can play an important role in all facets of identifying and analyzing options. They can help risk managers:

- Develop methods for identifying risk-reduction options;
- Develop and analyze options; and
- Evaluate the ability of each option to reduce or eliminate risk, along with its feasibility, costs, benefits, and legal, social, and cultural impacts.

Chapter 28 provides an overview of community involvement and its role in risk assessment and risk management.

Alternative Solutions to Unique Problems

Project XL, which stands for "eXcellence and Leadership," is a national pilot program that allows state and local governments, businesses, and Federal facilities to develop with EPA innovative strategies to test better or more cost-effective ways of achieving environmental and public health protection. In January 2001, EPA signed the 50th XL Final Project Agreement. Although EPA is no longer accepting proposals for new XL projects, EPA will continue to fulfill each of its commitments under Project XL and will track and monitor the progress of each XL pilot for the duration of the project.
See www.epa.gov/projectxl for more information.

Supplemental Environmental Projects (SEPs) are part of enforcement settlements connected with violations of an environmental statutory or regulatory requirement. As part of the enforcement settlement, a violator voluntarily agrees to undertake an environmentally beneficial project in exchange for a reduction in the penalty. See www.epa.gov/compliance/civil/programs/seps for more information.

27.5.4 Make Decisions about Which Options to Implement

In most risk management situations, decision-makers will have a number of options from which to choose. Which option is optimal depends on the particular situation (and in some cases may be driven by statutory requirements). The following seven are fundamental characteristics of sound risk management decision making:

- Base the decision on the best available scientific, economic, and other technical information;
- Be sure the decision accounts for the problem's multisource, multimedia, multichemical, and multirisk contexts;
- Choose risk management options that are feasible, with benefits reasonably related to their costs;
- Give priority to preventing risks, not just controlling them;
- Use alternatives to command-and-control regulation, where applicable;
- Be sensitive to political, social, legal, and cultural considerations; and
- Include incentives for innovation, evaluation, and research.

Options to be considered for air toxics fall into the following general categories:

- **Regulatory approaches**. Pursuant to various sections of the CAA, Congress has authorized EPA to regulate air toxics. Many S/L/T governments have also authorized agencies to regulate air toxics. Regulatory approaches include enforceable requirements that identified sources must meet (or else be subject to legal action, such as fines) as well as emissions-trading type requirements that focus on controls over sources in total while allowing flexible emissions among individual sources.

- **Voluntary approaches**. EPA and other regulatory agencies are looking beyond regulatory approaches to reduce risks from air toxics. Non-regulatory (voluntary) approaches are frequently the preferred option in a number of cases. Decision-makers at S/L/T agencies may not currently have specific regulatory authority to address specific air toxics problems identified in a risk analysis (particularly in a novel analysis such as a multi-source, community-based risk assessment). The types of problems identified may not lend themselves to regulatory solutions (e.g., they may require changes in the behavior of the exposed population). Voluntary programs may also allow sources to significantly reduce overall risk at much lower cost than various regulatory options. Various incentives such as tax reductions or consumer rebates can be used to encourage voluntary responses.

- **Permits and related authorities**. Permits offer opportunities for both regulatory and voluntary risk-management strategies. Many sources release air toxics to the atmosphere pursuant to permits and related authorities. Permits generally need to be renewed periodically and/or modified if conditions at the source change beyond some specified

amount. This may provide an opportunity to re-write permit conditions so as to reduce high-risk emissions. This might be coupled with voluntary measures or other flexible solutions to result in overall risk reduction (see box). Agencies may also work with emission sources to incorporate voluntary measures or other flexible solutions into the permit.

Example Factors to Consider When Evaluating Risk Management Options

- **Risk reduction benefits to be realized**. Risk management decisions often focus on the *incremental* risk associated with the chemical or other hazard being regulated in the absence of background risks. However, background risk may be important in certain situations. For example, if a monitoring program measures concentrations of air toxics being transported into a given study area that result in risks above an "acceptable" level, no level of emissions control within the study area will be able to reduce risk to an "acceptable" level, and the community may wish to address the incoming air toxics via discussions beyond the local community.

- **Level of uncertainty in the analysis**. In the face of highly uncertain risks, decision-makers have to carefully weigh the consequences of two or more options: making a decision to control emissions or exposures only to find out later that there was little actual risk (e.g., incurring unnecessary "cost" to the community), or making a decision *not* to control emissions or exposures only to find out later that the risks were real and large (e.g., incurring potentially preventable harm to the community).

- **Implementation costs**, both for voluntary approaches (e.g., marketing, process changes, tax incentives) as well as to regulatory agencies, the regulated community, and the general community (consumers).

- **Technical feasibility**. Short of shutting down the emission source altogether, is there an available technology to reduce or eliminate emissions?

- **Legal feasibility**. Does the decision-making body have legal authority to both establish and enforce requirements?

- **Effectiveness/timing**. Will the option provide effective management of the problem within a reasonable time-frame?

- **Political feasibility**. Does the option have the necessary political support?

- **Community Acceptance**. Do the stakeholders buy-in to the proposed risk reduction alternatives?

Each of these factors may be more or less important depending on the context for the risk management decision. For example, the risk manager may be required by statute to weigh economic factors less than technical factors.

27.5.5 Take Actions to Implement the Decisions

Traditionally, implementation has been driven by regulatory agencies' requirements. Businesses and governments (e.g., local municipalities) are generally the implementers. However, the chances of success may be significantly improved when other stakeholders also play key roles. Depending on the situation, action-takers may include public health agencies, other public

agencies, community groups, citizens, businesses, industries, unions/workers, and technical experts. These groups can help:

- Develop and implement a plan for taking action;
- Explain to affected communities what decision was made and why and what actions will be taken; and
- Monitor progress.

27.5.6 Conduct an Evaluation of the Action's Results

At this stage of risk management, decision-makers and other stakeholders review what risk management actions have been implemented and how effective they have been. Evaluating effectiveness involves monitoring and measuring, as well as comparing the actual benefits and costs to estimates made in the decision-making stage. The effectiveness of the process leading to implementation should also be evaluated at this stage. Evaluation provides important information about:

- Whether the actions were successful, whether they accomplished what was intended, and whether the predicted benefits and costs were accurate;
- Whether any modifications are needed to the risk management plan to improve success;
- Whether any critical information gaps hindered success;
- Whether any new information has emerged that indicates a decision or a stage of the process should be revisited;
- Whether the process was effective and how stakeholder involvement contributed to the outcome; and
- What lessons can be learned to guide future risk management decisions or to improve the decision-making process.

27.6 Information Dissemination

The Presidential/Congressional Commission on Risk Assessment and Risk Management noted that effective risk communication is critical to successful implementation of the risk management framework.[3] Risk communication engages both the communicator and the audience in listening and in explaining information and opinions about the nature of risk and other topics that express concerns, opinions, or reactions to risk messages.[5] The Commission made the following recommendations with respect to risk communication:

- The complex and often confusing process of communicating information about risks to diverse affected parties must be improved;

- Decisions about how to allocate resources to reduce risks can be made and explained partly on the basis of risk comparisons;

- The use of "bright lines" which distinguish between contaminant emissions and exposures associated with negligible risk levels and those associated with unacceptable risk levels, needs to be clarified;

- Moving from command-and-control regulation to non-regulatory approaches to risk reduction can increase both efficiency and effectiveness; and

- Criteria for judicial review, a common element in major regulatory actions, should be reaffirmed.

Chapter 29 provides an overview of risk communication and it's role in risk assessment and risk management.

References

1. National Research Council (NRC). 1983. *Risk Assessment in the Federal Government: Managing the Process* (The "Red Book"). National Academy Press, Washington, D.C.

2. National Research Council (NRC). 1994. *Science and Judgment in Risk Assessment* (The "Blue Book"). National Academy Press, Washington, D.C.

3. Presidential/Congressional Commission on Risk Assessment and Risk Management.1997. *Framework for Environmental Health Risk Management* (Final Report, Volume 1). Available at www.riskworld.com/Nreports/1996/risk_rpt.

   Presidential/Congressional Commission on Risk Assessment and Risk Management. 1997. *Risk Assessment and Risk Management In Regulatory Decision-Making* (Final Report, Volume 2). Available at www.riskworld.com/Nreports/1996/risk_rpt.

4. U.S. Environmental Protection Agency. 1995. *Guidance for Risk Characterization*. Science Policy Council, Washington, D.C., February 1995. Available at: epa.gov/osp/spc/rcguide.htm.

5. National Research Council. 1989. *Improving Risk Communication*. National Academy Press, Washington, D.C.

Chapter 28 Community Involvement

Table of Contents

28.1 Introduction 1

28.2 Why is Community Involvement Important? 1

28.3 When to Involve the Community 2

28.4 How to Involve the Community 2
- 28.4.1 Understand Goals, Objectives, and Responsibilities for Effective Community Involvement 4
- 28.4.2 Identify Community Concerns and Interest 5
- 28.4.3 Plan Community Involvement Strategy and Activities 5
- 28.4.4 Identify Possible Tools and Implement Community Involvement Activities 6
- 28.4.5 Provide Opportunity for Continued Public Interaction 7
- 28.4.6 Release of Risk Assessment and Risk Management Documents 8

References 10

28.1 Introduction

Community involvement can be an important aspect of the risk assessment and risk management process. Participation of local stakeholders, at various levels and in various forms, can help ensure a better understanding of the risk assessment results and will promote buy-in to the selected risk reduction strategies. Encouraging and facilitating community involvement also is sometimes required by law.

This chapter provides a broad overview of community involvement in air toxics risk assessment and risk management and identifies helpful references on this topic. Also included throughout this chapter are descriptions of successful air toxics projects and programs where community involvement was a central component of that success.

This chapter describes the key tools, resources, and other considerations for an effective study area-specific approach. It is not, however, intended to provide all the information about conducting community involvement activities. If additional information is needed, contact the community involvement specialist for your agency.

28.2 Why is Community Involvement Important?

When performing an air toxics risk assessment in a particular geographic area, the **community** is often thought of as the people who live within the area of impact of air toxic sources. However, other parties in the area, such as local industry, also may consider themselves part of the community.

In addition to the people who actually live and work in an area, a number of other stakeholders also may have a stake in the community's concerns (e.g., local officials, health professionals, local media). It is often helpful, when dealing with a community, to keep in mind that many different people (not just the people who live there) may have an interest in the risk assessment and management work being undertaken.

As noted above, many laws recognize and accommodate the idea that government decisions should be open to citizen input before a decision is finalized. This is realized through the required public meetings and public comment periods associated with many government actions. For example, the Clean Air Act (CAA) has a number of requirements to provide an opportunity for the public to review and comment on Agency proposals. In some cases, the public is brought in at an even earlier stage.

When risk assessors and risk managers have the opportunity to do so, they should consider including the public as early as possible in the process. Doing so can lead to some very positive benefits. For example, if the community participates early on and throughout the process, they will be in a better position to understand what assessors and risk managers are doing, and there is a better chance that they will believe that the work being done is in their best interest. The process works best when the community appreciates that assessors and managers are working with them and respecting their input (keeping them informed and involved). Ultimately, a community that is involved early on in the process is a community that may be more willing to support the risk assessment process and results. This may, in turn, foster the development of risk reduction strategies the community as a whole can live with and have a stake in.

In contrast, excluding the public from the process may result in community resentment and rejection of even a sound risk assessment and risk management approach. A "guardian-like" attitude toward the community that treats people as unknowledgeable and incapable of meaningful participation does not foster trust and can eventually undermine the process.

In addition to fostering the trust and acceptance of the community, there are many other positive reasons for early and ongoing involvement. For example, important unrecognized sources of emissions and exposure pathways may be identified through the community involvement process. Ultimately, it is important to recognize that community members know their community and understand the types of solutions that will be most accepted – after all, they live there!

28.3 When to Involve the Community

When appropriate, community involvement should begin at the earliest possible stage and span the entire risk and assessment and management process. The level of participation that community members have in some of the more technical phases of the assessment may be tailored to their background, expertise, and interest; however, this does not mean the community cannot serve an important role in the technical phase, as well. The approach taken, as well as the assumptions and limitations of the analysis, should be clearly explained to the community and their input should be valued in return.

For certain CAA requirements, the question of when to involve the public is established by law. For example, in the Title V permitting process the permitting agency must provide a public notice and an opportunity to comment on a draft new or revised permit when:

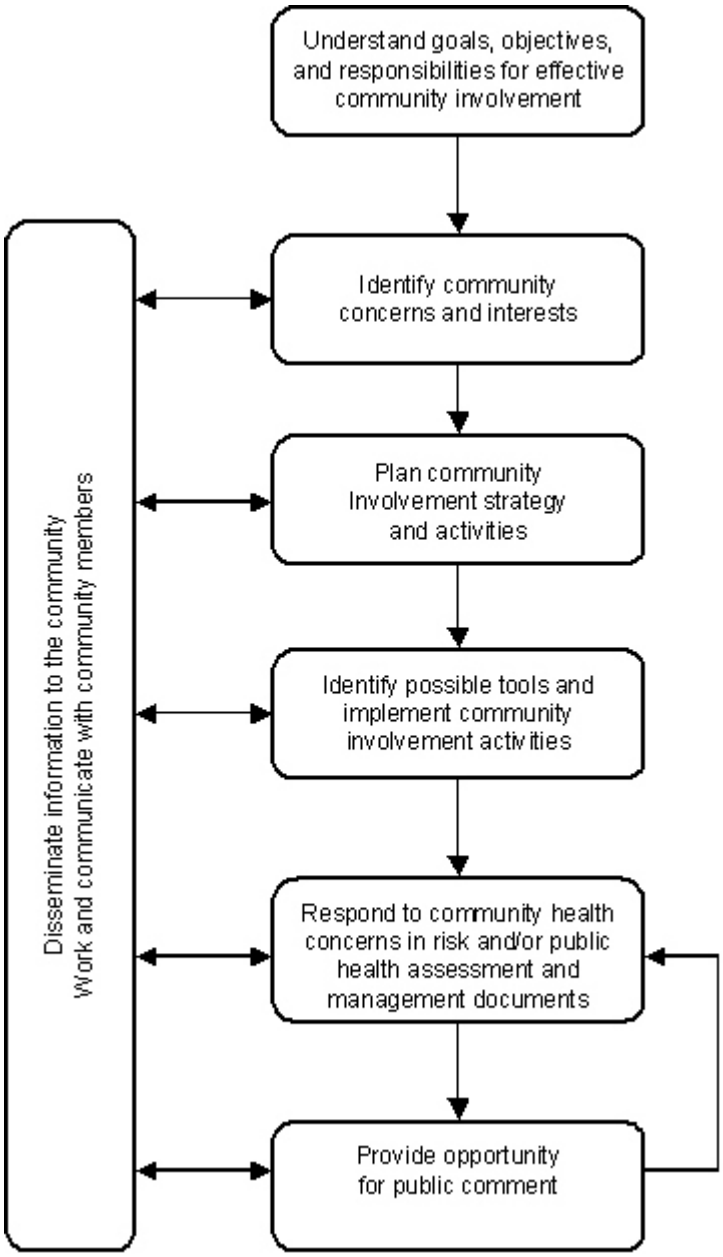
- A facility applies for its first Title V permit;
- A Title V permit is renewed (5 years after issuance);
- The permit is reopened because there is a material mistake in the permit or an update to the permit is needed because of new requirements (review is limited to the part of the permit that is being revised); and
- The facility makes a significant change in its operations and applies for a revision to its permit (review is limited to the part of the permit that is being revised).

For a community-level effort that may include non-regulatory aspects, on the other hand, a community involvement plan will need to be tailored to specific local needs, particularly if the ultimate risk reduction efforts will likely involve voluntary action on the part of industry and/or citizens. As noted above, involving the community at the beginning of and throughout the process will greatly enhance the likelihood that the air toxics risk reduction plan will receive community support (even if the community does not agree with all aspects of the analysis).

28.4 How to Involve the Community

Many different approaches have been developed for involving the community in a risk analysis and management strategy. Exhibit 28-1 illustrates the general framework used both by some programs in EPA and by the Agency for Toxic Substances and Disease Registry (ATSDR). This framework emphasizes the need for involving the community throughout the process.

Exhibit 28-1. ATSDR's Components of Effective Community Involvement



Source: Community Involvement in ATSDR's Public Health Assessment Process (see box of additional references at the end of this chapter)

In identifying community concerns and interests, it often is useful to develop a "conceptual map" of the key organizations and decision-making processes in a community. The map would include information such as who speaks for various parts of the community, who serves in formulating perspectives, and what is the process for obtaining consensus within the community.

TIP: Identify local associations or groups by asking community members, respected "elders," or other associations. This also can go a long way in demonstrating a commitment to involving and mobilizing all stakeholder groups, which helps to build trust and creates a more successful community-involvement process. ***But***, in seeking out community members, do not rely solely on existing community organizations. Very often community members are not well organized or represented by existing groups. Just because there is not an organization or group in the study area does not mean that you can bypass that part of the community.

28.4.1 Understand Goals, Objectives, and Responsibilities for Effective Community Involvement

At a minimum, goals and objectives for community involvement should include the following items. All study areas are different, however, and this list is just a suggested starting point (and may need to be expanded).

- Earning trust and credibility through open and respectful communications;
- Including the community in the design and implementation of risk assessment and risk management;
- Helping community members understand what the process involves;
- Assisting communities in understanding the possible health impact of exposure to air toxics;
- Informing and updating communities about risk management activities; and
- Promoting collaboration between decision-makers, communities, and other agencies and stakeholders when carrying out risk management activities.

To reach these goals and objectives, the following key principles are important:

TIP: Local public health providers, such as county health departments and hospitals can be a key partner in the risk analysis and management processes. These organizations often have resources (staff and funding) that can be used in community health activities. Because they are locally based, involving them as key partners in the process can create strong local leaders to promote sustainable activities once a study is complete.

- **Be aware of confidentiality and privacy issues**. Any personal information that analysts or decision-makers receive from community members should be respected, as appropriate.

- **Be aware of special needs and cultural differences**. When conveying information about air toxics and the risk management process, agencies should be aware of non-English speaking community members and other citizens who may need help in understanding complicated messages. Also, be sure to consider cultural symbolism. There are notable examples of the use of a symbol that is acceptable in one culture but that has an unacceptable meaning in another.

- **Maintain effective communication**. As part of the trust-building process, analysts and risk managers should keep community members informed of progress, opportunities for community involvement, how community input will be used, how community members can help to reduce exposures, and upcoming issues and events.

- **Respect community knowledge and values.** It is important to recognize that community knowledge can provide valuable information for the deliberative processes of risk assessment and risk management and potentially help to address data gaps. It is particularly important to try to understand people's interests (what they care about) during the process (more discussion of this subject is provided in the next section).

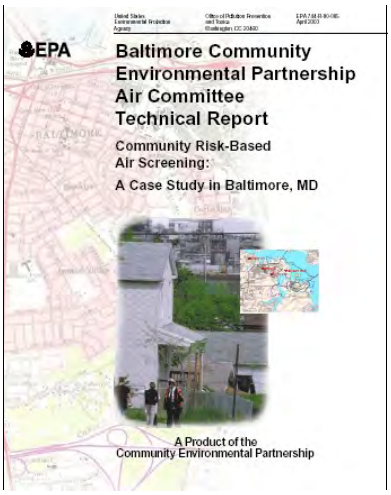
28.4.2 Identify Community Concerns and Interest

One important activity that risk assessors and risk managers can do at the outset of any study is simply to listen to the community. Since their concerns may or may not match those of the assessors and managers, the initial phase of community involvement often involves a fair amount of listening and discussion to help both groups develop a common understanding of what will and will not be studied during the course of the assessment. In those instances where a community concern is outside the scope of what can be studied (e.g., occasional combined stormwater/sewer overflows that cause odors), a willingness on the part of the assessment team to at least help identify resources or connect them to agencies that can address these concerns will go a long way to building trust and credibility. Not listening and not responding to community concerns at the outset may make the process of air toxics assessment and risk reduction more difficult in the long run and may set expectations that are ultimately not met.

28.4.3 Plan Community Involvement Strategy and Activities

Planning a community involvement strategy and activities is one of the most critical components for effective community involvement. The type and nature of communication and involvement activities will depend on (1) the needs and interests expressed by the community during the previous stages, (2) the potential public health issues, and (3) the resources available for communication and involvement activities. Exhibit 28-2 provides a broad list of issues to be considered when developing a community involvement strategy. Not all of these issues must have solutions initially; however, they may need to be addressed eventually.

Community Involvement Example. *Southern Baltimore & Northern Anne Arundel County Community Environmental Partnership (CEP).* In 1996, the residents, businesses, and organizations of five Baltimore, MD neighborhoods joined with local, State, and Federal governments in a CEP to begin a new effort to find ways to improve the local environment and economy. This CEP conducted a comprehensive screening of the cumulative concentration of air toxics from all the industrial and city facilities in and around the neighborhoods and developed a first-for-Maryland survey of cancer incidence at the neighborhood level. Based on this work, the CEP began work with local facilities on pollution prevention. The work of the Baltimore CEP was a learning experience for all of the people who participated. The Partnership tried a lot of new things - some of them worked and some didn't. Lessons learned from this work were carefully documented. The risk screening methodology and lessons learned are being translated into a how-to manual for community use. For more information on this manual and other CEPs, see http://www.epa.gov/oppt/cahp.



28.4.4 Identify Possible Tools and Implement Community Involvement Activities

An enormous number of tools and activities exist that risk assessors and managers can use to encourage community involvement – more than can be described here (the additional resources listed at the end of this chapter, however, should provide most of any team's needs in this regard). They range from the simple phone call, to block parties (at which food may be provided), to the complex mapping of emissions sources and populations. How many and which tools and activities should be used or initiated for a given situation depends on the phase of the risk or public health assessment and management process, the level of community interest, and the degree of hazard a study area poses. The formation of a partnership with stakeholders or community-based coalitions can be an effective way to involve the community, access technical expertise, achieve consensus, leverage resources, and obtain results.

Exhibit 28-2. Issues to Consider When Developing Community Involvement Strategies

Community health concerns:
- How many community members are concerned about the study area?
- What is the level of the community's concern?
- Is the level of community concern higher (or lower) than the actual risk would suggest?
- Are community concerns unknown?
- Would a physician enhance outreach at community meetings?
- Is information/outreach/health education available now or can this wait until reports are generated?

Demographics:
- How many community members are potentially affected?
- Are there any potentially sensitive populations that may be exposed?
- Do socio-demographic data suggest need for additional resources, such as translation?
- How do the community members receive information (e.g., newspaper, radio, word-of-mouth)?

Community interest in the risk assessment and management process:
- How involved in the process would the community like to be?
- How would the community like to be kept updated and informed (e.g., newsletters, e-mails)?
- How many community groups or activist groups are involved? How active are they?
- Should the risk assessment/management team facilitate the creation of a community group if one has not been formed?
- Can information be disseminated at cultural centers? Informal gatherings?

Media support:
- What has the community already heard from the media? Are there misconceptions that need to be dispelled?
- Will media support require more community involvement resources than usual?

Support of the community:
- Are there Native American communities affected by the pollution? Should a relevant agency be involved?
- Does the pollution involve an environmental justice issue, air toxics "hot spot," or other type of special sites?
- What past experiences has the community had with "the government"? Other agencies?
- Is there a higher than average need for resources, such as for more frequent community updates?
- How active will any regional agency representatives or other agencies be in community involvement efforts?

Exhibit 28-2. Issues to Consider When Developing Community Involvement Strategies (Continued)
Public health: • Is the study area a designated public health hazard? Is hazard acute or chronic? • Are environmental health risks largely unknown? • Is the study area considered a high priority? By whom? • Is there already some risk or health outcome results? Are biological data available? • Is a health connection plausible between contaminant exposures and community health concerns? • Are data available for review now ? When will they be available? • Are there toxics reduction steps already in process? **Community culture and setting**: • What are the current community priorities and projects? • What are the community organizations? • Who are the community leaders (unelected)? • What activities constitute community life? **Other**: • How many people on the study area team? Does everyone know their role? • What is the time-frame for report development and communication? • Will any special clearances will be required? At what levels? • Will document or graphics development resources be needed? • Are there schools or locations where community meetings can be held?

28.4.5 Provide Opportunity for Continued Public Interaction

While a risk assessment is underway, primary communication and involvement goals include updating the community on the status of the assessment, obtaining ongoing feedback on the process, obtaining additional information as needed or available from the community for the assessment, and recommending public health actions, if needed, about how community members can reduce exposures. Throughout this process, the risk assessment/management team should continue to listen to community concerns and clearly explain how they will respond to these concerns. The team also should leverage community outreach resources whenever possible. For instance, federal agencies, state health and environmental agencies, local health departments, citizens' advisory groups, and medical advisory groups may have funds for involving community members in the risk assessment/management process. Collaborating with partner organizations can strengthen community outreach depth and coverage.

Generally, community involvement strategies are situation-specific – risk assessment/management teams should determine which community

Non-English Speakers and Other Special Needs?

To ensure the participation of everyone in the community, agencies often use one or more of the following strategies:

- Offer translators and signers at community meetings, and check for wheelchair accessibility.
- Provide additional sessions of meetings that are offered exclusively in the community's secondary language(s).
- Seek out advocates for the severely disabled or others with special needs.
- Provide education and outreach materials in both English and secondary languages.
- Develop understandable and culturally appropriate messages and materials.

involvement strategies are appropriate given the potential seriousness of the risk, the abilities and involvement of the community, and the resources available for communication, training, and outreach. If resources for community outreach are limited, the team may wish to consider how they can best prioritize resources for community involvement.

When resources are limited, the team should look for community outreach opportunities during other community activities, if it would be culturally acceptable. For a determination of cultural acceptability, ask community leaders or "trusted elders."

Finally, some community analyses foster highly interactive relationships with community members and other stakeholders. For example, the risk assessment and risk management teams may establish ad hoc working groups to work on specific issues. These groups may include advisory members from the community or their representatives (e.g., community consultants) and may be more or less formal, as the circumstances require.

28.4.6 Release of Risk Assessment and Risk Management Documents

At the end of the analysis phase, the next stage of community involvement generally begins (i.e., after a draft risk assessment is written). Since the process of data gathering, analysis, and risk assessment preparation can take many months to years, community interest may have decreased significantly. However, once the risk assessment is ready for release, public interest often peaks again. To help ensure a fair and balanced release of information, the risk assessment/management team and their partners may consider using a more formal process to release the risk assessment. For example, the team may release the draft for a period of time for people to read and comment. During the review period, meetings may be held to help describe the results and how the analysis was done. Once the risk assessment document is finalized, there typically is a need to communicate the key results, limitations, and recommendations through a variety of materials including fact sheets, press releases, public meetings, and websites. The risk management strategy may be presented in a similar fashion, with a draft and final document presented to – if not also partly written by – the community.

If an agency or other parties will be conducting any follow-up activities in the area (such as additional environmental sampling or emissions monitoring, cost analyses, health education, health studies), then additional appropriate community involvement may be planned.

Additional References

Public Health Assessment Guidance Manual (2002 Draft Update) describes the process that ATSDR uses to sort through the many hazardous waste sites in the U.S. and to determine where, and for whom, public health actions should be undertaken. Chapter 4 addresses community involvement and communication. See www.atsdr.cdc.gov/HAC/PHAManual/cover.html.

The Annual EPA Community Involvement Conference brings together public participation and community involvement professionals from across all EPA programs, as well as their local, State, Federal, and tribal partners. Conference presentations are designed to emphasize the process of public participation and community involvement by focusing on techniques and approaches used in EPA's national and regional community involvement programs. See epancic.org for upcoming conferences as well as the proceedings of past conferences.

Public Involvement in Environmental Permits: A Reference Guide (2000) at www.epa.gov/permits/publicguide.htm was developed by EPA to help make it easier for state and local agencies to facilitate public participation in environmental permitting decisions for businesses and facilities under your authority. This guide provides basic information about public participation requirements and gives examples under several major permits issued by EPA's air, water, and waste programs. This guide also details what public participation activities are required under these programs, as a minimum, as well as those suggested activities that serve to augment the regulatory requirements.

Air Toxics Community Assessment and Risk Reduction Projects Database at yosemite.epa.gov/oar/CommunityAssessment.nsf/Welcome has been compiled to provide a resource of planned, completed, and ongoing community level air toxics assessments across the country. By sharing information about efforts at the local level to measure, understand, and address air toxics emissions, this database will help ensure that communities designing and implementing their own assessments will be able to build upon past efforts and lessons learned.

Community Involvement in ATSDR's Public Health Assessment Process (2002) provides an overview of how ATSDR works to involve communities in the public health assessment (PHA) process. It describes how ATSDR develops community involvement strategies and plans community involvement activities.

Additional References (continued)

Superfund Community Involvement Web Site provides communities with a range of tools, including guidance documents and other information to increase their understanding of Superfund and the services available to them (e.g., the Technical Outreach Services for Communities Program, Technical Assistance Grants). See www.epa.gov/superfund/action/community/index.htm.

Superfund Community Involvement Handbook (2002) presents legal and policy requirements for Superfund community involvement and additional suggestions for involving the community in the Superfund process. This handbook also provides guidance for community involvement outside of Superfund. See www.epa.gov/superfund/tools/cag/ci_handbook.pdf for more information.

Community Culture and the Environment: A Guide to Understanding a Sense of Place (2002) addresses the social and cultural aspects of community-based environmental protection. The document offers a process and set of tools for defining and understanding the human dimension of an environmental issue. The report, published by EPA's Office of Water, is available on the web from EPA's publication Web site. The report number is EPA/842/B-01/003.

Community Air Screening How To Manual: A Step-by-Step Guide to Using a Risk-based Approach to Identify Priorities for Improving Outdoor Air Quality (to be published in 2003) is being developed by the EPA's Community Assistance Technical Air Team to make air quality assessment tools more accessible to communities. It will present and explain a step-wise process that a community can follow to form a partnership, identify and inventory all local sources of air pollutants, review these sources to identify the hazards and potential risks, and set priorities and develop a plan for making improvements.

References

1. Agency for Toxic Substances and Disease Registry (ATSDR). 2002. Public Health Assessment Guidance Manual (Update): Draft for Public Comment.. Available at: http://www.atsdr.cdc.gov/HAC/PHAManual/cover.html.

Chapter 29 Risk Communication

Table of Contents

29.1 Introduction 1

29.2 Risk Perception 2

29.3 Your Risk Communication Strategy 2

29.4 Risk Comparisons 3

29.5 Implementing Risk Communication Strategies 5
29.5.1 Presentation of Risk Results 5
29.5.2 Working with the Media 8

References 14

29.1 Introduction

The purpose of an air toxics **risk assessment** is to evaluate the magnitude and extent of exposure to air toxics and the potential effects on humans and the environment. Risk assessments aid the process of developing risk management alternatives that minimize risk and maximize environmental benefits.

What is Risk Communication?
Risk communication is the way in which decision-makers communicate with various interested parties about the nature and level of risk, and about the risk reduction strategies to reduce the risk.

The purpose of **risk communication** is to help in the planning of the risk assessment and to convey the results of the risk assessment in a way that effectively supports risk management decisions; this is so that the risk management decisions both meet the goals of the project and provide some comfort level for stakeholders. Good risk communication strategies are a fundamental aspect of developing trust among various stakeholders and the community and are often considered an important first step that can begin even before conducting the risk assessment. Involving the community, establishing and maintaining relationships, and networking with other partners (e.g., agencies, organizations, officials, the media) are key elements in a risk communication strategy. Tailoring communications to the cultural diversity of the community is important because it may help establish the trust necessary to complete a risk assessment that meets all stakeholder and community needs. Risk management rooted in voluntary measures requires effective risk communication to get buy-in.

The subject of risk communication overlaps considerably with related topics discussed in Chapter 13, including EPA's philosophy of transparency, clarity, consistency, and reasonableness (TCCR) as described in its *Policy For Risk Characterization*.[1]

This chapter provides an overview of information developed by the Agency for Toxic Substances and Disease Registry (ATSDR) and other authors to assist the risk assessment team in communicating the context and results of the risk assessment to the public. Readers are encouraged to consult the references at the end of this chapter for a more complete discussion of this important topic. ATSDR also has an excellent website on risk communication resources (See http://www.atsdr.cdc.gov/HEC/primer.html).

Effective Risk Communication:
- Can determine and respond to community concerns;
- Can reduce tension between concerned communities and agency staff; and
- Can explain health risk information more effectively to communities.

ATSDR has published a handbook on risk communication for its staff.[2] Although focused on agency staff, this handbook clearly and effectively outlines the detailed steps necessary in order to develop an effective risk communication plan, and is applicable to all risk assessors and risk management teams. The tools and information in the ATSDR handbook (and discussed in this Chapter) will help the risk assessment team:

- Develop a communication strategy;
- Conduct community outreach and evaluation;
- Develop communication messages; and
- Interact effectively with the news media.

Why is Risk Communication Important?

1. Provides an opportunity to communicate health risks in a caring, concerned, and well-planned manner
2. Involves the community in the risk management process
3. Helps alleviate fear or anger and establish trust

29.2 Risk Perception

If people perceive themselves to be at risk, their perception is unlikely to change even if they are not being exposed or harmed. Elements that affect risk perception include experience, culture, level of education, outrage factors, who is affected/how they are affected (equal treatment), and the level of control exercised on an event or events. People's perceptions of the magnitude of risk also are influenced by factors other than numerical data. According to Covello[3] and other authors:[4]

- Risks perceived to be voluntary are more accepted than risks perceived to be imposed.
- Risks perceived to be under an individual's control are more accepted than risks perceived to be controlled by others.
- Risks perceived to have clear benefits are more accepted than risks perceived to have little or no benefit.
- Risks perceived to be fairly distributed are more accepted than risks perceived to be unfairly distributed.
- Risks perceived to be natural are more accepted than risks perceived to be manmade.
- Risks perceived to be generated by a trusted source are more accepted than risks perceived to be generated by an untrusted source.
- Risks perceived to be familiar are more accepted than risks perceived to be exotic.
- Risks perceived to affect adults are more accepted than risks perceived to affect children.

Two-way risk communication works best. Non-experts want access to information and to gain knowledge. Technical experts and officials also want to learn more about non-experts' interests, values and concerns. The audience includes government, industry, citizens, and both technical and non-technical people. They can all be included in the process as partners.

29.3 Your Risk Communication Strategy - The Overall Plan

In general, planning a risk communication strategy includes the following steps:

- Determine the goals of the communication effort;
- Identify communication restraints;
- Identify the audience(s);
- Identify audience concerns;

- Identify what the audience(s) knows about the issues, both correct information and misinformation;
- Design the message(s) to be sent out to the community;
- Design the "channels"/choose the best methods to reach people;
- Prepare to deliver/present the message;
- Anticipate communication problems;
- Evaluate the program; and
- Modify program as needed.

When working through this process, it is important to know and understand the communication limits and purpose, know your audience, and whenever possible, pretest your message(s). You also should communicate early, often, and fully and remember that for many of the people in your audience, perception is reality.

A good communication strategy also will use tested principles of good presentation, such as the use of simplified language to present important content and the ability to be objective (not subjective) and balanced. Presentations also should not be limited to just one form or just one medium.

Try to use spokespersons who can communicate knowledgeably, honestly, clearly, and compassionately and will listen and deal with specific concerns. Finally, it is important to make sure that the information provided in the risk communication strategy is conveyed to all segments of the audience at a level that they can understand and that the communication materials are honest and up-front about uncertainties. It is often better to say "I don't know" than to hedge.

The ability to establish constructive communication will be determined, in large part, by whether or not the audiences perceive the speaker to be trustworthy and believable. Public assessment of how much we can be trusted and believed is based upon four factors:[1]

- Empathy and caring;
- Competence and expertise;
- Honesty and openness; and
- Dedication and commitment.

29.4 Risk Comparisons

Many successful risk communication efforts have had one major thing in common – a portrayal that puts the calculated exposure risks from an assessment in perspective, with risk ranges the public can easily relate to and understand.

Risk comparisons can help to put risks into perspective. However, irrelevant or misleading comparisons can harm trust and credibility. Thus, while risk comparisons are commonly used, they should be used with caution, because some kinds of risk comparisons are more likely to be perceived as pre-conceived judgments about the acceptability of risks.[1] Guidelines for risk comparisons have been published,[5] and provide rankings of risk comparisons in terms of their acceptability to the community. The highest-ranking comparisons are those that presume a level of trust between the risk communicator and the public, and that consider the factors that people use in their perception of risk. Exhibit 29-1 describes several example risk comparison rankings.

The general rule-of-thumb is to select from the highest-ranking risk comparisons whenever possible. When there is no choice but to use a low-ranking risk comparison, do so cautiously, being aware that it could backfire. The fifth rank, which risk assessors rarely use, consists of comparisons of unrelated risks (e.g., involuntary vs. voluntary risks). These comparisons have been found to be very problematic. For example, the risk of driving without a seat belt is a voluntary risk, while exposure to air toxics is generally considered involuntary by community members. Covello et al. [5] provide specific examples of each of the comparison ranks, as associated with a manufacturing facility (http://www.psandman.com/articles/cma-4.htm). Risk comparison charts are also provided in Appendix B of that document (http://www.psandman.com/articles/cma-appb.htm), although the authors do not recommend their use in public presentations.

Exhibit 29-1. Relative Acceptability of Risk Comparisons

- **First-rank risk comparisons (most acceptable)**
  - Of the same risk at two different times
  - With a standard
  - With different estimates of the same risk
- **Second-rank comparisons (less desirable)**
  - Of the risk of doing something versus not doing it
  - Of alternative solutions to the same problem
  - With the same risk experienced in other places
- **Third-rank comparisons (even less desirable)**
  - Of average risk with peak risk at a particular time or location
  - Of the risk from one source of an adverse effect with the risk from all sources of the same effect
- **Fourth-rank comparisons (marginally acceptable)**
  - With cost; or one cost/risk ratio with another
  - Of risk with benefit
  - Of occupational risk with environmental risk
  - With other risks from the same source
  - With other specific causes of the same disease, illness, or injury
- **Fifth-rank comparisons (rarely acceptable – use with caution)**
  - Of risks that may seem unrelated to community members (e.g., smoking, driving a car, lightning)

EPA has included risk comparisons in some air toxics analyses. For example, the results section of EPA's *National-Scale Air Toxics Assessment* (http://www.epa.gov/ttn/atw/nata/) discusses general U.S. background risks from air toxics, originating from both mobile sources and other background sources:

- **Mobile Sources**. For on-road and non-road mobile sources, EPA estimates that more than 100 million people live in areas of the U.S. where the combined upper-bound lifetime cancer risk from all air toxics compounds exceeds 10 in a million. This risk estimate is dominated by the emissions of benzene, formaldehyde, acetaldehyde, and 1,3 butadiene. Regarding effects other than cancer, acrolein emissions are estimated to lead to exposures above the

reference concentration (i.e., a hazard quotient above 1.0) for approximately 200 million people in the U.S. EPA expects that in 2007, existing standards affecting emissions of air toxics compounds from new vehicles will reduce exposure from on-road sources by about 50 percent from 1996 levels, and that substantial reductions also will occur for non-road emissions.

- **Background Sources**. EPA estimates that combined upper-bound cancer risks associated with air toxics compounds from background sources are less than 100 in 1 million throughout the U.S. However, the entire U.S. population is estimated to exceed an upper-bound cancer risk level of 10 in a million due to background sources alone (note that in this study background concentrations include both uncontrollable emissions [e.g., persistent historic emissions, international or global pollutant transport, contributions from natural sources and emissions that can be controlled such as long-range pollutant transport within the U.S.]).

29.5 Implementing Risk Communication Strategies

In order to implement risk communication strategies, agencies may need to plan approaches to public presentations and working with the media. The purpose of communication with the public is to inform, educate, and enhance cooperative problem solving and conflict resolution.

29.5.1 Presentation of Risk Results

Risk communication strategies also consider the meaning of the information (e.g., will the listener understand how to use the information in forming opinions, making decisions, and taking actions). When risks are calculated for air toxics and the risk results are presented to the public, the community may not be familiar with quantitative risk data and what it means for them. In order to prevent panic and to encourage participation in and buy-in of risk management decisions, risk communication strategies are developed that not only reassure the community, but also explain the potential risks and uncertainties in an understandable, clear, and honest way. Effective communications also provide information in a community-compatible language or form. For example, if the community speaks Spanish, then the communications could be in Spanish as well as English. Similarly, if the community includes Native Americans, the communications could be in the appropriate language and employ appropriate symbolism. The effective communication of risks will allow stakeholders to better participate in management decisions that weigh the benefits of different alternatives against the costs of achieving "acceptable" levels of risks and the costs of disruptions associated with implementation.

When developing messages, it is important to consider the following questions:

- What does the community already know?
- Is this information factual?
- What does the community want to know?
- What does the community need to know?
- Can the information be misunderstood?

When developing a public education campaign, it is generally most effective if the campaign highlights no more than three primary messages. More than three primary messages may convolute the focus of the education campaign. Those developing public education campaigns

may wish to test their risk communication messages with trusted audience members before releasing them to the public. This can ensure that the messages are on-target and help avoid community objections that decision-makers may not have anticipated. It also is important to ensure that the message is culturally attuned and fits the language needs of the audience. "Outrage reducers" are outlined by risk communication specialist Peter Sandman (www.petersandman.com).

When developing risk-communication messages, decision-makers should (1) review the concerns and worries of their audience; (2) cover WHO, WHAT, HOW, WHEN, WHERE and WHY; and (3) develop messages that are consistent with their actions.

Different messages and channels may be needed for different audiences. To communicate effectively, the risk communicator should try to understand the audience's values, concerns, and perceptions. Credibility is enhanced by the degree to which the risk communicator correctly identifies, anticipates, and empathizes with the specific concerns of his or her audience(s), which may include:

- Health concerns;
- Safety concerns;
- Environmental concerns;
- Economic concerns;
- Aesthetic concerns;
- Lifestyle/cultural concerns;
- Data and information concerns;
- Fairness/Equity concerns;
- Trust and credibility concerns;
- Process/value concerns (e.g., who makes decisions and how); and
- Risk management concerns.

Audiences may include:

- Environmental groups;
- Civic organizations;
- Professional and trade organizations;
- Educational and academic groups;
- Religious groups;
- Other government agencies;
- Neighborhood/school organizations;
- Industries; and
- Other organizations.

It may be worthwhile to develop audience profiles for key audiences. Profiles describe the members of the audience, whom they trust and go to for information (decision-makers can seek these people out for advice on communicating with the community), what their prevailing attitudes and perceptions are, and what concerns and worries motivate their actions.

It is important to clearly communicate scientific information and uncertainty:

- Provide all information possible, as soon as possible;
- Communicate when there is progress being made;
- Maintain your relationship with the community;
- Be honest about what you do not know;
- Explain how you will work together to find the answers;
- Help the audience understand the process behind your findings;
- Avoid acronyms and jargon;
- Carefully consider what information is necessary; and
- Use familiar frames of reference to which the audience can relate.

Public interactions may also include availability sessions, informal discussions, or poster sessions. Presentations can occur in a variety of venues some of which are better suited than others to different situations. Determining the best channels for your message depends on understanding when to use which tool and knowing how the community prefers to receive information. Message delivery channels include:

1. **Presentations**: Speeches to public groups. Benefit: offers the audience a chance to ask questions; reaches many people at one time. Limitations: if poorly presented, can distort community perception; cannot sufficiently address individual concerns; can become argumentative or confrontational.

2. **Open Houses/Availability Sessions**: Informal meeting where public can talk to staff on a one-to-one basis. Benefit: allows for one-to-one conversation; helps build trust and rapport.

3. **Small Group Meetings**: Sharing information with interested community members and government officials. Benefit: allows two-way interaction with the community. Limitations: may require more time to reach only a few people; may be perceived by community groups as an effort to limit attendance; be sure your information is identical or you may be accused of telling different stories to different groups.

4. **Briefings**: Can be held with key officials, media representatives, and community leaders; generally not open to the public. Benefit: allows key individuals to question risk assessment staff before release of public information. Limitations: should not be the only form of community communication; bad feelings may arise if someone feels that they were left off the invite list.

5. **Community mailings**: Sends information by mail to key contacts and concerned/involved members of the community. Benefit: delivery of information quickly; may require less planning than a meeting. Limitation: no opportunity for feedback.

6. **Exhibits**: Visual displays to illustrate health issues and proposed actions. Benefits: creates visual impact. Limitations: one-way communication tool, no opportunity for community feedback.

7. **Fact Sheets**: To introduce new information. Benefit: brief summary of facts and issues; provides background for information discussed during a meeting. Limitations: one-way communication tool; needs to be well-written and understandable.

8. **Newsletters**: To inform community of ongoing activities and findings. Benefit: explains findings; provides background information. Limitations: can backfire if community members do not understand or misinterpret contents.

9. **News Release**: Statement for the news media to disseminate information to large numbers of community members. Benefit: reaches large audience quickly and inexpensively. Limitations: may exclude details of possible interest to the public; can focus unneeded attention on a subject.

10. **Public Meetings**: Large meeting open to the public where experts present information and answer questions and community members ask questions and offer comments. Benefit: allows community to express concerns and agency to present information. Limitations: can intensify conflicts, rather than resolve controversies.

Presentations require a careful balancing act between effectively conveying key messages and avoiding a range of pitfalls. Important "Dos" and "Don'ts" to avoid presentation pitfalls, are outlined in Exhibit 29-2.

29.5.2 Working with the Media

The media can be a primary source of information on risks to the public. Effective news media relations have many benefits, complementing other communication efforts. What people read, see, or hear in news coverage can lend credibility to agencies associated with air toxics risk assessment, and can help to make it a familiar topic for public discussion. News coverage can inform people about air toxics issues and help them ask appropriate questions. Skill in media relations can help risk communications avoid or dispel rumors, respond to criticism, defuse controversy, and even turn adversity to advantage.

News coverage is crucial to engaging the attention of decision-makers and earning the support of opinion leaders. Also, because the news media pay distribution costs, helping journalists cover the issues is a cost-effective way to communicate.

The best approach to the media, as with the public, is to be open and honest, provide information tailored to the needs of each type of media, such as graphics and other visual aids, and provide background material. Journalists also should welcome such materials as fact sheets, press kits, and lists of experts. Establishing an information center also can be an effective way to make materials available to the news media (and to the general public). It also is very important that the material and discussions you have with the media clearly articulate the messages that you want to find their way into print or onto the TV or radio.

Like other communication efforts, working with the news media is done best when it is based on a strategy and follows a systematic process. A good strategy seeks opportunities to match the goals and objectives of the organization with the interests of journalists. As in other communication strategies, assessing the needs of the audience – journalists – is important to reaching them effectively.

After you determine that the rules of your organization concerning contacts with the media have been met, here are a few suggestions on how to deal with news reporters:

- When a reporter calls, be sure to get a name and media affiliation; if what the reporter wants is not clear to you, ask for a clear explanation; if you are uneasy with a reporter's query, decline in a friendly way to continue the conversation.

- Reporters are often under deadline pressure, but you can take enough time to respond effectively; don't get pressured into hasty comments that might backfire.

- Do not hesitate to ask for more information about a story before responding to a request for an interview.

In working with journalists, it is vital to develop good interpersonal relationships. How can you do that? One rule of thumb followed by experienced practitioners is to adhere to the "Five Fs" – Fast, Factual, Frank, Fair, and Friendly (Exhibit 29-3).[6]

- **Interviews**. Frequently, the best way to get a message out is through an in-person interview. You should generally assume that all statements you make are "on the record." Exhibit 29-4 outlines some techniques to prevent poor transmittal of your message.

- **Press Releases**. Press releases may not be an effective way to transmit a message. However, in some cases, releases that are targeted to particular media outlets and purposes can be useful. For example, the publication of a report on air toxics risk might be newsworthy and of concern to the community, and thus would be sent to local community newspapers. Remember that your press release should emphasize, upfront, the messages that you want to get out to the public.

- **Other Platforms**. You may have the opportunity to communicate your message through other platforms such as:
  - Letters to the Editor. Keep them short, to the point, and prompt.
  - Commentaries. Radio broadcasts and newspapers print a number of opinion pieces each day. Bear in mind that submissions are numerous, acceptances rare.
  - Talk Radio (and TV). Talk shows may request experts to address various environmental issues.

Exhibit 29-2. Presentation Dos and Don'ts
• **Pitfall**: **Jargon** **Do**: Define all technical terms and acronyms. **Don't**: Use language that may not be understood by even a portion of your audience. • **Pitfall**: **Humor** **Do**: Direct it at yourself, if used. **Don't**: Use it in relation to safety, health, or environmental issues. • **Pitfall**: **Negative Allegations** **Do**: Refute the allegation without repeating it. **Don't**: Repeat or refer to them. • **Pitfall**: **Negative Words and Phrases** **Do**: Use positive or neutral terms. **Don't**: Refer to national problems (problems unrelated to the issue at hand), i.e., "This is not Love Canal." • **Pitfall**: **Reliance on Words** **Do**: Use visuals to emphasize key points, but be culturally correct for the audience. **Don't**: Rely entirely on words. • **Pitfall**: **Temper** **Do**: Remain calm. Use a question or allegation as a springboard to say something positive. **Don't**: Let your feelings interfere with your ability to communicate positively. • **Pitfall**: **Clarity** **Do**: Ask whether you have made yourself clear. **Don't**: Assume you have been understood. • **Pitfall**: **Abstractions** **Do**: Use examples, stories, and analogies to establish a common understanding, but test them out first to make sure they are clear, make your point, and are culturally acceptable. • **Pitfall**: **Nonverbal Messages** **Do**: Be sensitive to nonverbal messages you are communicating. Make them consistent with what you are saying. **Don't**: Allow your body language, your position in the room, or your dress to be inconsistent with your message. • **Pitfall**: **Attacks** **Do**: Attack the issue. **Don't**: Attack the person or organization. • **Pitfall**: **Promises** **Do**: Promise only what you can deliver. Set and follow strict orders. **Don't**: Make promises you can't keep or fail to follow up. • **Pitfall**: **Numbers** **Do**: Emphasize performance, trends, and achievements. **Don't**: Focus on or emphasize large negative numbers.

Exhibit 29-2. Presentation Dos and Don'ts (continued)

Pitfall: **Guarantees**
Do: Emphasize achievements made and ongoing efforts.
Don't: Say there are no guarantees.

Pitfall: **Speculation**
Do: Provide information on what is being done.
Don't: Speculate about worst cases.

Pitfall: **Money**
Do: Refer to the importance you attach to health, safety, and environmental issues; your first obligation is to public health.
Don't: Refer to the amount of money spent as a representation of your concern.

Pitfall: **Organizational Identity**
Do: Use personal pronouns ("I," "we").
Don't: Take on the identity of a large organization.

Pitfall: **Blame**
Do: Take responsibility for your share of the problem.
Don't: Try to shift blame or responsibility to others.

Pitfall: **"Off the Record"**
Do: Assume everything you say and do is part of the public record.
Don't: Make side comments or "confidential" remarks.

Pitfall: **Risk/Benefit/Cost Comparisons**
Do: Discuss risks and benefits carefully (consider putting them in separate communications).

Pitfall: **Risk Comparison**
Do: Use them to help put risks in perspective.
Don't: Compare unrelated risks.

Pitfall: **Health Risk Numbers**
Do: Stress that true risk is between zero and the worst-case estimate. Base actions on federal and state standards, when possible, rather than risk numbers.
Don't: State absolutes or expect the lay public to understand risk numbers.

Pitfall: **Technical Details and Debates**
Do: Focus your remarks on empathy, competence, honesty, and dedication.
Don't: Provide too much detail or take part in protracted technical debates.

Pitfall: **Length of Presentations**
Do: Limit presentations to 15 minutes.
Don't: Ramble or fail to plan the time well.

Source: ATSDR Risk Communication Primer[2]

Exhibit 29-3. The "Five Fs" of Media Relations
Fast. Respect journalists' deadlines. If a journalist telephones for information, return the call immediately, even if it is past normal office hours. A phone message returned the next day is often too late. By then, the story already may have been aired or printed. **Factual**. Be factual, and make the facts interesting. Stories are to be based on facts. Journalists also appreciate a dramatic statement, creative slogan, or personal anecdote to help illustrate your point. Give the source of any facts and statistics provided. **Frank**. Be candid. Never mislead journalists. Be as open as possible and respond frankly to their questions. As long as there is an explanation of the reason, most journalists will understand and respect a source even if he or she is not able to answer a question completely or at all. **Fair**. Organizations should be fair to journalists if they expect journalists to be fair to them. Favoring one news outlet consistently, for example, will lose the confidence of the others. **Friendly**. Like everyone else, journalists appreciate courtesy. Remember their names; read what they write; listen to what they say; know their interests; thank them when they cover the issues in a factual, unbiased way.

Exhibit 29-4. Interviewing Techniques
• Always think carefully before you answer a question. People often ramble - and say something they wish they hadn't if they answer too quickly. Take a moment to consider what you want to say. If you need more time, ask for the question to be repeated. • Don't talk just to keep a conversation going with a reporter. Experienced reporters will be silent because often people they interview will talk to fill awkward voids and then say something they don't mean to say. • Ask the reporter to make your affiliation clear in the story. • Listen carefully to questions and respond clearly. Avoid jargon. If you have a key idea that you want to get across, repeat it several times, perhaps using different words. This is especially useful for broadcast: no matter how the tape is edited, you will make your point. • Don't hurry: speak slowly, and in short, concise sentences. State your position in simple, easy-to-understand language. Use everyday examples and analogies, when possible. • Never talk down to a reporter. You are partners in getting your message across. Arrogance will come across negatively to an audience. An "attitude" can turn an interview into a confrontation. • Don't lose your temper! No matter how antagonized you feel, recognize that this can be a tactic to get you to say something you do not wish to say. • If you don't know the answer to a reporter's question, or cannot answer, just refrain from answering. A lie or bad guess will return to haunt you. You will lose credibility. • Some reporters may ask to tape an interview over the telephone. This is a common practice for radio reporters to obtain "sound bites" and to get accurate quotes. The reporter should inform you of the taping before it begins. Do not repeat an allegation – it could be taken out of context.

Additional Suggested References

Calow, P. 1997. H*andbook of Environmental Risk Assessment and Management.* Blackwell Publishers.

Crawford-Brown, D. 1999. *Risk-based Environmental Decisions: Culture and Methods.* Kluver Academic Publishers.

Johnson, B.B., Sandman, P.M., and Miller, P. 1992. Testing the Role of Technical Information in Public Risk Perception by RISK. *Issues in Health and Safety*, Fall 1992:341-364.

Lundgren, R.E. 1994. *Risk Communication: A Handbook for Communicating Environmental, Safety, and Health Risks.* Battelle Press, Columbus, OH.

Langford, Ian. 2002. An existential approach to risk perception. *Risk Analysis* 22(1):101-120.

U.S. Environmental Protection Agency. 1992. *Air Pollution and the Public: A Risk Communication Guide for State and Local Agencies.* EPA 450/3-90/025.

For an additional list of risk communication references, see http://www.psandman.com/articles/cma-bibl.htm.

References

1. U.S. Environmental Protection Agency. 1995. *Policy for Risk Characterization* ("Browner Memorandum"). Science Policy Council, Washington, DC., March 1995. Available at: http://64.2.134.196/committees/aqph/rcpolicy.pdf

2. Agency for Toxic Substances and Diseases Registry (ATSDR). 1994. *Tools and Techniques for Effective Health Risk Communication*. This is an update of the ATSDR *Primer on Health Risk Communication Principles and Practices*, October 1994. Available at: http://www.atsdr.cdc.gov/HEC/primer.html#EARNING

3. Covello, V.T., Sandman, P. 2001. Risk communication: Evolution and Revolution, in Wolbarst A. (ed.). *Solutions to an Environment in Peril*. John Hopkins University Press, Baltimore, MD: pp. 164-178. Available at: http://www.phli.org/riskcommunication/article.htm

4. Fischhoff B, Lichtenstein S, Slovic P, Keeney D. 1981. *Acceptable Risk*. Cambridge University Press, Cambridge, Massachusetts.

5. Covello, V.T., Sandman, P.M., Slovic, P. 1988. *Risk Communication, Risk Statistics and Risk Comparisons: A Manual for Plant Managers*. Chemical Manufacturers Association, Washington, D.C., 1988. Available at: http://www.psandman.com/articles/cma-0.htm

6. Cutlip, S.M., Center, A.H., and Broom, G.M. 1985. *Effective Public Relations*. Prentice-Hall, Englewood Cliffs, New Jersey.

PART VI

SPECIAL TOPICS

Introduction to Part VI

Part VI of this Reference Manual provides an overview of three special topics related to air toxics risk assessment.

- Public Health Assessment (Chapter 30) provides an overview of the process by which public health agencies may evaluate the public health implications posed by the emissions from air toxic sources in a community. The public health assessment, if performed, is a complementary process to risk assessment.

- Probabilistic Risk Assessment (Chapter 31) discusses the process by which probability distributions are used to characterize variability or uncertainty in risk estimates, a process aimed at describing risks as a distribution (or range) of potential outcomes.

- Use of Geographical Information Systems (GIS) in Risk Assessment (Chapter 32) provides an overview of the software and geographic data that allow efficient storage, analysis, and presentaiton of spatially explicit and geographically referenced information that can help in the process of conducting risk assessments and reporting results

Chapter 30 Public Health Assessment

Table of Contents

30.1 Introduction 1

30.2 History of Public Health Assessment 2

30.3 Relationship of Public Health Assessment to Risk Assessment 3

30.4 What Is Public Health Assessment? 4

30.5 How Is a Public Health Assessment Conducted? 6
- 30.5.1 Conduct Scoping 6
- 30.5.2 Obtain Study Area Information 7
- 30.5.3 Community Involvement/Outreach/Response to Community Concerns 7
- 30.5.4 Exposure Evaluation 8
- 30.5.5 Health Effects Evaluation 9
- 30.5.6 Draw Public Health Conclusions 12
- 30.5.7 Recommend Public Health Actions 13
- 30.5.8 Prepare PHA Documents 13

References 14

30.1 Introduction

An adjunct to conducting air toxic risk assessments is public health assessments, which uses public health tools (e.g., health questionnaires, epidemiology) to investigate the incidence and prevalence of disease and to find out the current or past health of individuals. While public health methods are not always used for air toxics risk assessments, they can provide useful information to answer the question of whether there is evidence that there is a public health concern, particularly if disease rates are elevated in the assessment area.

Air toxics risk assessment, the main topic of this manual, focuses on assessing the potential risk that people have for experiencing adverse health effects from exposure to air toxics. The outcome of a risk assessment is a statement about the likelihood that exposure may result in disease (e.g., the probability of people developing cancer). The risk assessment process links the potential exposures to emissions from (often) specific sources to the likelihood of disease occurring.

However, in any community, concerns about more than just estimates of the likelihood of risk often come up. For example, communities where risk assessments are being performed often express concern about current health effects that may have resulted from past exposures. Questions like "was my cancer caused by air pollution" are often on the minds of people who live where an air toxics risk assessment is being performed.

The risk assessment process, while a powerful predictive tool for evaluating public health impacts from air pollution, is not amenable to answering these types of questions. Nevertheless, questions about disease and past exposures will inevitably come up as the air toxics risk assessment study moves forward. The risk assessment and risk management team will almost always have to explain that their assessment tool (risk assessment) is not being used to answer questions about existing cases of disease.

To help risk assessors and other stakeholders respond to these types of questions, this chapter provides information on a complementary process to risk assessment called **Public Health Assessment** or **PHA**. It is taken largely from the ATSDR *Public Health Assessment Guidance Manual*.[1] A PHA for air toxics is an analysis and statement of the public health implications posed by a source or group of sources of air toxics on a given geographic area. It usually is conducted by a public health agency such as the Agency for Toxics Substances and Disease Registry or ATSDR (a federal Agency within the Centers for Disease Control and Prevention) or one of their partner state or local public health agencies. PHAs *are not* generally performed by EPA or state, local, or tribal air agencies since PHAs often rely on specialized medical and epidemiological expertise and due to the difficulty facing these agencies in obtaining and reviewing medical information for individuals. PHAs are normally performed:

- In response to a request by concerned community members or physicians;
- In response to a real or perceived increase in a health problem noted during routine disease surveillance systems; and/or
- As part of a broader program such as a proactive analysis of region-specific air quality.

The types of air toxics assessments most likely to include a PHA are those where the pollutants have a clearly identifiable effect, where the exposure is relatively widespread, or where there is a high level of public concern. A PHA will not necessarily be needed every place an air toxics risk assessment is performed. However, the use of the PHA process, in conjunction with the risk assessment process, is becoming a more common practice for the purpose of providing holistic evaluations of air toxics impacts on communities.

PHAs are performed by ATSDR at each Superfund site on the National Priorities List. ATSDR also performs PHAs when petitioned. The term **public health assessment** (**PHA**) as used here, refers to a broad range of assessment types – from screening-level health consultations to comprehensive epidemiological assessments – that are commonly performed by ATSDR in its work. The PHA process, while commonly thought of as a Superfund-related activity, is amendable to a wide range of exposure scenarios, including the evaluation of air toxics impacts at the community level.

A PHA may involve an assessment of relevant **environmental data**, **health outcome data** (e.g., cancer statistics), and **community concerns** generally associated with a study area where air toxics are or have been released. A PHA identifies populations living or working on or near areas for which more extensive public health actions or studies are indicated and is generally more qualitative, more focused on actual, measurable harm, and past and current exposures.

This chapter describes the history of PHAs, what they are, how they compare to and work in concert with risk assessments, and how they are conducted. Several case studies are included to help illustrate the diversity of PHAs and how they compare with and are used with risk assessments.

30.2 History of Public Health Assessment

PHA as a tool for characterizing and protecting the health of a society can be traced back thousands of years. The ancient Babylonians, Egyptians, Greeks, and Romans were among the first known civilizations to describe associations between diseases and sources such as place, water conditions, climate, eating habits, and housing. One of the first documented public health "assessments" (though later proven incorrect) connected the presence of "bad air" around swamps and marshes with the prevalence of malaria, one of the world's most devastating diseases. (It was determined later that the prevalence of malaria was associated not with air, but with mosquitos, the transmission vector for the disease, which breed in standing water associated with those places.) Infectious diseases continued to dominate public health concerns until the industrial revolution, although the problems of poor urban air quality from the use of coal were well documented as early as the end of the 16th century.

The earliest "bad air"?

The modern use of PHA for air toxics in the U.S. probably began in the mid-1900s in response to events such as the incapacitating smog episodes in Los Angeles in the 1940s, the polluted air inversion that killed 20 people in Donora, Pennsylvania in 1948, and the atmospheric nuclear weapons tests in Nevada in the 1950s. Myriad state and local public health agencies shouldered much of the burden of air pollutant health assessment at first. Then, at the federal level, the Federal Air Pollution Control Act of 1955 authorized the Public Health Service (PHS) to conduct

research and technical assistance and work towards a better understanding of the causes and effects of air pollution.

In 1980, ATSDR was created specifically to conduct PHAs at hazardous waste (Superfund) sites. That role has expanded over time to address additional pollution sources, including air toxics. ATSDR is not a regulatory agency like EPA, but rather is a public health agency that conducts assessments and makes recommendations to EPA and others when specific actions at study areas in question are needed to protect the public's health. ATSDR conducts PHAs when petitioned by concerned community members, physicians, state or federal agencies, or tribal governments. State and local public health agencies also play an important role with regard to PHAs for air toxics and other hazards.

30.3 Relationship of Public Health Assessment to Risk Assessment

Both the PHA and the quantitative risk assessment address the potential human health effects of environmental exposures, but they use different approaches and have different purposes. As illustrated in Exhibit 30-1, the PHA tends to be less quantitative than the risk assessment and to focus more on actual past and current exposures. The PHA evaluates observed health outcome and related data (e.g., cancer clusters, breathing problems, toxics residues in biologic samples) to determine whether rates of disease or death are or could be elevated in a community and, if so, whether these outcomes are due to a specific source. The risk assessment, on the other hand, starts with a specific source and evaluates estimated potential health outcomes, or risks. The PHA's subsequent conclusions generally complement the risk assessment process and help inform the decisions that the state, tribal, or local agency is reaching about a given study area. Similarly, the risk assessment provides considerable data to the PHA.

In addition to its focus on health outcome data, such as cancer or asthma incidence, the PHA also helps put community-provided data and information and community concerns into perspective, which in turns helps both (1) the community better understand whether they have been exposed to hazardous substances and, if so, what that means in terms of possible health outcomes, and (2) the decision-maker better determine what needs to be done to prevent or further study these exposures (e.g., emissions reductions, health education, biologic monitoring).

The PHA may use similar techniques to those of the quantitative risk assessment, but primarily as tools either to clearly rule out the existence of public health hazards, to determine that a clinical disease is really likely in the community, or to identify areas for additional study. At a minimum, the PHA helps to identify a baseline in the level of disease in a community so that later studies will have a basis for comparison.

Exhibit 30-1. PHAs and Risk Assessments: Differences and Similarities

In a PHA...	In a risk assessment...
OVERALL	
More qualitative More community involvement Conduct less frequently	More quantitative Less community involvement Conducted more frequently
EXPOSURE ASSESSMENT	
Similar for air sampling and modeling Biomonitoring possible Past, current/future	Air sampling Fate/transport modeling Future/hypothetical
TOXICITY ASSESSMENT	
Similar (for health effects screening)	Similar (for toxicity)
CHARACTERIZATION	
Margin for exposure comparisons Public health implications Needed public health actions Informs the risk assessment	Modeled risk Informs the PHA

30.4 What Is Public Health Assessment?

A PHA is an evaluation of relevant **environmental data**, **health outcome data**, and **community concerns** associated with a study area where hazardous substances have been released. A PHA identifies populations living or working on or near areas for which more extensive public health actions or studies are indicated.

PHAs can range from simple to complex, with the former activity often termed a **health consultation** rather than PHA. This more simple form generally is conducted in response to a

ATSDR Definition of PHA

The evaluation of data and information on the release of hazardous substances into the environment in order to assess any [past], current, or future impact on public health, develop health advisories or other recommendations, and identify studies or actions needed to evaluate and mitigate or prevent human health effects (42 *Code of Federal Regulations*, Part 90, published in 55 *Federal Register* 5136, February 13, 1990).

specific question or request for information pertaining to a hazardous substance or facility. It often contains a time-critical element that necessitates a rapid response. More complex forms of a PHA can involve a wide geographical area, many pollution sources, and take months or years to complete.

Understanding and responding to study area-specific community health concerns is an important part of the PHA process. These investigations can be conducted to confirm case reports, determine an unusual disease occurrence, and explore potential risk factors. One frequently cited concern is the **disease cluster** – the occurrence of a specific disease or condition above the expected number for a given geographic location and time period (e.g., the high incidence of leukemia in a given area). The health agency needs to learn what people in the area know about a source and source-related exposures and what concerns they may have about its impact on their health. Therefore, starting early in the assessment process, the health agency generally gathers information and comments from the people who live or work near the source(s), including area residents, civic leaders, health professionals, and community groups. Throughout the PHA process, the health agency should communicate with the public about the purpose, approach, and results of its public health activities.

The PHA process is iterative and dynamic and may lead to a variety of products or public health actions. The findings may be communicated in public health assessment or public health consultation documents, which serve as an aid for developing additional public health actions. The audience for such products often includes environmental and public health agencies, communities, and the public health agency itself.

During the course of the PHA process, the public health agency may identify the need to prevent or better define exposures or illnesses in a particular community. The agency's response to such a need might include:

- Issuing a **public health advisory** (if there is an urgent health threat);
- Initiating an **exposure investigation** (to better define study area exposures);
- Recommending a **health study** (to identify elevated illness or disease rates in a community); and/or
- Conducting **health education** (for the study area community or health professionals within the community).

The PHA process also can serve as a triage mechanism, enabling the public health agency to prioritize and identify additional steps needed to answer public health questions. The science of environmental health is still developing, and sometimes information on the health effects of certain substances is not available. When this occurs, rendering certain questions unanswerable by the available literature, the public health agency will suggest what further research studies and/or health education services are needed.

30.5 How Is a Public Health Assessment Conducted?

PHAs generally are conducted by public health agency assessors, often supported by a multi-disciplinary team of scientists, health communication specialists, health educators, and/or medical professionals. The health agency solicits and evaluates information from other local, state, tribal, and/or federal agencies; parties responsible for operating sources at a particular study area; and the community. All of these stakeholders play an integral role in the PHA process. The public health agency promotes a team approach to ensures that information used in the assessment is accurate and up-to-date, ensure that community concerns are identified and addressed, and fosters cooperative efforts in implementing recommendations and public health activities.

Many technical resources exist that provide details about conducting a PHA (see Exhibit 30-2), and, thus, only a broad overview is provided here. One of the most comprehensive resources is the ATSDR *Public Health Assessment Guidance Manual*.[1] The ATSDR manual focuses on site-specific PHAs such as Superfund sites; nevertheless, it also can be used to assess air emissions within a limited geographical area. As described in detail in the ATSDR manual, the steps of a PHA — whether conducted by ATSDR or a state or local public health agency, and whether comprehensive or limited to a screening assessment – can be multifaceted and interactive. Exhibit 30-3 illustrates this by providing an overview of a typical PHA process. The following subsections describe this process in more detail.

30.5.1 Conduct Scoping

The first step is to establish an overall understanding of the study area and begin to identify the most pertinent issues. The objective is to quickly gain some baseline information about the study area and start developing a strategy for conducting the PHA. To help ensure a consistent approach across study areas, the following steps are followed during this initial phase:

- Initiate study area scoping by performing an initial review of permits and other sources of study area information, identifying any past health agency or partner activities, identifying and communicating with study area contacts, and determining the need for a study area visit to observe actual conditions and speak with study area representatives.

- Define roles and responsibilities of team members (internal and external).

- Establish communication mechanisms (internal and external) by developing a schedule for team meetings, thinking about how to present the findings of the assessment, and developing health communication strategies.

- Develop a study area strategy for completing the various steps in the PHA process and develop a strategy, identifying the tools and resources that might be needed to evaluate the study area, communicate the findings, and implement public health actions.

- Based on information obtained during study area scoping, develop an approach that focuses on the most pertinent public health issues.

Exhibit 30-2. Selected Public Health Assessment Resources
• Agency for Toxic Substances and Disease Registry (ATSDR; www.atsdr.cdc.gov), which publishes the *Public Health Assessment Guidance Manual* (current draft is available online; *Guidance for ATSDR Health Studies* (1996; available online), *Environmental Data Needed for Public Health Assessments* (1994, available online), and other guidance. • National Institute of Environmental Health Sciences (NIEHS; www.niehs.nih.gov), which publishes *Environmental Health Perspectives* and sponsors multidisciplinary biomedical research, prevention and intervention efforts, and communication strategies that encompass training, technology transfer, and community outreach. • American Public Health Association (APHA; www.apha.org), which publishes the *American Journal of Public Health* and provides many other resources related to environmental public health. • National Association of County and City Health Officials (NACCHO; www.naccho.org), which publishes the *Protocol for Assessing Community Excellence in Environmental Health* (2000) and *Assessment to Action: Improving the Health of Community Affected by Hazardous Waste* (2002). • National Association of Local Boards of Health (NALBOH) (www.nalboh.org), which maintains an up-to-date database of contact information for all local boards of health, provides technical assistance to existing boards of health, and will soon publish the *Environmental Health Primer*.

30.5.2 Obtain Study Area Information

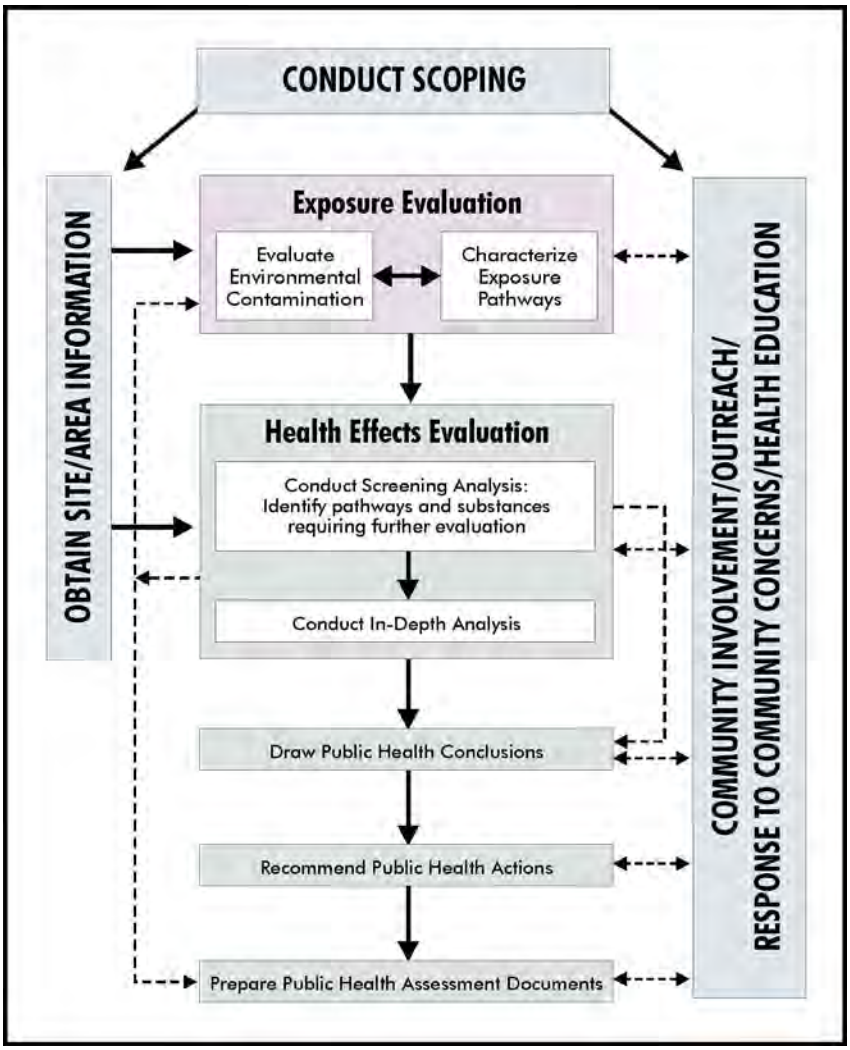
Throughout the PHA process, various team members will collect information about the study area, although the initial collection of information is typically the most intensive. Information sources typically include interviews (in-person or via telephone); study area-specific investigation reports prepared by federal, state, and local environmental and health departments; and study area visits. Gathering pertinent study area information requires a series of iterative steps, including gaining a basic understanding of the study area, identifying data needs and sources, conducting a study area visit, communicating with community members and other stakeholders, critically reviewing study area documentation, identifying data gaps, and compiling and organizing relevant data to support the assessment.

30.5.3 Community Involvement/Outreach/Response to Community Concerns

The community associated with a study area is both an important resource for and a key audience in the PHA process. Community involvement activities should be developed and implemented with the following objectives in mind:

- Earning trust and credibility through open, compassionate, and respectful communications.
- Helping community members understand what the PHA process involves and what it can and cannot do.
- Providing opportunities for communities to become involved in the PHA activities.
- Promoting collaboration between the public health agency, communities, and other agencies.
- Informing and updating communities about the health agency's work.
- Assisting communities in understanding the possible health impact of exposures to hazardous substances.

Exhibit 30-3. Overview of a Typical Public Health Assessment Process



Chapter 28 of this reference manual provides a more detailed discussion of community involvement and outreach.

30.5.4 Exposure Evaluation

For the exposure evaluation, public health assessors review environmental data to determine the sources of pollutants and exposure pathways/routes. The conceptual model described in Chapter 6 should be a reasonable starting point for the PHA exposure evaluation. Generally, the public health agency involved does not collect its own environmental sampling data, at least at first, but rather reviews information provided by federal, state, and local government agencies and/or their contractors, businesses, and the public. Assessors can indicate what further environmental sampling may be needed and may collect environmental and biologic samples when appropriate. This step involves two key substeps:

- **Evaluate Environmental Contamination Data**. This step involves determining what pollutants people may be exposed to and in what concentrations. This evaluation involves assessing the quality and representativeness of available monitoring data and measurements or modeled estimates of exposure point concentrations. This is an important way to ensure that any public health conclusions and recommendations for the study area are based on appropriate and reliable data. Both sampling data and modeling techniques described in Chapters 9, 10, 18, and 19 are sometimes used to generate data for PHAs. Evaluation of environmental contamination data typically proceeds simultaneously with the exposure pathway evaluation.

Exposure Investigations

When a PHA exposure evaluation concludes that additional exposure information is needed, an exposure investigation generally is conducted. An exposure investigation is the collection and analysis of study area-specific information to determine if human populations have been exposed to air toxics. This information may include environmental sampling, exposure-dose reconstruction, biologic or biomedical testing, and/or evaluation of medical information.

- **Characterize Exposure Pathways**. During the exposure pathway characterization, the assessor evaluates who may be or has been exposed to study area contaminants, for how long, and under what conditions. This involves identifying and studying the following five components of a "complete" exposure pathway: a source of air toxics; a mechanism for release into the air and, in some cases, transfer between media (i.e., the fate and transport of environmental contamination); an exposure point or area; an exposure route (e.g., ingestion, dermal contact, inhalation); and a potentially exposed population. The overall purpose of this evaluation is to understand how people might become exposed to study area contaminants and to identify and characterize the size and susceptibility of the potentially exposed populations. If no complete or potentially complete exposure pathways are identified, no public health hazards exist and there is no need to perform further scientific evaluation. When complete environmental or biologic data are lacking for a study area, an exposure investigation may be recommended to better assess possible impacts to public health.

30.5.5 Health Effects Evaluation

If the exposure evaluation shows that people have been or could be exposed to pollutants such as air toxics, the public health assessor will evaluate whether this contact could have resulted in harmful effects. Assessors use existing scientific information to determine the health effects that may result from exposures. Public health agencies recognize that children, because of their play activities and their growing bodies, may be particularly vulnerable to exposures to air toxics. Developing fetuses also may be more vulnerable to such exposures. Thus, the impact to children and developing fetuses is considered first when evaluating the health threat to a community. The health effects evaluation is composed of two basic substeps: a screening analysis and a more in-depth analysis.

- **Screening Analysis**. Screening is a first step in understanding whether the detected concentrations to which people may be exposed are harmful. The screening analysis is a fairly standard process developed to help health assessors sort through the large volumes of environmental data for a study area. It enables the assessor to safely rule out substances that are not at levels of health concern and to identify substances and pathways that need to be

examined more closely. For complete or potential exposure pathways identified in the exposure pathway evaluation, the screening analysis may involve comparing media concentrations at points of exposure to "screening" values (based on protective default exposure assumptions) and estimating exposure doses based on study area-specific exposure conditions. The assessor then compares estimated doses with health-based guidelines to identify substances requiring further evaluation. Exhibit 30-4 describes several of the ATSDR-derived comparison values available. See Chapter 12 for how these values are used in an air toxics risk assessment.

Exhibit 30-4. Definitions of ATSDR-Derived Comparison Values
Environmental Media Evaluation Guides (EMEGs). EMEGs are estimated contaminant concentrations that are not expected to result in adverse noncarcinogenic health effects based on ATSDR evaluation. EMEGs are based on ATSDR MRLs and conservative assumptions about exposure, such as intake rate, exposure frequency and duration, and body weight. Minimal Risk Levels (MRLs). An MRL is an estimate of daily human exposure to a substance (in mg/kg/day for oral exposures and parts per million [ppm] for inhalation exposures) that is likely to be without noncarcinogenic health effects during a specified duration of exposure based on ATSDR evaluations. Cancer Risk Evaluation Guides (CREGs). CREGs are estimated contaminant concentrations that would be expected to cause no more than one excess cancer in a million (10^{-6}) persons exposed during their lifetime (70 years). ATSDR's CREGs are calculated from EPA's cancer slope factors (CSFs) for oral exposures or unit risk values for inhalation exposures. These values are based on EPA evaluations and assumptions about hypothetical cancer risks at low levels of exposure. Reference Media Evaluation Guides (RMEGs). ATSDR derives RMEGs from EPA's oral reference doses, which are developed based on EPA evaluations. RMEGs represent the concentration in water or soil at which daily human exposure is unlikely to result in adverse noncarcinogenic effects.

- **In-depth Analysis**. For those pathways and substances that were identified in the screening analysis as requiring more careful consideration, the assessor will examine a host of factors to help determine whether study area-specific exposures are expected to result in illness. In this in-depth analysis, exposures are studied in conjunction with substance-specific toxicologic, medical, and epidemiologic data. Through this analysis, the assessor will be answering the following question: Based on available exposure, toxicologic, epidemiologic, medical, and study area-specific **health outcome data**, are adverse health effects expected in the community?

Answering this last question can be very challenging. For example, evaluating epidemiological data involves addressing a number of criteria to assist in judging the causal significance of associations revealed in studies (epidemiology is described in more detail in Exhibit 30-5). Individual criteria, if met, support a causal relationship but do not prove it. The more criteria that are met, the more likely it is that an observed health effect is causally related to the exposure under study. The criteria for evaluating causation are:

- **Time sequence**. Exposure must precede the onset of the disease. A logical sequence of events must be demonstrated.

Exhibit 30-5. What Are Epidemiologic Data and How Might They Be Used in an In-Depth Analysis?
Epidemiologic data are one of the key distinguishing features of PHAs compared to most quantitative risk assessments. Understanding the strengths and weaknesses of the various types of epidemiologic studies will help determine the suitability of a particular study in supporting and drawing study area and substance-specific public health conclusions. Because of the inherent limitations and uncertainties associated with environmental epidemiologic evaluations (generally due to the lack of adequate exposure data or sample size), however, *epidemiologic data should be used with caution*. The health assessor should call upon an epidemiologist to assist in evaluating the applicability and usability of literature-based or study area-specific epidemiologic data. The types of epidemiologic data that may be available and how they may be used are briefly summarized below, in order of greatest potential utility: • **Analytical studies**, such as case-control or cohort studies, evaluate the role of various risk factors in causing illness or disease by relying on comparisons between groups. Depending on the quality of the study, it may provide insight to the study area-specific exposure situation under evaluation. Study area-specific analytical studies that meet certain design criteria examine study area-specific exposures and health outcomes in community members. When available, these studies are the most relevant to the PHA. These data are rarely initially available, but the PHA process may lead to a recommendation to collect such data. Depending on the individual study design and health outcome studied, results may provide some insight on the presence or absence of a particular illness of concern in the community. Unfortunately, establishing a definitive link with a study area-related exposure is generally difficult if not impossible. • **Descriptive (or ecological) studies** examine differences in disease rates among populations over time or in different geographical locations and may be helpful in identifying plausible associations between a particular substance and disease. However, descriptive studies provide limited information on causal relationships (i.e., the degree of exposure or causal agent). • **Case reports** that describe an effect in an individual or small group can be considered in the in-depth analysis, but may have limited usefulness due to the generally small size of the affected population and sometimes anecdotal nature of the reports.

- **Strength of association**. The stronger the association, the more likely it is causal. The relative magnitude of the incidence of disease in those exposed compared to the incidence in those who are not exposed can be a valuable measure of the strength of the association.

- **Dose-response relationship**. The probability and/or severity of the effect should increase with increasing intensity and duration of exposure.

- **Specificity of association**. If the effect is unusual or is specific to the studied exposure, a causal relationship is more easily demonstrated.

- **Consistency**. A relationship should be reproducible (i.e., observed in other studies or analyses).

- **Biologic plausibility (or coherent explanation)**. The link between the "cause" and the effect should make sense biologically, by what is known about the disease and the exposure under study. The findings should be validated by what is known about animal models.

Similarly, biologic sampling results (biomarkers) need to be interpreted with caution. Specifically, issues to consider include: (1) as with environmental sampling data, biologic data need to be collected by trained professionals and analyzed in a standard way; (2) detected levels may not be the result of study area-related exposures (e.g., blood lead levels resulting from non-air toxics sources such as flaking paint); (3) results will likely only represent a snapshot of conditions in time; (4) the association between detected levels and clinical effects may not be understood based on scientific knowledge; (5) "normal" ranges, particularly for trace elements, may not be known; and (6) the people tested may not be fully representative of the exposed population, resulting from a small sample size and variations in exposures across the exposed population due to different activity patterns.

30.5.6 Draw Public Health Conclusions

Upon completing the exposure and health effects evaluations, the assessor will draw conclusions regarding the degree of hazard posed by a study area - that is, they will conclude either that the study area does not pose a public health hazard, that the study area does pose a public health hazard, or that insufficient data are available to determine whether any public health hazards exist. The process also involves assigning a **hazard conclusion category** for the study area or for an individual exposure pathway (Exhibit 30-6).

Exhibit 30-6. Summary of ATSDR Conclusion Categories

Category	Definition
1. Urgent Public Health Hazard	Applies to study areas that have certain physical hazards or evidence of short-term (less than 1 year), study area-related *exposure to hazardous substances that could result in adverse health effects and require quick intervention to stop people from being exposed.*
2. Public Health Hazard	Applies to study areas that have certain physical hazards or evidence of chronic, study area-related *exposure to hazardous substances that could result in adverse health effects*.
3. Indeterminate Public Health Hazard	Applies to study areas *where critical information is lacking* (missing or has not yet been gathered) to support a judgment regarding the level of public health hazard.
4. No Apparent Public Health Hazard	Applies to study areas where exposure to study area-related chemicals might have occurred in the past or is still occurring, but the *exposures are not at levels expected to cause adverse health effects*.
5. No Public Health Hazard	Applies to study areas where *no exposure* to study area-related hazardous substances exists.

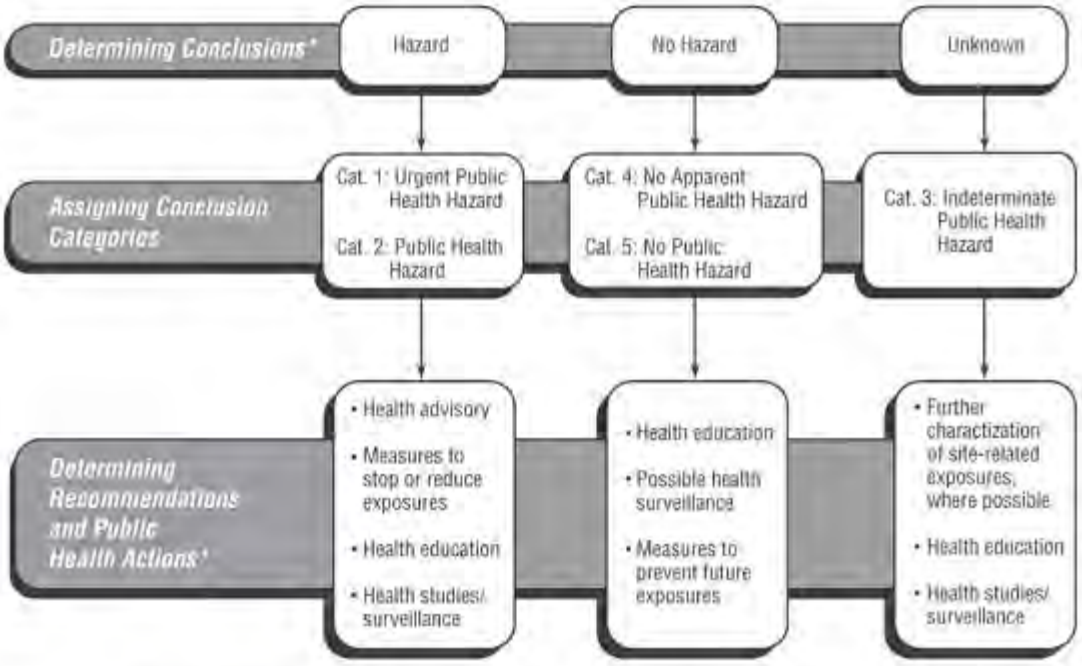
30.5.7 Recommend Public Health Actions

After drawing conclusions, the public health assessor – usually in cooperation with other team members and stakeholders – will develop recommendations for actions, if any, to prevent harmful exposures, obtain more information, or conduct other public health actions. These actions generally will be detailed in a public health action plan, which will ultimately be part of the PHA document (or possibly the public health consultation document) developed for the study area. Note that some public health actions may be recommended earlier in the process. See Exhibit 30-7 for an overview of the conclusions and recommendations process.

30.5.8 Prepare PHA Documents

The public health assessor may develop various materials during the PHA process to communicate information about the assessment, including outreach materials, health advisories that alert the public and appropriate officials to the existence of an imminent public health threat, and, at the end of the assessment process, a report that summarizes the approach, results, conclusions, and recommendations. This report generally is either a **public health assessment** (PHA) document or a **public health consultation** (PHC).

Exhibit 30-7. Overview of Typical PHA Conclusion and Recommendation Process



*Some conclusions and recommended public health actions may come early in the public health assessment process.

References

1. Agency for Toxic Substances and Disease Registry (ATSDR). 2002. Public Health Assessment Guidance Manual (Update): Draft for Public Comment.. Available at: http://www.atsdr.cdc.gov/HAC/PHAManual/cover.html.

Chapter 31 Probabilistic Risk Assessment

Table of Contents

31.1 Introduction 1

31.2 Tiered Approach for Risk Assessment 2

31.3 Methods for Probabilistic Risk Assessment 4

31.4 Presenting Results for Probabilistic Risk Assessment 6

References 10

31.1 Introduction

Probabilistic risk assessment (PRA) uses probability distributions to characterize variability or uncertainty in risk estimates. In a PRA, one or more variables in the risk equation is defined as a probability distribution rather than a single number. Similarly, the output of a PRA is a range or probability distribution of risks experienced by the receptors. Note that the ability to perform a PRA often is limited by the availability of distributional data that adequately describe one or more of the input parameters. For example, data often are insufficient to assess toxicity in a probabilistic manner (and therefore, dose-response values such as inhalation unit risks (IURs) and reference concentrations (RfCs) are included in a PRA analysis as point values). This general lack of data impacts both human health and ecological receptors.

The primary advantage of PRA is that it can provide a quantitative description of the degree of variability or uncertainty (or both) in risk estimates for both cancer and noncancer health effects and ecological hazards. The quantitative analysis of uncertainty and variability can provide a more comprehensive characterization of risk than is possible in the point estimate approach.

Another significant advantage of PRA is the additional information and potential flexibility it affords the risk manager. Risk management decisions are often based on an evaluation of high-end risk to an individual – for deterministic analyses, this is generally developed by the combination of a mix of central tendency and high-end point values for various exposure parameters (see Part II, Chapters 9 and 13). When using PRA, the risk manager can select a specific upper-bound level from the high-end range of percentiles of risk, generally between the 90th and 99.9^{th} percentiles.

PRA may not be appropriate for every analysis. The primary disadvantages of PRA are that it generally requires more time, resources, and expertise on the part of the assessor, reviewer, and risk manager than a point estimate approach. The chief obstacle to using PRA in air toxics risk assessments is usually the lack of well-documented frequency distributions for many input variables.

A detailed discussion of PRA is beyond the scope of this document. Two documents provide more detailed introductory information and guidance and should be reviewed if a PRA is contemplated:

U.S. EPA. 2001. *Risk Assessment Guidance for Superfund (RAGS), Volume III - Part A, Process for Conducting Probabilistic Risk Assessment*. Office of Solid Waste and Emergency Response. December. EPA 540-R-02-002, OSWER 9285.7-45, PB2002 963302, available at: http://www.epa.gov/superfund/programs/risk/rags3a/index.htm.

National Council on Radiation Protection and Measurements (NCRP). 1996. *A Guide for Uncertainty Analysis in Dose and Risk Assessments Related to Environmental Contamination*. NCRP Commentary No. 14, May 1996.

This chapter provides a general overview of PRA as it applies to air toxics risk assessment. It revisits the tiered approach to risk assessment, introduces calculation algorithms, and identifies advanced statistical methods currently available to support risk policy decisions.

31.2 Tiered Approach for Risk Assessment

The tiered approach is a process for a systematic, informed progression to increasingly more complex risk assessment methods including PRA. Exhibit 31-1 presents a schematic representation of the tiered approach. Higher tiers reflect increasing complexity and, in many cases, will require more time and resources. Higher tiers also reflect increasing characterization of variability and/or uncertainty in the risk estimate, which may be important for making risk management decisions. Central to the concept of a systematic, informed progression is an iterative process of evaluation, deliberation, data collection, work planning, and communication. All of these steps should focus on deciding: (1) whether or not the risk assessment, in its current state, is sufficient to support risk management decisions (a clear path to exiting the tiered process is available at each tier), and (2) if the assessment is determined to be insufficient, whether or not progression to a higher tier of complexity (or refinement of the current tier) would provide a sufficient benefit to warrant the additional effort.

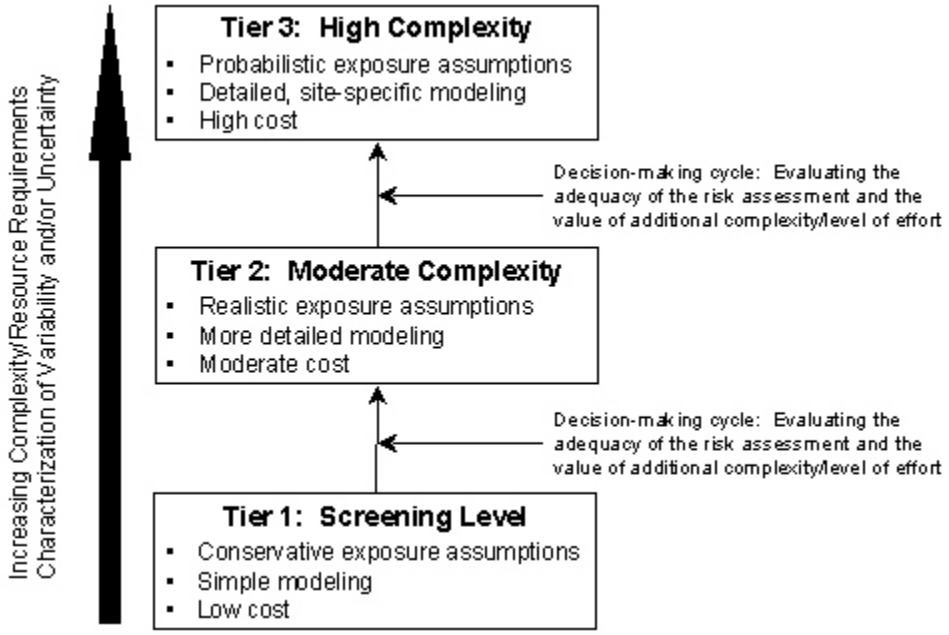
- The problem formulation step precedes Tier 1 and includes scoping and refinement of the conceptual site model, including exposure pathways/routes, and identifying chemicals of potential concern (COPCs).

- In Tier 1, deterministic (point estimate) risk assessment is then performed using the basic methodology described in Part II (inhalation) and/or Part III (multipathway) of this Reference Manual. In deciding whether the results of a deterministic risk assessment are sufficient for decision-making or whether more refined analyses should be implemented, two factors generally are considered: (1) the magnitude of the estimates of risk (i.e., the value of hazard indices [HIs] or cancer risks for COPCs), and (2) the level of confidence in these estimates. In a Tier I deterministic risk assessment, quantitative risk estimates can be easily calculated, but the level of confidence associated with these calculations can be difficult to assess. For example, variability in exposure levels among individual members of the population can generally only be assessed semi-quantitatively by considering central tendency and high-end exposure estimates. Uncertainty can often be evaluated only as confidence limits on certain point estimates (e.g., the concentration term).

  In some cases, the results of a Tier 1 risk analysis may be sufficient for decision-making. For example, a deterministic analysis may indicate very low levels of risk for some air toxics. If the assessment is considered to be overly conservative (even in light of uncertainties), this may be sufficient for a “no action” decision for those chemicals. The same analysis may indicate a very high potential for risk for other air toxics. EPA generally recommends that the risk manager proceed to higher tiers only when site decision-making would benefit from additional analysis beyond the point-estimate risk assessment (i.e., when the risk manager needs more complete or certain information to complete the risk management process).

Thus, only the combinations of COPC-exposure pathway-receptors of highest potential concern are generally analyzed using higher level techniques such as PRA.

Exhibit 31-1. Example of a Tiered Approach for Risk Assessment



Adapted from Volume III of EPA's Risk Assessment Guidance for Superfund[1]

- Tier 2 is represented as an intermediate-level analysis using more realistic exposure assumptions (e.g., use of actual receptor locations) and more detailed modeling (e.g., a model that requires additional site-specific inputs). Although not depicted, Tier 2 could incorporate a sensitivity analysis to identify the most important parameters that are driving the risk estimate for specific receptors or population groups. Tier 2 also could incorporate limited (one-dimensional) Monte Carlo techniques.

- Tier 3 is represented as an advanced analysis using probabilistic techniques such as two-dimensional Monte Carlo analysis. Results of sensitivity analyses (Tier 2 or Tier 3) could be used to assess risk distributions for the high-end individuals within the population. The one-dimensional Monte-Carlo simulation does not separate variability and uncertainty associated with the risk estimates. If necessary, separate analyses of uncertainty and variability can be performed in Tier 3. Techniques such as two-dimensional Monte Carlo simulation can be used to estimate the relative impact of natural variability and lack of data on the overall uncertainty in the risk estimate, and can be used to direct additional data gathering or to support mitigation decisions.

The deliberation cycle provides an opportunity to evaluate the direction and goals of the assessment as new information becomes available. It may include evaluations of both scientific and policy information. (Also note that, while a three-tiered approach was provided in Exhibit

31-1, the tiered approach is really more of a continuum from a point where the analysis is done with little data and conservative assumptions to a point where there is an extensive data set and fewer assumptions. In between, there can be a wide variety of tiers of increasing complexity, or, as discussed in Chapter 3, there may only be a few reasonable choices between screening methods and highly refined analyses. The three tiered approach is only provided here as an illustration of the concept, not a prescriptive, fixed methodology.)

31.3 Methods for Probabilistic Risk Assessment

As discussed in previous chapters, there are a number of approaches available for analyzing uncertainty in risk assessments. For simple screening level analyses, or analyses where there are only a few major sources of uncertainty, sensitivity analyses may be used to estimate the impacts of likely variations in the key parameter values. Where scenario uncertainty is important (that is, there are multiple sequences of events that could contribute to risk), decision tree or Bayesian statistical analysis are commonly used. The most common numerical technique for PRA (analyses in which a large number of variables need to be evaluated simultaneously) in large-scale air risk assessments is Monte Carlo simulation. Monte Carlo simulation integrates varying assumptions, usually about exposure, to come up with possible distributions (or ranges) of risk instead of point estimates. A continuous probability distribution can be displayed in a graph in the form of either **probability density functions** (PDFs) or corresponding **cumulative distribution functions** (CDFs); however, for clarity, it is recommended that both representations be presented in adjacent (rather than overlaid) plots.

Exhibit 31-2 illustrates a PDF and CDF for a normal probability distribution for adult body weight. Both displays represent the same distribution, but are useful for conveying different information. PDFs are most useful for displaying (1) the relative probability of values; (2) the most likely values (e.g., modes); and (3) the shape of the distribution (e.g., skewness, kurtosis, multimodality). CDFs can be used to display (1) percentiles, including the median; (2) high-end risk range (e.g., 90th to 99th percentiles); (3) confidence intervals for selected percentiles; and (4) stochastic dominance (i.e., for any percentile, the value for one variable exceeds that of any other variable). Note that it is helpful to include a text box with summary statistics relevant to the distribution (e.g., mean, standard deviation).

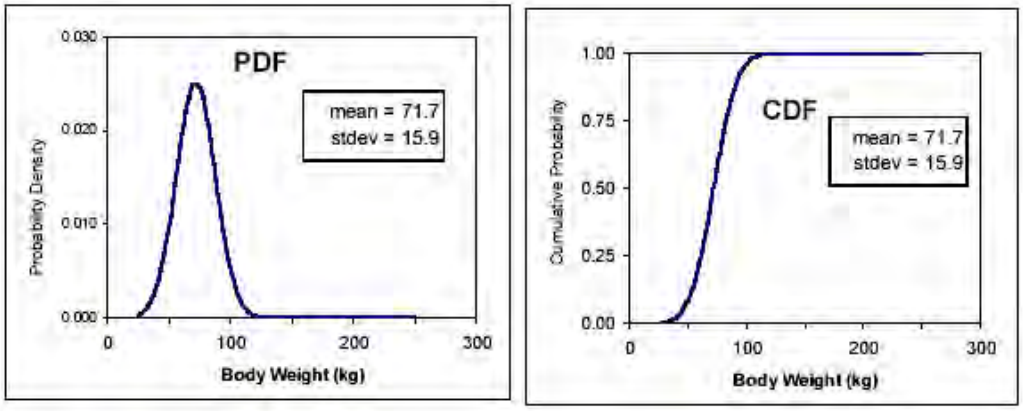
These results expressed as probability distributions help risk managers decide whether and what actions are necessary to reduce risk. Monte Carlo simulation has been widely used to explore problems in many disciplines of science as well as engineering, finance, and insurance.[1] The process for a Monte Carlo simulation is illustrated in Exhibit 31-3. In its general form, the risk equation can be expressed as a function of a toxicity term (as a point value) and multiple exposure variables (V_n) represented as distributions (not point values):

$$\text{Risk} = f(V_1, V_2, V_3, \ldots V_n) \times \text{Toxicity} \qquad \text{Equation 31-4}$$

The first decision(s) the risk assessor has to make is which of the “Vs” are going to be evaluated probabilistically. Ideally, every model input that is variable or uncertain should be evaluated to provide a comprehensive characterization of uncertainty in exposure estimates. In practice, the

number of variables that can be addressed systematically is severely limited by lack of data related to variability, uncertainty, or both. Sensitivity analyses can often be used to focus the analysis on the variables that contribute most to the overall uncertainty in risks.

Exhibit 31-2. Examples of Probability Density and Cumulative Distribution Functions



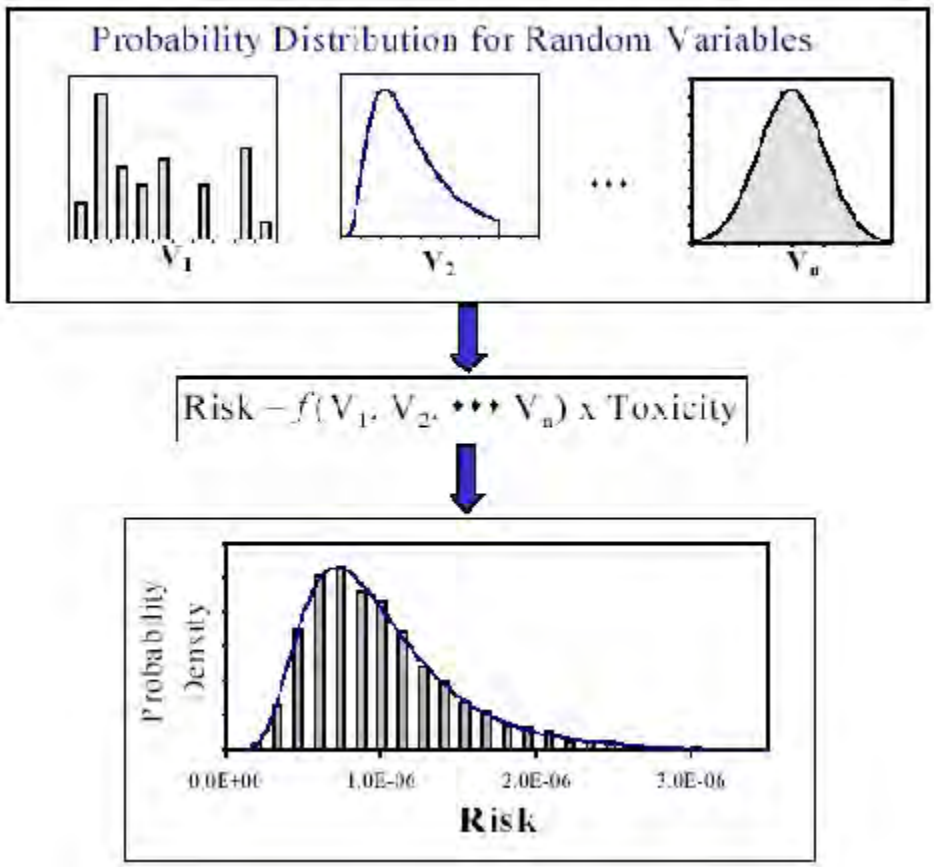
Example of a normal distribution that characterizes variability in adult body weight (males and females combined). The arithmetic mean = 71.7 kg, and standard deviation = 15.9 kg. Body weight may be considered a continuous random variable. The left panel shows a bell-shaped curve and represents the PDF, while the right panel shows an S-shaped curve and represents the CDF. Both displays represent the same distribution (including summary statistics), but are useful for conveying different information.
Source: Finley and Paustenbach[2]

Solutions for equations with PDFs are typically too complex for even an expert mathematician to calculate the risk distribution analytically. However, numerical techniques applied with the aid of computers can provide very close approximations of the solution. This is illustrated here for the simplified case in which the assessment variables are statistically independent, that is, the value of one variable has no relationship to the value of any other variable. In this case, the computer selects a value for each variable (V_n) at random from a specified PDF and calculates the corresponding risk. This process is repeated many times (e.g., 10,000), each time saving the set of input values and corresponding estimate of risk. For example, the first risk estimate might represent a hypothetical individual who drinks 2 L/day of water and weighs 65 kg, the second estimate might represent someone who drinks 1 L/day and weighs 72 kg, and so forth. Each calculation is referred to as an **iteration**, and a set of iterations is called a **simulation**.

Each iteration of a Monte Carlo simulation should represent a plausible combination of input values (i.e., exposure or ecotoxicity variables), which may require using bounded or truncated probability distributions. However, risk estimates are not intended to correspond to any one person. The "individuals" represented by Monte Carlo iterations are "virtual," and the risk distributions derived from a PRA allow for inferences to be made about the likelihood or probability of risks occurring within a specified range for an exposed human or ecological

population. A simulation yields a set of risk estimates that can be summarized with selected statistics (e.g., arithmetic mean, percentiles) and displayed graphically using PDF and CDF for the estimated risk distribution.

Exhibit 31-3. Conceptual Model of Monte Carlo Analysis



Random variables (V_1, V_2, ...V_n) refer to exposure variables (e.g., body weight, exposure frequency, ingestion rate) that are characterized by probability distributions. A unique risk estimate is calculated by sampling each set of the random values and calculating a result. Repeated sampling results in a frequency distribution of risk can be described by a probability density function. In human health risk assessments, the toxicity term is usually expressed as a point estimate. In ecological risk assessments, the toxicity term may be expressed as a point estimate or as a probability distribution.

31.4 Presenting Results for Probabilistic Risk Assessment

The complexity of risk evaluation, and particularly of probabilistic methods, may pose a significant barrier to understanding among the affected and interested parties (and thus to the utility of the analysis). In the past, regulatory decisions have been evaluated primarily in terms of point estimates of risk and simple dichotomous decision rules (e.g., "If the point estimate of risk

is above a certain level, take a certain action. If not, take another action.”). In contrast, it may not be intuitively obvious, even to relatively sophisticated audiences, how to relate the outputs of quantitative uncertainty evaluation to a particular decision. For example, important aspects of a regulatory decision may rest on relatively subtle statistical distinctions (e.g., the difference between a 95th percentile risk estimate and a 95th percent upper confidence limit on a risk estimate), and the challenges in presenting such information can be formidable. In its recent guidance, EPA has begun to define concrete approaches to presenting risks and uncertainty information to decision-makers and stakeholders.[5]

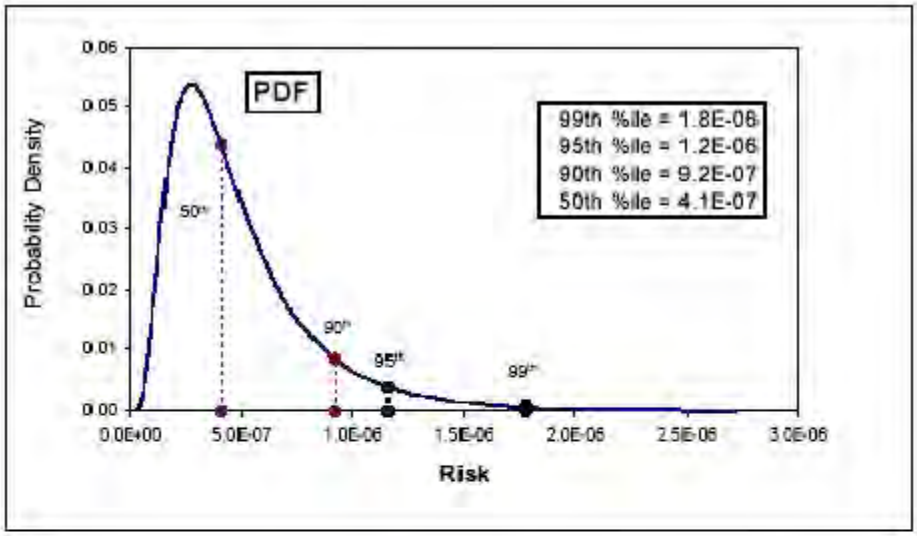
The key factors for successful communication of PRA include early and continuous involvement of affected and interested parties, a well-developed communication plan, good graphics, a working knowledge of the factors that may influence perceptions of risk and uncertainty, and a foundation of trust and credibility. A certain amount of training for interested stakeholders will likely be necessary to help them understand the complexities of not only risk assessment in general, but the intricacies of higher levels of analysis. Part III of this Reference Manual provides guidance on community involvement and risk communication.

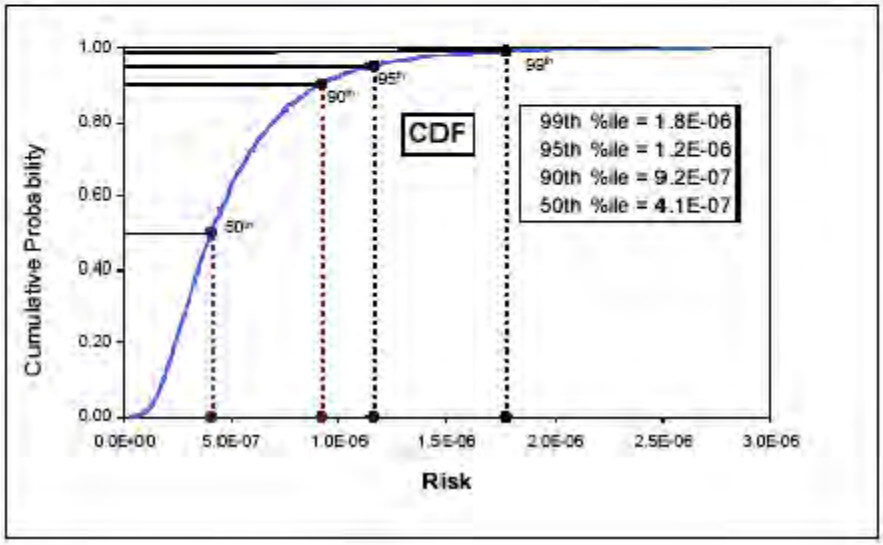
When summarizing results of PRA, graphs and tables should generally also include the results of the point estimates of risk (e.g., central tendency and high-end).

Consistent with EPA’s guidance on risk characterization,[3] the central tendency and high-end cancer risks and noncancer hazards, along with decision points, should be highlighted on graphics. The discussions accompanying the graph should emphasize that these values represent risks to the average and high-end individuals, respectively, and serve as a point of reference to EPA’s decision point. The distribution of risks should be characterized as representing variability among the population based on differences in exposure. Similarly, graphics that show uncertainty in risk estimates can be described using terms such as “confidence interval,” “credible interval,” or “plausible range,” as appropriate. The graphics need not highlight all percentiles. Instead, selected percentiles that may inform risk management decisions (such as the 5th, 50th, 90th, 95th, and 99th percentiles) should be the focus. Exhibit 31-4 presents an example of a PDF for variability in risk with an associated text box for identifying key risk descriptors.

By understanding the assumptions regarding the inputs and modeling approaches used to derive point estimates and probabilistic estimates of risk, a risk communicator will be better prepared to explain the significant differences in risk estimates that have been developed. Special emphasis should be given to the model and parameter assumptions that have the most influence on the risk estimates, as determined from the sensitivity analysis.

Exhibit 31-4. Example of Presenting the Results of a Probabilistic Risk Assessment





Hypothetical PRA results showing a PDF (top panel) for cancer risk with selected summary statistics for central tendency and high-end percentiles. This view of a distribution is useful for illustrating the shape of the distribution (e.g., slightly right-skewed) and explaining the concept of probability as the area under a curve (e.g., most of the area is below $1x10^{-6}$, but there is a small chance of $2x10^{-6}$). Although percentiles can also be overlayed on this graphic, a CDF (bottom panel) may be preferable for explaining the concept of a percentile.

Additional References on Uncertainty Analysis

Burmaster, D.E. and Anderson, P.D. 1994. Principles of good practice for the use of Monte Carlo techniques in human health and ecological risk assessments. *Risk Analysis* 14: 477-481.

Cullen, A.C. and Frey, H.C. 1999. *Probabilistic Techniques in Exposure Assessment.* New York: Plenum Press.

Fayerweather, W.E., Collins, J.J., Schnatter, A.R., Hearne, F.T., Menning, R.A., and Reyner, D.P. 1999. Quantifying uncertainty in a risk assessment using human data. *Risk Analysis* 19: 1077-1090

Finkel, A.M. and Evans, J.S. 1987. Evaluating the benefits of uncertainty reduction in environmental health risk management. *Journal of the Air Pollution Control Association*. 37: 1164-1171.

Frey, H.C. 1992. Quantitative analysis of uncertainty and variability in environmental policy making. Pittsburgh: Carnegie Mellon University.

Hattis, D. and Burmaster, D.E. 1994. Assessment of variability and uncertainty distributions for practical risk assessments. *Risk Analysis* 14: 713-730.

Hope, B. K. 1999. Assessment of risk to terrestrial receptors using uncertainty analysis - A case study. *Human and Ecological Risk Assessment* 5(1): 145-170.

Moore, D.R.J., Sample, B.E., Suter, G.W., Parkhurst, B.R., and Teed, R.S. 1999. A probabilistic risk assessment of the effects of methylmercury and PCBs on mink and kingfishers along East Fork Poplar Creek, Oak Ridge, Tennessee, USA. *Environmental Toxicology and Chemistry* 18: 2941-2953.

National Research Council (NRC). 1991. *Human Exposure Assessment for Airborne Pollutants.* Washington DC: National Academy Press.

Roberts, S.M. 1999. Practical issues in the use of probabilistic risk assessment and its applications to hazardous waste sites. *Human and Ecological Risk Assessment.* 5(4): 729-868. Special Issue.

Smith, R.L.. 1994. Use of Monte Carlo simulation for human exposure assessment at a Superfund site. *Risk Analysis* 14(4): 433-439.

U.S. Environmental Protection Agency. 1985. *Methodology for Characterization of Uncertainty in Exposure Assessments.* Washington DC, EPA-600/8-85-009).

References

1. Rugen, P. and B. Callahan. 1996. An overview of Monte Carlo: A fifty year perspective. *Human Health and Ecological Risk Assessment* 2(4): 671-680.

2. Finley, B. and D. Paustenbach. 1994. The benefits of probabilistic exposure assessment: Three case studies involving contaminated air, water, and soil. *Risk Analysis* 14(1): 53-73.

3. U.S. Environmental Protection Agency. 1992. *Guidance on Risk Characterization for Risk Managers and Risk Assessors.* Risk Assessment Council, Washington, DC, February 26, 1992.

Chapter 32 Use of Geographic Information Systems (GIS) in Risk Assessment

Table of Contents

32.1 Introduction 1

32.2 Selecting a GIS 2

32.3 Acquiring and Using Demographic Data 4
- 32.3.1 U.S. Census Data 5
- 32.3.2 Current and Small-Area Demographic Estimates 5
- 32.3.3 Public Health Applications 7
- 32.3.4 Data Access and Distribution 7

32.4 Cartographic Concepts 7
- 32.4.1 Generalization, Simplification, and Abstraction 10
- 32.4.2 Map Projections 10

32.5 Using the Internet as a GIS Tool 10

32.6 Current GIS Applications at EPA 11
- 32.6.1 ORD/ESD 12
- 32.6.2 ATtILA 12
- 32.6.3 ReVA 12

32.7 GPS Technology 13

References 15

32.1 Introduction

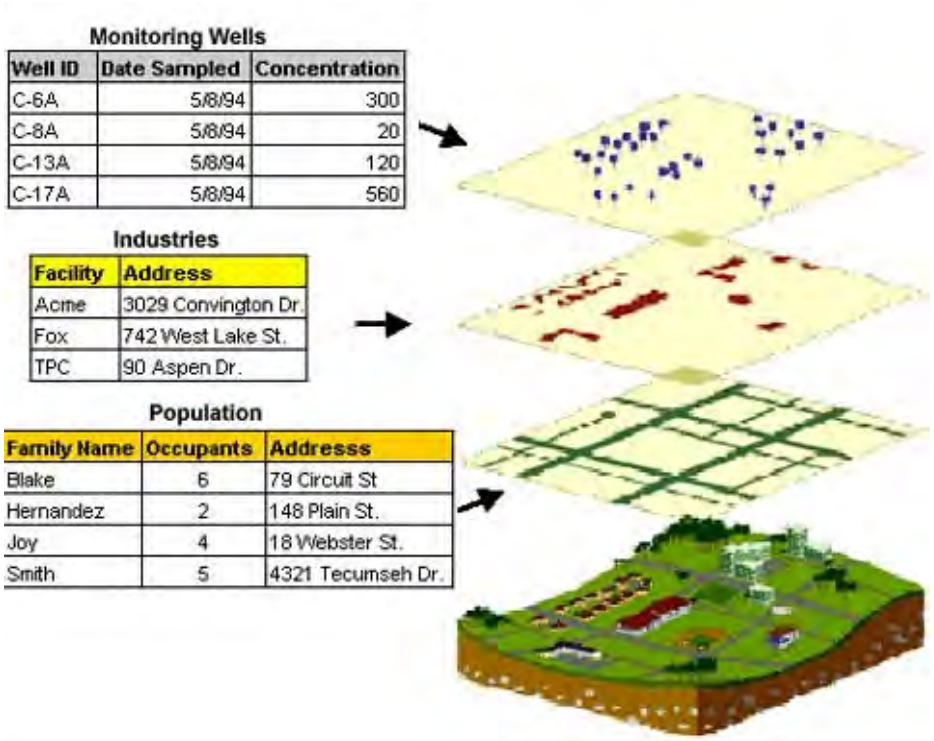
A **geographic information system (GIS)** can be defined as an organized collection of software and geographic data that allow efficient storage, analysis, and presentation of spatially explicit and geographically referenced information. Traditional methods of processing such data have been extremely labor intensive, such as manually digitizing a map from an aerial photograph and then adding information about chemical contaminants. A GIS provides a powerful analytical tool that can be used to create and link spatial and descriptive data for problem solving, spatial modeling and presentation of results in tables or maps. For air toxics risk assessment, GIS can be a powerful tool for displaying and analyzing data during the planning, scoping, and problem formulation phases, during the exposure assessment, and displaying and evaluating the results of the risk characterization. It is also a very helpful means for communicating information to risk managers and other stakeholders.

GIS data generally consist of two components: (1) graphical data about geographic features (e.g., rivers, land use, political boundaries), and (2) tabular data about features in the geography (e.g., population, elevation, modeled ambient concentrations of air toxics). GIS combines these different types of data using a "layering" technique that references each type of data to a uniform geographic coordinate system (usually a grid such as latitude and longitude coordinates). Layered data can then be analyzed using special software to create new layers of data (see Exhibit 32-1).

Over the last several years, GIS applications have evolved from very specialized and expensive analyses that required specialized computers (e.g., supercomputers and workstations) to user-friendly desktop applications utilized by everyday users to do such mundane tasks as print maps or driving directions. Libraries of geographical information developed for general use (e.g., topographical maps, infrastructures, natural resources), and for use by EPA and other regulatory agencies, can be easily downloaded from different servers and used in air toxics risk assessments. One example of a GIS Web-based application is EPA's Envirofacts system[(1)] which provides website access to several EPA databases that provide information about environmental activities that may affect air, water, and land anywhere in the United States (with much of the data available in GIS format).

This chapter provides an overview of GIS and its application to air toxics risk assessment. More detailed information is provided in the Agency for Toxic Substances and Disease Registry (ATSDR)/Southern Appalachian Assessment GIS (SAAGIS) publication *Introduction to ArcView and Spatial Analysis Techniques for Public Health Professionals*.[(2)]

Exhibit 32-1. Example Conceptual Model Using GIS



Example of layering within a GIS. The location of monitoring wells, industries, and potential receptors (homes) are all referenced to the same geographic coordinates. This allows spatial analysis of the overlap of sources, contaminant plumes, and receptors, as well as a visual means to communicate complex data sets.

32.2 Selecting a GIS

After risk assessors decide to use a GIS, they must choose a software system. A variety of GIS software is available from commercial vendors. A key feature in selecting a GIS is identifying a minimal set of capabilities needed. Important functional capabilities to consider include: data capture, data storage, data management, data retrieval, data analysis, and data display.[3]

- **Data Capture**. All data used in a GIS must have a spatial component. This means that all information brought into the system must be geo-referenced (i.e., correspond to some physical location). Data capture is the process of incorporating map and attribute data into the GIS. **Geocoding**, which is the conversion of analog data to geo-referenced digital format, is a common way for GIS users to bring map and attribute data into their GIS analyses. Two common methods of geocoding are scanning and digitizing. Both involve taking non-digital information (e.g., a hard-copy map), and converting it into a digital format. In addition to paper files, GIS users often import files from common formats such as AutoCAD DXF. The

newly imported digital information (e.g., the boundary of a state), is geo-referenced by coordinates so that it corresponds to a physical location.

In addition to graphical data, GIS incorporates tabular data for objects included in a data layer. For example, the graphical data associated with a home could consist of its size and location. The tabular data associated with that home consists of attributes such as who lives there, when it was built, where its water supply comes from, and what type of heating system it uses. These attributes would be listed in a table that is linked to the physical location of the house by the GIS. While obtaining geographical base layers that show boundaries is essential, data capture also involves attribute data, which necessitates that the GIS software package have some level of database manager associated with the program. A useful program will generally have features that allow it to import common database files such as those from dBASE®, Access®, Excel®, and Paradox®. The different software packages will vary in their ability to check the characteristics of the databases.

- **Data Storage**. A GIS can incorporate a tremendous amount of data into a map. Space is a key issue related to data storage in a GIS. With the decrease in cost of disk storage, the development of high-density storage media (e.g., CD-ROM), and the incorporation of compression methods, space is not as critical an issue as it has been in the past. However, GIS is still relatively memory-intensive. GIS microcomputer software can take up tens of megabytes of space without data, and a more complete workstation version may use hundreds of megabytes of space. Add to this the datasets with very high resolution (that can move into the gigabyte range in size), and there is a the potential for a significant storage problem. Some storage problems can be resolved by establishing data sets on a common server, accessible to multiple users.

- **Data Management**. A powerful GIS is one which has the ability to manage both map and attribute data. Every GIS is built around the software capabilities of a database management system (DBMS). A DBMS is software that is capable of storing, selecting, retrieving, and reorganizing attribute information. It allows data entry, data editing, and supports several different types of output. Functions include the ability to select records based on their value. Several database functions can work independently of the GIS functions.

- **Data Retrieval**. A GIS will support the retrieval of features by their attributes or by their spatial characteristics. A basic retrieval based on spatial characteristics is used to show the position of a single feature. In addition, a GIS is capable of allowing the operator to use the map as a query vehicle. A simple way of doing this is to point to a feature and retrieve the list of attributes for that feature. The database management function also is important for the data retrieval capacity because it allows for the selection and retrieval based on an attribute. **Buffering** is one retrieval operation that defines a GIS. Buffering allows the user to retrieve features within a specified distance of a point, line, or area. **Overlay** is another spatial retrieval operation in which non-overlapping regions are joined to create a new area. More sophisticated retrieval operations also are available.[(2)]

- **Data Analysis**. GIS systems vary a great deal in their data analysis capabilities. Basic tasks that should be included in a GIS are: spreadsheet and database analysis, computing new attributes, generating summary statistics, creating reports, statistics such as mean and variance, significance testing, and plotting residuals. In addition, selected geometric tests should be included (e.g., point-in-polygon analysis, surface partitioning).

- **Data Display**. GIS software displays information visually as data layers of a map. GIS users must select the correct map projection to make sure that their maps are not distorted. For example, large areas, such as continents, must be projected with the earth's curvature taken into consideration. Small areas can be projected essentially as flat. GIS software gives users as wide variety of map projection options to ensure that maps are as accurate as possible. Section 32.4.2 discusses map projections in further detail.

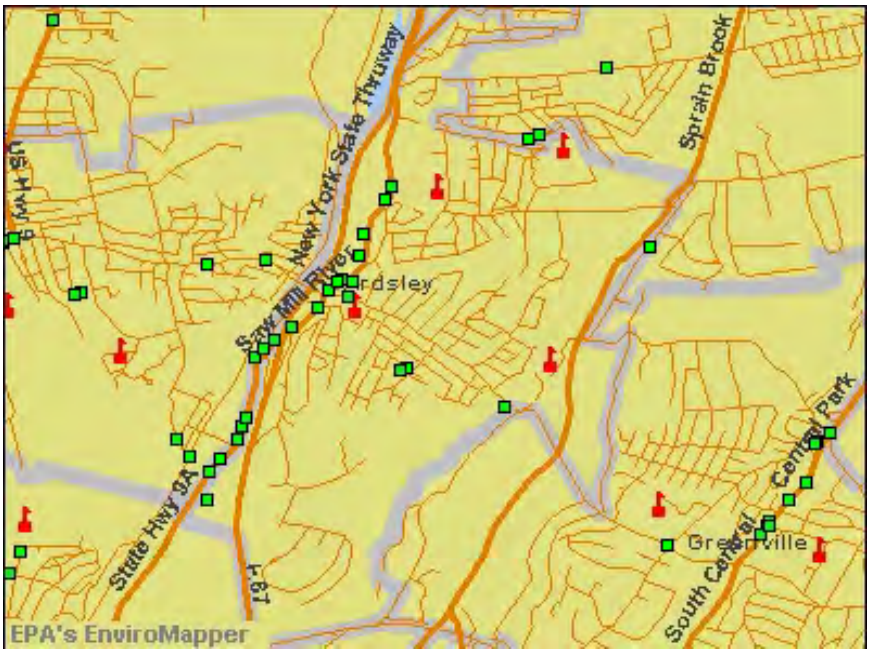
  Different data sources and agencies provide digital data that has been processed using different coordinate systems and map projections. Risk assessors may want to use data layers from many different sources to create a single map. For example, a topography layer from the U.S. Geologic Survey might be combined with a layer showing census blocks from the U.S. Census and a layer showing lead smelters from EPA. Software that can handle a variety of coordinate systems and map projections is essential to GIS capability to overlay layers created from many different sources.

32.3 Acquiring and Using Demographic Data

Demography is the study of the size, composition, distribution, and change in population. Geographers focused on population studies are also interested in the spatial distribution of demographic characteristics.[4] Data from the U.S. Bureau of the Census decennial census is the most common source of residential population information for states, the District of Columbia, and many U.S. territories (e.g., Puerto Rico, U.S. Virgin Islands, American Samoa, and Guam). These data also provide the base for current year population estimates and projections. Risk assessors are often interested in using demographic data because it allows them to identify sensitive sub-populations, such as children or the elderly. A GIS lets risk assessors combine demographic data with data on the location of sources (or estimated ambient air concentrations) to visualize where human health is potentially at risk (see Exhibit 32-2).

Within a GIS, political and statistical geographic area boundary files are linked to the attribute data (e.g., age, race, housing value) describing residents and housing units in that area using Federal Information Processing Standard (FIPS) codes. These codes provide unique identifiers for various geographic areas. When analyzing census data that is nested within the data hierarchy (e.g., census blocks within census tracts), it is best to include the FIPS codes for the larger geographic areas in that hierarchy to ensure that you are using a unique identifier. For example, connecting the FIPS codes for block 201, census tract 12, Fulton county, state of Georgia, results in the unique identifier "13089001200201" for that block. Because the codes are nominal numerals, it is best to treat them as character data (or strings) rather than numbers in the GIS database (although this may not be consistent across data sources).

Exhibit 32-2. Illustration of the Use of GIS to Identify Sensitive Receptors Close to Emissions Sources



In this map, the squares represent hazardous waste sites, and the flagged symbols represent schools. Schools and other locations where sensitive subpopulations may occur that are close to air toxics emissions sources may be of particular interest in a risk assessment.

32.3.1 U.S. Census Data

U.S. census data describing the residential population and housing in the U.S. provide the most complete picture of our nation and its subareas, which makes them very valuable demographic data. Exhibit 32-3 shows the type of information collected in the 2000 census. Many of the Census 2000 data files are available for use in GIS.

32.3.2 Current and Small-Area Demographic Estimates

An issue with census data is that the information represents a "snapshot" in time (generally based on April 1 of the census year). As one moves forward in time, such data may be less reflective of the actual demographic conditions in the study area. This problem is more pronounced for small-area data (e.g., census tracts and block groups). While the census data typically are appropriate for screening-level assessments (e.g., some air quality models include the 2000 census data), more refined assessments may require more current information, which is available from several commercial sources.

Exhibit 32-3. Information Collected in the 2000 Census	
During census years, households received and were asked to respond to one of two census forms – the "short form," which gives the "100-percent component," or the "long form," which gives the "sample component." Questions on the short form were also found on the long form and thus, were (theoretically) asked of every household in the nation. Basic population and housing data were gathered in this way. More detailed population information was obtained from the long form sent to a sample of households. On average, approximately one in six households received the long form. The rate varied from one in two households in some smaller areas, to one in eight households for more densely populated areas.	
100 Percent Component from the Short Form	
Population • Name • Household relationship • Sex • Age • Hispanic or Latino origin • Race	**Housing** • Tenure – owned or rented
Sample Component from the Long Form	
Population *Social characteristics* • Marital status • Place of birth, citizenship, year of entry to the U.S. • School enrollment and attainment • Ancestry • Residency five years ago (migration) • Language spoken at home and ability to speak English • Veteran status • Disability • Grandparents as care givers *Economic characteristics* • Labor force status • Place of work and journey to work • Occupation, industry, and class of worker • Work status in 1999 • Income in 1999	**Housing** • Units in structure • Year structure built • Number of rooms and number of bedrooms • Year moved into residence • Plumbing and kitchen facilities • Telephone service • Vehicles available • Heating fuel • Farm residence *Financial Characteristics* • Value of home or monthly rent paid • Utilities, mortgage, taxes, insurance, and fuel costs
Source: U.S. Census. *Census 2000 Basics.* Available at: http://www.census.gov/mso/www/c2000basics/00Basics.pdf	

A number of commercial entities provide annual small-area population and housing estimates and projections. Estimates are calculated using the most recent decennial census as the population base and incorporating other, often proprietary, data sources to refine the estimates. In addition to providing updated demographics, some vendors have developed segmentation systems that classify the U.S. population into distinct lifestyle segments or clusters depending on residential location ("geodemographics"). The idea of clustering is based on the notion that, more often than not, people will choose to live near others like themselves. This is important to public health because assessors can be more efficient in identifying and understanding where potential hazards are concentrated, as well as developing messages that reach people living in those areas.

32.3.3 Public Health Applications

The use of census data is central for public health communication planning, program planning, implementation and information dissemination. For example, the Georgia Division of Public Health used demographic information to target mammography programs in factory towns classified as "Mines & Mills" because women in those communities were found to have higher rates of breast cancer.[5] As another example, the Centers for Disease Control (CDC) Office of Communication has collaborated with a number of centers on projects that integrate epidemiological and other data for communication planning including HIV status awareness and hantavirus prevention.[6] Because exposure to air toxics is often influenced significantly by proximity to sources, spatial information is essential to identifying areas where human health might be adversely impacted.

32.3.4 Data Access and Distribution

There are numerous sources for acquiring U.S. census data. In addition to the Census Bureau's data access tools, including Factfinder, its Web-based data dissemination system, many public and private organizations are including census data with GIS or mapping software (e.g., ESRI, EPA LandView, HUD Community 2020, Geolytics, Claritas, CACI). State governments, universities, and non-governmental organizations (e.g., CIESIN) are also sources for data. Costs associated with obtaining the data vary.

32.4 Cartographic Concepts

While spatial information and GIS can be extremely useful, people must have assistance in observing and studying the great amount and variety of information that is represented on maps. Geographic data are extensive and voluminous, so cartography, a technique that is fundamentally concerned with reducing the spatial characteristics of a large area, makes maps readable and meaningful. A map is more than a reduction of information to an understandable level. If it is well made, it is a carefully designed instrument for recording, calculating, analyzing, and in general, understanding the interrelation of things in their spatial relationship. This section provides an overview of cartography. A more complete discussion can be found in *The Geographer's Craft Project*.[7]

One of the most useful approaches to the study of cartography is to view maps as a form of visual communication – a special purpose language for describing spatial relationships. Cartography is related to, but different from other forms of visual communication. Cartographers must pay special attention to coordinate systems, map projections, and issues of scale and direction that are in most cases of relatively little concern to other graphic designers or artists. But, because cartography is a type of graphical communication, some insights to the demands of cartography can be gleaned from the literature of graphical communication and statistical graphics. By stressing cartography as a form of communication, it is easier to make the point that maps are really symbolic abstractions – or representations – of real world phenomena. In most cases, this means that the world represented on a map has been greatly simplified, or generalized, with symbols being used like words to stand for real things. Some of the most important decisions cartographers make in the process of cartographic design revolve around: (1) how much to simplify the situation being depicted; and (2) how to symbolize the relationships being represented. In order to make good choices, cartographers often ask themselves the following questions:

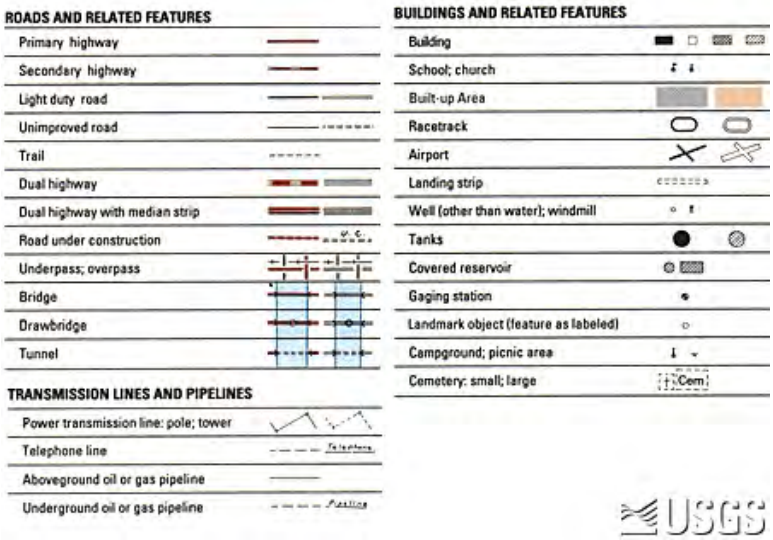
- What is the motive, intent, or goal of the map?
- Who will read the map?
- Where will the map be used?
- What data is available for the composition of the map?
- What resources are available in terms of both time and equipment?

By identifying the most important points to be conveyed by the map along with the map's main audience, cartographers can prioritize where to direct the audience's attention with larger symbols or brighter colors.

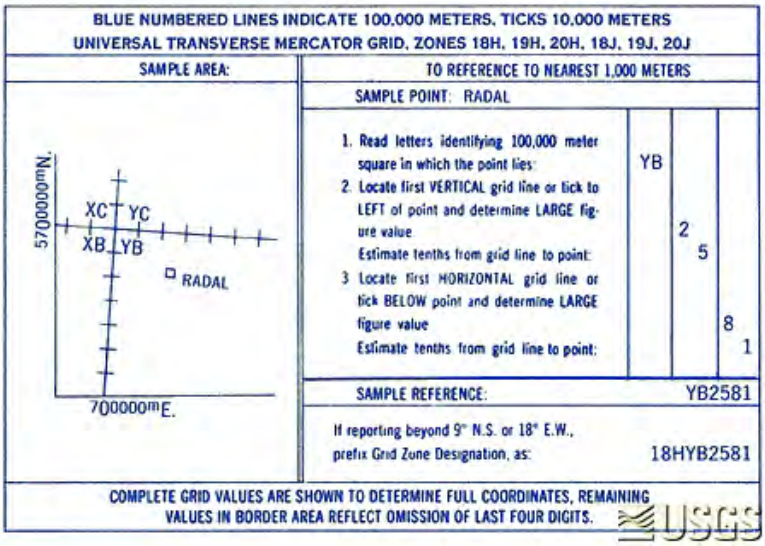
Basic Map Elements

A legend and symbols that inform the viewer of distance, scale, and direction, are basic elements to any map. The USGS (http://edc.usgs.gov/earthshots/slow/Help-GardenCity/legendstext) provides examples of common map legends.

Example U.S. Geological Survey (USGS) Topographic Map Legend



Example Legend for Universal Transverse Mercator (UTM) Projection Zones



32.4.1 Generalization, Simplification, and Abstraction

As noted above, cartography is a process of abstraction in which features of the real world are generalized or simplified to meet the demands of the theme and audience. Not all elements or details have a bearing on the pattern or process being studied and so some are eliminated to draw the reader's attention to those facts that are relevant. Too much detail can even hide or disguise the message of a map. The amount of detail that can be included is very much dependent on the scale at which the map will be produced (see Exhibit 32-4).

Map Making Tips

- Experiment with different layouts
- Think carefully about every element on your map and whether it has an essential function
- Less is more

32.4.2 Map Projections

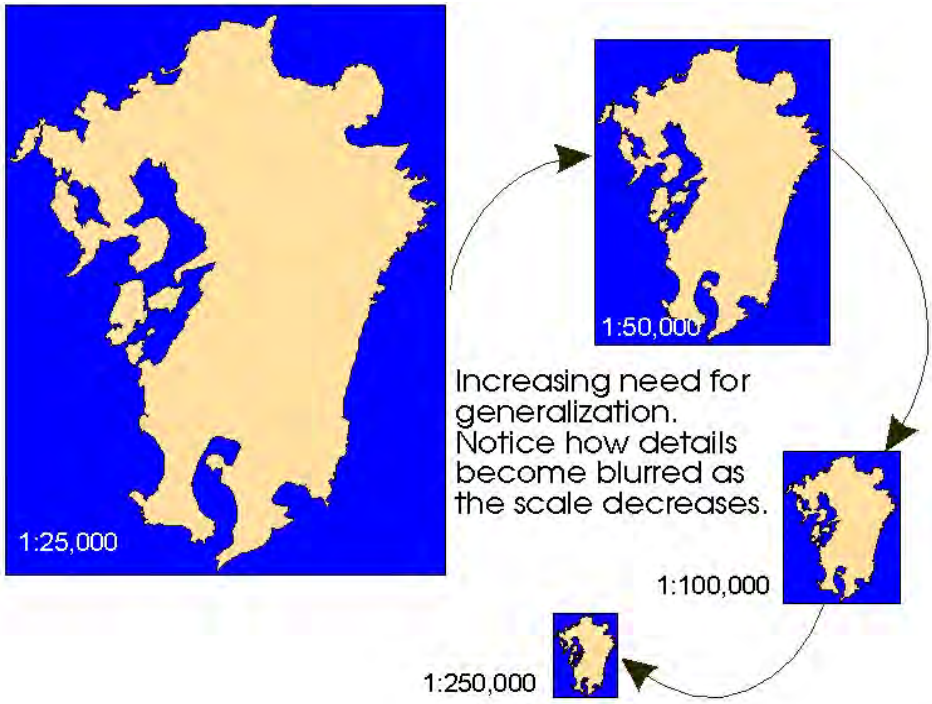
As section 32.2 notes, the projection used to create a map influences the representation of area, distance, direction, and shape. This is readily apparent when looking at a flat map of the world versus looking at a spherical map of the world (i.e., a globe). Maps that ignore the natural shape of the earth distort the places they are trying to represent. It should be noted when these characteristics (e.g., area, distance, direction, and shape), are of prime importance to the interpretation of any map. Some widely used locational reference systems such as the U.S. State Plane Coordinate system and Universal Transverse Mercator system are based on predefined projective geometries that are implicit in the use of the coordinate systems themselves. GIS software packages make it easy for users to choose an appropriate map projection.

32.5 Using the Internet as a GIS Tool

The internet can be a valuable resource for GIS users looking for data. Many federal agencies provide digital data free for download that can be used with GIS. The Census Bureau, EPA, and the United States Geological Survey are all good sources of GIS data. For example, in addition to demographic data, the Census bureau distributes what are called **T**opologically **I**ntegrated **G**eographic **E**ncoding and **R**eferencing (TIGER) files. The TIGER/Line files are a digital database of geographic features, such as roads, lakes, political boundaries, and census statistical boundaries, available for the entire United States. The database contains tabular information about these features such as their location in latitude and longitude, the name, the type of feature, and other important attributes. GIS clearinghouses, universities, and data supply companies are also good places to look for data. A Web search engine can help users locate sites that contain the type of data needed for a given project.

Once users locate relevant data, they must then get the data onto their computer. GIS coverages can take up a lot of computer memory, so choosing the right file transfer method is very important. Many websites allow direct downloads. This type of transfer involves clicking a link and specifying a target directory. Other data providers require users to go through a **file transfer protocol** (FTP) site. FTP sites allow people to exchange large data files more readily than with other protocols.

Exhibit 32-4. Effect of Scale on Detail and Abstraction



Finally, the internet can serve as a resource for users looking for technical support or advice. Most users will find that GIS software manufacturers offer online support. Some companies even have online courses.

32.6 Current GIS Applications at EPA

EPA is an excellent source of GIS data and information for risk assessors. Several offices and branches can serve as resources for those interested in learning more about GIS and its uses, especially in the areas of landscape, land cover, and land use. GIS helps EPA integrate geo-spatial data on a region (e.g., landscape, elevation, climate, slope) with information about potential exposures to give risk assessors a comprehensive picture of that region's hazards.

Because projected land use may be an important input to air models, risk assessors may want more information on landscape change models. For an overview on this subject, see EPA's *Projecting Land-Use Change: A Summary of Models for Assessing the Effects of Community Growth and Change on Land-Use Patterns.*[8]

32.6.1 ORD/ESD

EPA's Office of Research and Development/Environmental Sciences Division (ORD/ESD) conducts research, development, and technology transfer programs on environmental exposures to ecological and human receptors. GIS is an important tool for the type of chemical and physical stressors characterization conducted, especially with ESD's emphasis on ecological exposure. The Division develops landscape and regional assessment capabilities through the use of advanced spatial monitoring and analysis techniques, such as remote sensing and GIS. For more information, go to http://www.epa.gov/nerlesd1/.

32.6.2 ATtILA

Another EPA resource is the Landscape Ecology Branch's ATtILA program, which stands for *Analytical Tools Interface for Landscape Assessments.* The Branch uses ATtILA, which is a GIS, to conducts multiple-stressor regional assessments based largely on geo-spatial landscape data. As part of these assessments, ATtILA generates complicated landscape metrics, which are quantitative measurements of the environmental condition or vulnerability of an area (e.g., ecological region). ATtILA provides an interface that allows users to easily calculate many common landscape metrics regardless of their level of GIS knowledge, despite the complexity of developing the metrics. Four metric groups are currently included in the package (e.g., Landscape Characteristics, Riparian Characteristics, Physical Characteristics, and Human Stresses). ATtILA runs within ArcView®, and is designed to be flexible enough to accommodate spatial data from a variety of sources. More information is available at: http://www.epa.gov/nerlesd1/land-sci/northern_california/attila/background.html.

32.6.3 ReVA

Also from EPA's ORD is the Regional Vulnerability Assessment (ReVA) program. This program is an approach to regional scale, priority-setting assessment meant to expand cooperation among the laboratories and centers of ORD, by integrating research on human and environmental health, ecorestoration, landscape analysis, regional exposure and process modeling, problem formulation, and ecological risk guidelines. Currently, ReVA is working in the Mid-Atlantic region to predict future environmental risk. This will help EPA prioritize efforts to protect and restore environmental quality efficiently and effectively. ReVA is being developed to identify those ecosystems most vulnerable to being lost or permanently harmed in the next 5 to 25 years and to determine which stressors are likely to cause the greatest risk. The goal of ReVA is not exact predictions, but identification of the undesirable environmental changes expected over the coming years.

Many functions work together to provide ReVA's regional assessment capability. GIS puts into a spatial context data on stressors and effects from many sources. Research guides how to apply this data at the landscape and regional scale and helps EPA understand how socioeconomic drivers affect environmental condition. The transfer of data and analytical tools to regional managers is also critical for this tool to be useful. ReVA is considered a GIS because it is designed to analyze the spatial distribution of sensitive ecosystems by analyzing known

distributions of plant and animal populations or communities within ecosystems. Modern methods in landscape ecology and characterization help further identify the locations of ecosystems that are vulnerable to future stress through features such as topography (i.e. increased erosion potential) and habitat patch configurations. Multimedia assessments across water, air, terrestrial, and demographic variables are possible at various scales with this tool. For more information on ReVA, see http://www.epa.gov/reva/approach.htm.

32.7 GPS Technology

Global Positioning System (GPS) technology can be integrated with GIS. GPS technology allows users with the appropriate technology to obtain almost the exact location of any GPS receiver. This means that cars can get driving directions while moving, hikers can always know their exact position for navigating in and out of the wilderness, and the military can track movements of troops or vehicles. For risk assessments, the location of specific sources (i.e., vents) or receptor locations can be accurately determined with GPS. GPS is funded and controlled by the U.S. Department of Defense (DOD). While there are many thousands of civil users of GPS world-wide, the system was designed for, and is operated by the U.S. military.

The system works through specially coded satellite signals that can be processed in a GPS receiver, enabling the receiver to compute position, velocity, and time (see Exhibit 32-5). Four GPS satellite signals are used to compute positions in three dimensions and the time offset in the receiver clock (see Exhibit 32-6).

The GPS provides two levels of service – a Standard Positioning Service (SPS), and a Precise Positioning Service (PPS). Access to the PPS is restricted to U.S. Armed Forces, U.S. Federal agencies, and selected allied armed forces and governments. The SPS is available to all users on a continuous, worldwide basis, free of any direct user charge. A nationwide differential GPS service (NDGPS) is being established pursuant to the authority of Section 346 of the Department of Transportation and Related Agencies Appropriation Act. When complete, this service will provide uniform differential GPS coverage of the continental U.S. and selected portions of Hawaii and Alaska regardless of terrain, man-made, and other surface obstructions. NDGPS accuracy is specified to be 10 meters or better. Typical system performance is better than 1 meter in the vicinity of the broadcast site. Achievable accuracy degrades at an approximate rate of 1 meter for each 150 km distance from the broadcast site.[9]

Receiver costs vary depending on capabilities. Small civil SPS receivers can be purchased for under $200. Receivers that can store files for post-processing cost more ($2,000 to 5,000). Receivers that can act as DGPS reference receivers (computing and providing correction data) and carrier phase tracking receivers (and two are often required) can cost many thousands of dollars ($5,000 to $40,000).

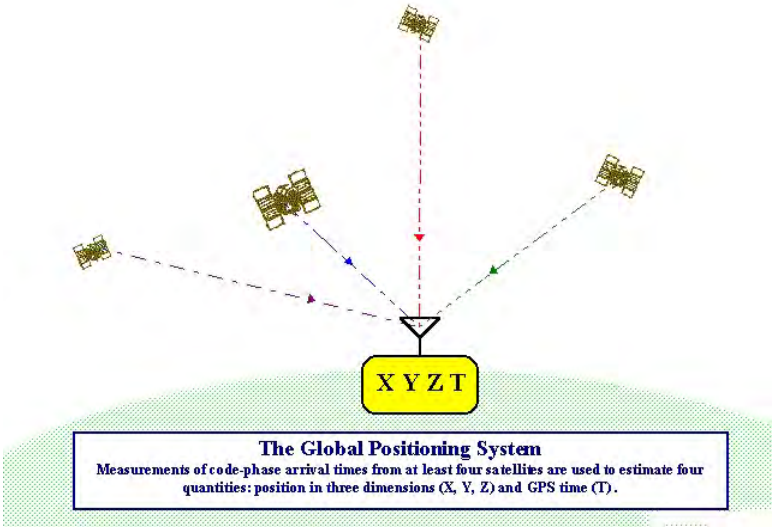
Receivers are important because they are the intermediary part of the system that connect real world data to GIS. Satellites send signals to the receiver and users and store the information. Sometimes, the user will have to manually record position and time readings and then type those

into a computer later. Other times the user can plug the receiver into a special port on her computer and download the digital data directly.

Exhibit 32-5. Global Positioning System (GPS) Satellites

GPS satellites orbit the Earth every 12 hours, sending signals to receivers around the world

Exhibit 32-6. Positioning and Time from Four GPS Satellites



Measurements of code-phase arrival times from at least four satellites are used to estimate four quantities: position in three dimensions (X, Y, and Z) and GPS time (T).

References

1. U.S. Environmental Protection Agency. 2004. *Envirofacts Data Warehouse.* Available at: http://www.epa.gov/enviro/index.html (Last accessed March 2004).

2. Agency for Toxic Substances and Disease Registry. 2004. *Introduction to ArcView and Spatial Analysis Techniques for Public Health Professionals.* An ATSDR/SAAGIS publication (in press).

3. Clarke, K.C. 1997. *Getting Started with Geographic Information Systems.* Prentice Hall, Upper Saddle River, NJ.

4. Plane, D.A., and Rogerson, P.A. 1994. *The Geographical Analysis of Population.* John Wiley & Sons, Inc, New York, NY.

5. Weiss, M.J. 2000. *The Clustered World.* Little, Brown and Company, Boston, MA.

6. Pollard, W., and Kirby, S. 1999. *Geographical Information Systems (GIS), Public Health Data, and Syndicated Market Research Data Bases in Health Communication.* Presented at the National Conference on Health Statistics, Washington, D.C.

7. Kenneth E.F. and Crum, S. The Geographer's Craft Project. Department of Geography, University of Texas at Austin.

8. U.S. Environmental Protection Agency. 2000. *Projecting Land-Use Change: A Summary of Models for Assessing the Effects of Community Growth and Change on Land-Use Patterns*. Office of Research and Development, Cincinnati, OH. EPA/600/R00/098.

9. U.S. Department of Defense and U.S. Department of Transportation. 2001. *2001 Federal Radionavigation Plan*, DOT/VNTSC/RSPA-01/3; DOD-4650.5. Available at: http://www.navcen.uscg.gov/pubs/frp2001/FRP2001.pdf. (Last accessed March 2004.)

Glossary

This list of glossary terms was compiled from existing EPA definitions and supplemented, where necessary, by additional terms and definitions. The wording of selected items may have been modified from the original in order to assist readers who are new to risk assessment more readily comprehend the underlying concept of the glossary entry. As such, these glossary definitions constitute neither official EPA policy nor preempt or in any way replace any existing legal definition required by statute or regulation.

A

Absorbed Dose – the amount of a substance that has penetrated the absorption barriers (e.g., skin, lung tissue, gastrointestinal tract) of an organism through either physical or biological processes.

Absorption - The process of taking in, as when a sponge takes up water. Chemicals can be absorbed through the skin into the bloodstream and then transported to other organs. Chemicals can also be absorbed into the bloodstream after breathing in or swallowing.

Absorption Barrier - Exchange barriers of the body that allow differential diffusion of various substances across a boundary. Examples of absorption barriers are the skin, lung tissue, and gastrointestinal tract wall.

Abiotic Degradation - Degradation via purely physical or chemical mechanisms. Examples include hydrolysis and photolysis.

Acceptable Risk - The likelihood of suffering disease or injury that will be tolerated by an individual, group, or society. The level of risk that is determined to be acceptable may depend on a variety of issues, including scientific data, social, economic, legal, and political factors, and on the perceived benefits arising from a chemical or process.

Accuracy - The measure of the correctness of data, as given by the difference between the measured value and the true or standard value.

Active Monitor - A type of personal exposure monitoring device that uses a small air pump to draw air through a filter, packed tube, or similar device.

Activity Patterns - A series of discrete events of varying time intervals describing information about an individual's lifestyle and routine. This information typically includes the locations visited, the amount of time spent in the locations, and a description of what the individual was doing in each location.

Acute Effect - Any toxic effect produced with a short period of time following an exposure, for example, minutes to a few days

Acute Exposure Limits - A variety of short-term exposure limits to hazardous substances, designed to be protective of human health. Published by different organizations, each limit has a different purpose and definition.

Acute Exposure - One dose (or exposure) or multiple doses (or exposures) occurring within a short time relative to the life of a person or other organism (e.g., approximately 24 hours or less for humans).

Actual Risk - The damage to life, health, property, and/or the environment that may occur as a result of exposure to a given hazard. Risk assessment attempts to estimate the likelihood of actual risk.

Additive Effect - The overall result of exposure to two or more chemicals, in which the resulting effect is equal to the sum of the independent effects of the chemicals. "Effects" or "Response Addition" is a method employed in EPA risk assessments of mixtures in which the components act or are presumed to act independently (without interaction).

Additive Dose - The overall result of exposure to two or more chemicals, when each chemical behaves as a concentration or dilution of the other chemicals in the mixture. The response of the combination is the response expected from the equivalent dose of an index chemical. The equivalent dose is the sum of component doses scaled by their toxic potency relative to the index chemical.

Adjusted Exposure Concentration - Also called a refined exposure concentration, an estimate of exposure concentration that has been refined, usually by application of an exposure model, to better understand how people in a particular location interact with contaminated media.

Administered Dose - The amount of a substance received by a test subject (human or animal) in determining dose-response relationships, especially through ingestion or inhalation.

Advection - In meteorology, the transfer of a property, such as heat or humidity, by motion within the atmosphere, usually in a predominantly horizontal direction. Thermal advection, for example, is the transport of heat by the wind. Advection is most often used to signify horizontal transport but can also apply to vertical movement. Large-scale horizontal advection of air is a characteristic of middle-latitude zones and leads to marked changes in temperature and humidity across boundaries separating air masses of differing origins.

Adverse Environmental Effect - Defined in the CAA section 112(a)(7) as "any significant and widespread adverse effect, which may reasonably be anticipated, to wildlife, aquatic life, or other natural resource, including adverse impacts on populations of endangered or threatened species or significant degradation of environmental quality over broad areas."

Adverse Health Effect - A health effect from exposure to air contaminants that may range from relatively mild and temporary (e.g., eye or throat irritation, shortness of breath, or headaches) to permanent and serious conditions (e.g., birth defects, cancer or damage to lungs, nerves, liver, heart, or other organs), and which negatively affects an individual's health or well-being, or reduces an individual's ability to respond to an additional environmental challenge.

Affected (or Interested) Parties - Individuals and organizations potentially acted upon or affected by chemicals, radiation, or microbes in the environment or influenced favorably or adversely by proposed risk management actions and decisions.

Agent - A chemical, physical, or biological entity that may cause deleterious, beneficial, or no effects to an organism after the organism is exposed to it.

Aggregate exposure - The combined exposure of an individual (or defined population) to a specific agent or stressor via relevant routes, pathways, and sources.

Aggregate risk - The risk resulting from aggregate exposure to a single agent or stressor.

AirData - An EPA website (http://www.epa.gov/air/data/info.html) that provides access to yearly summaries of United States air pollution data, taken from EPA's air pollution databases. The data include all fifty states plus District of Columbia, Puerto Rico, and the U. S. Virgin Islands. AirData has information about where air pollution comes from (emissions) and how much pollution is in the air outside our homes and work places (monitoring).

Air Emissions - The release or discharge of a pollutant into the air.

Air Pressure (Atmospheric Pressure, Barometric Pressure) - The pressure experienced above the Earth's surface at a specific point as a result of the weight of the air column, extending to the outer limit or top of the atmosphere. Consequently, pressure declines exponentially with height, the rate of decrease being a function of the temperature of the atmosphere. Atmospheric pressure is generally measured, in meteorology, either in the SI unit hectopascals (hPa) or in the c.g.s. unit of the same size, the millibar (mb) using a mercury or aneroid barometer, or a barograph. In the U.S., surface atmosphere pressure is measured in inches of mercury (Hg).

Air Mass - A large volume of air with certain meteorological or polluted characteristics (e.g., a heat inversion or smogginess) while in one location. The characteristics can change as the air mass moves away.

Air Toxic - Any air pollutant that causes or may cause cancer, respiratory, cardiovascular, or developmental effects, reproductive dysfunctions, neurological disorders, heritable gene mutations, or other serious or irreversible chronic or acute health effects in humans. See hazardous air pollutant.

Ambient Medium (e.g., Ambient Air) - Material surrounding or contacting an organism (e.g., outdoor air, indoor air, water, or soil), through which chemicals can reach an organism.

Ambient Water Quality Criteria (AWQC) - A ecological benchmark level for aquatic contaminants, published by EPA Office of Water, which is designed to protect 95 percent of all aquatic species in freshwater or marine environments. Criteria have been developed for both acute and chronic exposures, although for a limited number of chemicals.

Ample Margin of Safety - This term has regulatory significance in EPA's air toxics program. It was interpreted by the Agency in the 1989 notice of final benzene NESHAP (FR54:38044-38072), and reiterated in the 1990 amendments to the Clean Air Act (sections 112(f) and 112(c)).

AMTIC - Ambient Monitoring Technology Information Center. An EPA website that contains information and files on ambient air quality monitoring programs, details on monitoring methods, monitoring-related documents and articles, information on air quality trends and nonattainment areas, and federal regulations related to ambient air quality monitoring. [http://www.epa.gov/ttn/amtic/, 2003]

Analysis - The systematic application of specific theories and methods, including those from natural science, social science, engineering, decision science, logic, mathematics, and law, for the purpose of collecting and interpreting data and drawing conclusions about phenomena. It may be qualitative or quantitative. Its competence is typically judged by criteria developed within the fields of expertise from which the theories and methods come.

Analysis Plan - A plan that provides all the details of exactly how each part of the risk assessment will be performed. It usually describes in detail what analyses will be performed, how they will be performed, who will perform the work, schedules, resources, quality assurance/quality control requirements, and documentation requirements.

Animal Studies - Toxicity investigations using animals. Such studies may employ animals as surrogates for humans with the expectation that the results are pertinent to humans or for investigation of effects pertinent to animals (e.g., for ecological risk assessment).

Antagonistic Effect - The situation where exposure to two chemicals together has less effect than the sum of their independent effects.

AP-42 - A compilation of air pollutant emission factors. Volume I of the fifth edition addresses stationary point and area source emission factors. AP-42 is accessible on the Air CHIEF website (http://www.epa.gov/ttn/chief/ap42/) and is also included on the Air CHIEF CD-ROM.

Applied Dose - The amount of a substance in contact with an absorption boundary of an organism (e.g., skin, lung, gastrointestinal tract) and is available for absorption.

Area of Impact – The geographic area affected by a facility's emissions (also known as the zone of impact).

Area Source (legal sense) - A stationary source that emits less than 10 tons per year of a single hazardous air pollutant (HAP) or 25 tons per year of all HAPs combined.

Area Source (modeling sense) - An emission source in which releases are modeled as coming from a 2-dimensional surface. Emissions from the surface of a wastewater pond are, for example, often modeled as an area source.

Area Use Factor - For an animal, the ratio of its home range, breeding range, or feeding/foraging range to the area of contamination or the site area under investigation.

Assessment Endpoint - An explicit expression of the environmental value to be protected. An assessment endpoint includes both an ecological entity and specific attributes of that entity. For example, salmon are a valued ecological entity; reproduction and population maintenance (i.e., the attribute) form an assessment endpoint.

Assessment Questions - The questions asked during the planning/scoping phase of the risk assessment process to determine what the risk assessment will evaluate.

Atmospheric Stability (Stability) - the degree of resistance of a layer of air to vertical motion.

ATSDR (Agency for Toxic Substances and Disease Registry) - An Agency of the U.S. Department of Health and Human Services, whose goal is to serve the public by using the best science, taking responsive public health actions, and providing health information to prevent harmful exposures and diseases to toxic substances. Its website (www.atsdr.cdc.gov) includes information on hazardous substances [e.g., toxicological profiles, minimal risk levels (MRLs)], emergency response, measuring health effects, hazardous waste sites, education and training, publications, and special issues (e.g., Children Health).

Averaging Time - The time period over which something is averaged (e.g., exposure, measured concentration).

B

Background Levels - The concentration of a chemical already present in an environmental medium due to sources other than those under study. Two types of background levels may exist for chemical substances: (a) Naturally occurring levels of substances present in the environment, and (b) Anthropogenic concentrations of substances present in the environment due to human associated activities (e.g., automobiles, industries).

Background Source - Any source from which pollutants are released and contribute to the background level of a pollutant, such as volcano eruptions, windblown dust, or manmade source upwind of the study area.

Benchmark Dose - An exposure due to a dose of a substance associated with a specified low incidence of risk, generally in the range of 1% to 10%, of a health effect; or the dose associated with a specified measure or change of a biological effect.

Benthic Burial Rate (k_b) - Rate of the deposition of the sediment suspended in a surface water body column to the benthic sediment surface that becomes no longer available for resuspension in the water column, effectively becoming part of the sediment "sink."

Best Available Control Technology (BACT) - An emission limitation based on the maximum degree of emission reduction (considering energy, environmental, and economic impacts) achievable through application of production processes and available methods, systems, and techniques. BACT does not permit emissions in excess of those allowed under any applicable Clean Air Act provisions. Use of the BACT concept is allowable on a case by case basis for major new or modified emissions sources in attainment areas and applies to each regulated pollutant.

Best Professional Judgement - Utilizing knowledge based on education and experience to determine the best course of action during the course of performing a risk assessment project.

Bias - systematic error introduced into sampling or analysis by selecting or encouraging one outcome or answer over others.

Binational Toxics Strategy - A Canada-United States jointly-sponsored program that provides a framework for actions to reduce or eliminate persistent toxic substances, especially those which bioaccumulate, from the Great Lakes Basin.

Bioaccumulation - The net accumulation of a substance by an organism as a result of uptake from and or all routes of exposure (e.g., ingestion of food, intake of drinking water, direct contact, or inhalation).

Bioavailability - The ability to be absorbed and available to interact with the metabolic processes of an organism.

Bioaccumulation Factor (BAF) - The concentration of a substance in tissue of an organism divided by its concentration in an environmental medium in situations where the organism and its food are exposed (i.e., accounting for food chain exposure as well as direct chemical uptake). [EPA, 1999: Residual Risk Report to Congress. EPA453R99001.]

Bioassay - A test conducted in living organisms (*in vivo*) or with living cells (*in vitro*) to determine the hazard or potency of a chemical by its effect on animals, isolated tissues, or microorganisms. [Based on Air Risk Information Support Center, OAQPS, March 1989: Glossary of Terms Related to Health, Exposure, and Risk Assessment. EPA/450/3-88/016.]

Bioavailability - A measure of the degree to which a dose of a substance becomes physiologically available to the body tissues depending upon adsorption, distribution, metabolism and excretion rates. [Air Risk Information Support Center, OAQPS, March 1989: Glossary of Terms Related to Health, Exposure, and Risk Assessment. EPA/450/3-88/016.]

Bioconcentration - The net accumulation of a substance by an organism as a result of uptake directly from an environmental medium (e.g., net accumulation by an aquatic organism as a result of uptake directly from ambient water, through gill membranes or other external body surfaces).

Bioconcentration Factor (BCF) - The concentration of a substance in tissue of an organism divided by the concentration in an environmental medium (e.g., the concentration of a substance in an aquatic organism divided by the concentration in the ambient water, in situations where the organism is exposed through the water only).

Biological Medium - Any one of the major categories of material within an organism (blood, adipose tissue, breath), through which chemicals can move, be stored, or be biologically, physically, or chemically transformed.

Biological Monitoring - The measurement of chemicals in biological media (e.g., blood, urine, exhaled breath) to determine whether chemical exposure in humans, animals, or plants has occurred.

Biologically Effective Dose - The amount of chemical that reaches the cells or target site where an adverse effect may occur.

Biomagnification or Biological Magnification - The process whereby certain substances, such as pesticides or heavy metals, transfer up the food chain and increase in concentration. For example, a biomagnifying chemical deposited in rivers or lakes absorbs to algae, which are ingested by aquatic organisms, such as small fish, which are in turn eaten by larger fish, fish-

eating birds, terrestrial wildlife, or humans. The chemical tends to accumulates to higher concentration levels with each successive food chain level.

Biotic Degradation (Biodegredation) - Decomposition or metabolism of a substance into more elementary compounds by the action of organisms (e.g., bacteria, fungi).

Bounding Estimate - An estimate of exposure or risk that is higher or lower than that incurred by any person in the population. Bounding estimates are useful in developing statements that exposures or risks are within an estimated range.

Blue Book - The 1994 National Research Council (NRC) report entitled *Science and Judgement in Risk Assessment*.

Body Weight (Mass) - The weight or mass of an individual's body. It can apply to a human or an ecological receptor.

Breathing Zone - Air in the vicinity of an organism from which respired air is drawn. Personal monitors are often used to measure pollutants in the breathing zone.

Bright Line - Specific levels of risk or of exposure that are meant to provide a practical distinction between what is considered "safe" and what is not.

Building Downwash (Plume Downwash) - The interaction of a plume with a structure, such as a building, which causes the plume to fall to ground.

C

CalEPA (California Environmental Protection Agency) - An Agency within the California State government whose goal is to protect human health and the environment and to assure the coordinated deployment of State resources against the most serious environmental risks. There are six boards that address environmental issues, including air quality, pesticides, toxic substances, waste management, water control, and the Office of Environmental Health Hazard Assessment (OEHHA). Note that OEHHA is responsible for developing and providing state and local government agencies with toxicological and medical information relevant to decisions involving public health and is a good resource for such information.

Cancer - A group of related diseases characterized by group of diseases characterized by the uncontrolled growth of abnormal cells.

Cancer Incidence - The number of new cases of a disease diagnosed each year.

Cancer Risk Estimates - The probability of developing cancer from exposure to a chemical agent or a mixture of chemicals over a specified period of time. In quantitative terms, risk is expressed in values ranging from zero (representing an estimate that harm certainly will not occur) to one (representing an estimate that harm certainly will occur). The following are examples of how risk is commonly expressed: 1.E-04 or 1×10^{-4} = a risk of 1 additional cancer in an exposed population of 10,000 people (i.e., 1/10,000); 1.E-5 or 1×10^{-5} = 1/100,000; 1.E-6 or 1×10^{-6} = 1/1,000,000.

Cancer Risk Evaluation Guides (CREGs) - Developed by ATSDR, the concentration of a chemical in air, soil or water that is expected to cause no more than one excess cancer in a million persons exposed over a lifetime. The CREG is a *comparison value* used to select contaminants of potential health concern and is based on the *cancer slope factor* (CSF).

Cancer Slope Factor (CSF) - An upper bound (approximating a 95% confidence limit) on the increased cancer risk from a lifetime exposure to an agent. This estimate, usually expressed in units of proportion (of a population) affected per mg/kg/day, is generally reserved for use in the low-dose region of the dose-response relationship; that is, for exposures corresponding to risks less than 1 in 100. This term is usually used to refer to oral slope factors (i.e., slope factors used for assessing ingestion exposure).

Carcinogen(ic) - An agent capable of inducing cancer.

Carcinogenesis - The origin or production of a benign or malignant tumor. The carcinogenic event modifies the genome and/or other molecular control mechanisms of the target cells, giving rise to a population of altered cells.

Census Bureau (Bureau of the Census) - A Bureau within the Department of Commerce, this is the country's preeminent statistical collection and dissemination agency of national demographic information. It publishes a wide variety of statistical data about people, housing, and the economy of the nation. The Census Bureau conducts approximately 200 annual surveys and conducts the decennial census of the United States population and housing and the quinquennial economic census and census of governments.

Census Block - An area bounded by visible and/or invisible features shown on Census Bureau maps. A block is the smallest geographic entity for which the Census Bureau collects and tabulates 100-percent decennial census data.

Census Tract - A small, relatively permanent statistical subdivision of a county or statistically equivalent entity, delineated for data presentation purposes by a local group of census data users or the geographic staff of a regional census center in accordance with Census Bureau guidelines. Designed to be relatively homogeneous units with respect to population characteristics, economic status, and living conditions at the time they are established, census tracts generally contain between 1,000 and 8,000 people, with an optimum size of 4,000 people. Census tract boundaries are delineated with the intention of being stable over many decades, so they generally follow relatively permanent visible features. However, they may follow governmental unit boundaries and other invisible features in some instances; the boundary of a state or county (or statistically equivalent entity) is always a census tract boundary.

Census Tract (or Census Block) Internal Point - A set of geographic coordinates (latitude and longitude) that is located within a specified geographic entity such as a Census Tract or Census Block. For many Census Tracts or Blocks, this point represents the approximate center of the Census Tract or Block; for some, the shape of the entity or the presence of a body of water causes the central location to fall outside the Census Tract or Block or in water, in which case the point is relocated to land area within the Census Tract or Block. The geographic coordinates are shown in degrees to six decimal places in census products.

Chemical Abstracts Service Registry Number (CASRN) - A unique, chemical-specific number used in identifying a substance. The registry numbers are assigned by the Chemical Abstract Service, a division of the American Chemical Society. (Note that some mixtures of substances, such as mixtures of various forms of xylene, are also given CAS numbers.)

Chemicals of Potential Concern - Chemicals that may pose a threat to the populations within the study area. These are the chemicals which are carried through the risk assessment process.

Chemical Speciation - Detailed identification of the specific identities and forms of chemicals in a mixture.

Chemical Transformation - The change of one chemical into another.

Chronic Exposure - Continuous exposure, or multiple exposures, occurring over an extended period of time or a significant fraction of the animal's or the individual's lifetime.

Chronic Health Effects - An effect which occurs as a result of repeated or long term (chronic) exposures.

Coefficient of Variation (CV) - A dimensionless measure of dispersion, equal to the standard deviation divided by the mean, often expressed as a percentage.

Cohort - A group of people within a population that can be aggregated because the variation in a characteristic of interest (e.g., exposure, age, education level) within the group is much less than the group-to-group variation across the population.

Community - The persons associated with an area who may be directly affected by area pollution because they currently live in or near the area, or have lived in or near the area in the past (i.e., current or past residents), members of local action groups, local officials, tribal governments, health professionals, and local media. Other entities, such as local industry, may also consider themselves part of the community.

Comparative Risk Assessment - The process of comparing and ranking various types of risks to identify priorities and influence resource allocations.

Conceptual Model - A written description and/or a visual representation of actual or predicted relationships between humans or ecological entities and the chemicals or other stressors to which they may be exposed.

Conductivity (Conductance) - The ability of a material to carry and electrical current.

Confidence Interval - A range of values that has a specified probability (e.g., 95 percent) of containing the statistical parameter (i.e., a quantity such as a mean or variance that describes a statistical population) in question. The confidence limit refers to the upper or lower value of the range.

Coning - In pollution studies, emissions from a chimney stack under atmospheric conditions of near neutral stability such that concentrations of a pollutant at a given distance downwind from

the stack may be described by a normal or Gaussian distribution, being the same for both vertical and horizontal cross-sections perpendicular to the flow.

Consumption Rate - The average quantity of an item consumed or expended during a given time interval, expressed in quantities by the most appropriate unit of measurement per applicable stated basis.

Continuous Monitoring - The measurement of the air or water concentration of a specific contaminant on an uninterrupted, real-time basis by instrumental methods.

Control Technology/Measures - Equipment, processes or actions used to reduce air pollution at the source.

Convection - The transfer and mixing of heat by mass movement through a fluid (e.g., air or water). It is one of the major mechanisms for the transfer of heat within the atmosphere, together with conduction and radiation. The convection process is of major importance in the troposphere, transferring sensible heat and latent heat from the Earth's surface into the boundary layer, and by promoting the vertical exchange of air-mass properties (e.g., heat, water vapor, and momentum) throughout the depth of the troposphere. Convection is generally accepted to be vertical circulation, whereas advection is usually horizontal.

Cost-Benefit Analysis - An evaluation of the costs which would be incurred versus the overall benefits of a proposed action, such as the establishment of an acceptable exposure level of a pollutant.

Criteria Air Pollutant - One of six common air pollutants determined to be hazardous to human health and regulated under EPA's National Ambient Air Quality Standards (NAAQS). The six criteria air pollutants are carbon monoxide, lead, nitrogen dioxide, ozone, sulfur dioxide, and particulate matter. The term "criteria pollutants" derives from the requirement that EPA must describe the characteristics and potential health and welfare effects of these pollutants. It is on the basis of these criteria that standards are set or revised.

Critical Effect - The first adverse effect, or its known precursor, that occurs to the most sensitive species as the dose rate of an agent increases.

Cumulative Risk - The combined risk from aggregate exposures to multiple agents or stressors.

Cumulative Risk Assessment - An analysis, characterization, and possible quantification of the combined risks to health or the environment from multiple agents or stressors.

Cumulative Distribution Function (CDF) - The CDF is alternatively referred to in the literature as the distribution function, cumulative frequency function, or the cumulative probability function. The cumulative distribution function, $F(x)$, expresses the probability the random variable X assumes a value less than or equal to some value x, $F(x) = \text{Prob}(X \leq x)$. For continuous random variables, the cumulative distribution function is obtained from the probability density function by integration, or by summation in the case of discrete random variables.

Cumulative Risk Assessment - An analysis, characterization, and possible quantification of the combined risks to health or the environment from multiple agents or stressors.

D

Data Integrity - Refers to security (i.e., the protection of information from unauthorized access or revision) to ensure that the information is not compromised through corruption or falsification. Data integrity is one of the constituents of data quality.

Data Objectivity - A characteristic indicating whether information is being presented in an accurate, clear, complete, and unbiased manner, and as a matter of substance, is accurate, reliable, and unbiased. Data objectivity is one of the constituents of data quality.

Data Quality - The encompassing term regarding the quality of information used for analysis and/or dissemination. Utility, objectivity, and integrity are constituents of data quality.

Data Quality Objectives (DQOs) - Qualitative and quantitative statements derived from the DQO process that clarify study objectives, define the appropriate type of data, and specify tolerable levels of potential decision errors that will be used as the basis for establishing the quality and quantity of data needed to support the decisions.

Data Quality Objectives Process - A systematic planning tool to facilitate the planning of environmental data collection activities. Data quality objectives are the qualitative and quantitative outputs from the DQO Process.

Data Utility - Refers to the usefulness of the information to the intended users. Data utility is one of the constituents of data quality.

Delivered Dose - The amount of the chemical available for interaction by any particular organ or cell.

Deposition (Wet and Dry) - The removal of airborne substances to available surfaces that occurs as a result of gravitational settling and diffusion, as well as electrophoresis and thermophoresis in the absence of active precipitation (Dry) or in the presence of active precipitation (Wet).

Deposition (Flux) - The removal of airborne substances from the air to available surfaces that occurs as a result of gravitational settling and diffusion, as well as electrophoresis and thermophoresis.

Dermal - Referring to the skin. Dermal absorption means absorption through the skin.

Dermal Exposure - Contact between a chemical and the skin. [EPA, 1997: Terms of Environment, http://www.epa.gov/OCEPAterms/.]

Detection Limit - The lowest concentration of a chemical that can reliably with analytical methods be distinguished from a zero concentration.

Deterministic - A methodology relying on point (i.e., exact) values as inputs to estimate risk; this obviates quantitative estimates of uncertainty and variability. Results are also presented as point values. Uncertainty and variability may be discussed qualitatively, or semi-quantitatively by multiple deterministic risk estimates.

Developmental Toxicity - The potential of an agent to cause abnormal development. Developmental toxicity generally occurs in a dose-related manner, may result from short-term exposure (including single exposure situations) or from longer term low-level exposure, may be produced by various routes of exposure, and the types of effects may vary depending on the timing of exposure because of a number of critical periods of development for various organs and functional systems. The four major manifestations of developmental toxicity are death, structural abnormality, altered growth, and functional deficit.

Dietary Composition - The fractions of different foods that constitute a given diet.

Differential Heating - The property of different surfaces which causes them to heat and cool at different rates.

Direct Exposure - Contact between a receptor and a chemical where the chemical is still in the medium to which it was originally released. For example, direct exposure occurs when a pollutant is released to the air and a person breathes that air.

Direct-read Monitor - Using a pump to draw the air sample through the detector, this type of air toxics monitoring device provides a direct reading of the pollutant measurement. The monitor may be designed as a table-top unit, for example, or it may be rack-mounted such as for use in an ambient air monitoring station.

Dispersion - Pollutant or concentration mixing due to turbulent physical processes.

Disease Cluster - An unusual number, real or perceived, of health events (i.e., reports of cancer) grouped together in time and location.

Dose - The amount of substance available for interaction with metabolic processes or biologically significant receptors after crossing the outer boundary of an organism. The potential dose is the amount ingested, inhaled, or applied to the skin. The applied dose is the amount of a substance presented to an absorption barrier and available for absorption (although not necessarily having yet crossed the outer boundary of the organism). The absorbed does is the amount crossing a specific absorption barrier (e.g., the exchange boundaries of skin, lung, and digestive tract) through uptake processes. Internal dose is a more general term denoting the amount absorbed without respect to specific absorption barriers or exchange boundaries. The amount of the chemical available for interaction by any particular organ or cell is termed the delivered dose for that organ or cell.

Dose-Response Assessment - A determination of the relationship between the magnitude of an administered, applied, or internal dose and a specific biological response. Response can be expressed as measured or observed incidence, percent response in groups of subjects (or populations), or as the probability of occurrence within a population.

Dose-Response Curve - A graphical representation of the quantitative relationship between administered, applied, or internal dose of a chemical or agent, and a specific biological response to that chemical or agent.

Dust Resuspension - Involves the deposition of dust from the air and its subsequent resuspension or re-entrainment into the atmosphere.

E

Ecological Risk Assessment - The process that evaluates the likelihood that adverse ecological effects may occur or are occurring as a result of exposure to one or more stressors.

Eddy - In the atmosphere, a distinct mass within a turbulent fluid that retains its identity and behaves differently for a short period within the general larger volume flow. An eddy thus ranges in size from microscale turbulence (1 cm for example) to many hundreds of kilometers in the form of frontal cyclones and anticyclones. The smallest scale eddies are critical in the process of, for example, heat and water vapor transfer from the Earth's surface into the air, while frontal cyclones transport heat toward the poles.

Emission Factor - The relationship between the amount of pollution produced and the amount of raw material processed or product produced. For example, an emission factor for a blast furnace making iron could be the number of pounds of particulates released per ton of raw materials used.

Emission Inventory - A listing, by source, of the amount of air pollutants discharged into the atmosphere in a particular place. Two of the more important publicly available emissions inventories for air toxics studies are the National Emissions Inventory (NEI) and the Toxics Release Inventory (TRI).

Emission Rate - The amount of a given substance discharged to the air per unit time, expressed as a fixed ratio (e.g., tons/yr).

Emissions Inventory Improvement Program (EIIP) - A jointly sponsored effort of the State and Territorial Air Pollution Program Administrators/Association of Local Air Pollution Control Officials (STAPPA/ALAPCO) and EPA, and is an outgrowth of the Standing Air Emissions Work Group (SAEWG). The goal of EIIP is to provide cost-effective, reliable inventories by: (1) Improving the quality of emissions information, and (2) Developing system(s) for collecting, calculating, and reporting emissions data. The goal is achieved by developing a set of "preferred and alternative methods" for all inventory associated tasks. This standardization improves the consistency of collected data and results in increased usefulness of emissions information.

Emissions Monitoring - The periodic or continuous physical surveillance or testing to determine the pollutant levels discharged into the atmosphere from sources such as smokestacks at industrial facilities and exhaust from motor vehicles, locomotives, or aircraft.

Emissions Tracking System (ETS) - This EPA system contains all emissions data submitted under various clean air market programs. Data from Continuous Emissions Monitoring Systems at utilities sends the emission data to the utility's computer system, which then compiles the data for submission to EPA on a quarterly basis. At the end of each calendar year, EPA compares tons of emissions emitted with the allowance holdings of the utility unit to ensure that it is in compliance with the relevant program.

Endocrine Disruptor - Substances which interfere with endocrine system function.

Environmental Data - Any measurements or information that describe environmental processes, location, or conditions; ecological or health effects and consequences; or the performance of environmental technology. Environmental data include information collected directly from measurements, produced from models, and compiled from other sources such as data bases or the literature.

Environmental Media Evaluation Guides - Environmental Media Evaluation Guides (EMEGs) are concentrations of a contaminant in water, soil, or air that are unlikely to be associated with any appreciable risk of deleterious noncancer effects over a specified duration of exposure. EMEGs are derived from ATSDR minimal risk levels by factoring in default body weights and ingestion rates. Separate EMEGS are computed for acute (14 days), intermediate (15-364 days), and chronic (365 days) exposures.

Environmental Medium - Any one of the major categories of material found in the physical environment (e.g., surface water, ground water, soil, or air), and through which chemicals or pollutants can move.

Epidemiology - The study of disease patterns in human populations.

Epidemiologic Study, Case Study - A medical or epidemiologic evaluation of one person or a small group of people to gather information about specific health conditions and past exposures.

Epidemiologic Study, Descriptive - An evaluation of the amount and distribution of a disease in a specified population by person, place, and time.

Epidemiologic Study, Analytical - An evaluation of the association between exposure to hazardous substances and disease by testing scientific hypotheses.

Exposure - Contact made between a chemical, physical, or biological agent and the outer boundary of an organism.

Exposure Assessment - An identification and evaluation of a population exposed to a toxic agent, describing its composition and size, as well as the type, magnitude, frequency, route and duration of exposure.

Exposure Concentration - The concentration of a chemical in its transport or carrier medium (i.e., an environmental medium or contaminated food) at the point of contact.

Exposure Duration - The total time an individual is exposed to the chemical being evaluated or the length of time over which contact with the contaminant lasts.

Exposure Factors - Any of a variety of factors that relate to how an organism interacts with or is otherwise exposed to environmental pollutants (e.g., ingestion rate of contaminated fish). Such factors are used in the calculation of exposure to toxic chemicals.

Exposure Frequency - The number of occurrences in a given time frame (e.g., a lifetime) of contact or co-occurrence of a stressor with a receptor.

Exposure Investigation (in Public Health Assessment) - The collection and analysis of site-specific information and biologic tests (when appropriate) to determine whether people have been exposed to hazardous substances.

Exposure Modeling - The mathematical equations simulating how people interact with chemicals in their environment.

Exposure Pathway - The course a chemical or physical agent takes from a source to an exposed organism. An exposure pathway includes a source and release from a source, an exposure point, and an exposure route. If the exposure point differs from the source, a transport/exposure medium (e.g., air) or media (in cases of intermedia transfer) also is included.

Exposure Profile - The exposure profile (ecological) identifies the receptors and describes the exposure pathways and intensity and spatial and temporal extent of exposure. It also describes the impact of variability and uncertainty on exposure estimates and reaches a conclusion about the likelihood that exposure will occur. The profile may be a written document or a module of a larger process model.

Exposure Route - The way a chemical enters an organism after contact (e.g., by ingestion, inhalation, dermal absorption).

Exposure Scenario - A set of conditions or assumptions about sources, exposure pathways, concentrations of toxic chemicals, and populations (numbers, characteristics and habits) which aid the investigator in evaluating and quantifying exposure in a given situation.

Exposure Unit (in Geographical Information System applications) - The geographical area in which a receptor moves and contacts the contaminated medium during the period of exposure.

F

Factor Information Retrieval System (FIRE) - A database management system containing EPA's recommended emission estimation factors for criteria and hazardous air pollutants. FIRE includes information about industries and their emitting processes, the chemicals emitted, and the emission factors themselves. FIRE allows easy access to criteria and hazardous air pollutant emission factors obtained from the Compilation of Air Pollutant Emission Factors (AP-42), Locating and Estimating (L&E) documents, and the retired AFSEF and XATEF documents.

Fate and Transport - A description of how a chemical is carried through and changes in the environment.

Fate and Transport Analysis - The general process used to assess and predict the movement and behavior of chemicals in the environment.

Fate and Transport Modeling - The mathematical equations simulating a physical system which are used to assess and predict the movement and behavior of chemicals in the environment.

Fence Line - Delineated property boundary of a facility.

Field Study - Scientific study made in the ambient air to collect information that can not be obtained in a laboratory.

Food Chain - A sequence of organisms, each of which uses the next lower member of the sequence as a food source.

Forage - (1) Edible parts of plants, other than separated grain, that can provide feed for grazing animals or can be harvested for feeding, including browse, and herbage. (2) To search for or to consume forage (of animals).

Fugitive Release - Emission of a chemical to the air that does not occur from a stack, vent, duct, pipe or other confined air stream (e.g., leaks from joints).

Fumigation - (1) The use of a chemical compound in a gaseous state, often to kill pests such as insects, nematodes, arachnids, rodents, weeds, and fungi in confined or inaccessible locations or in the field. (2) a pattern of plume dispersion produced when a convective boundary layer grows upward into a plume trapped in a stable layer. The elevated plume is suddenly brought downward to the ground, producing high surface concentrations.

Future Scenario - A scenario used in risk assessment to anticipate potential future exposures of individuals (e.g., a housing development could be built on currently vacant land).

G

Geographic Information Systems (GIS) - A computer program that allows layering of different types of spatial information (i.e., on a map) to provide a better understanding of the characteristics of a certain place.

Generally Available Control Technology (GACT) Standard - These standards are less stringent standards than the Maximum Available Control Technology (MACT) standards, and are allowed at the Administrator's discretion for area sources according to the 1990 Clean Air Act Amendments for area sources.

Grab Sample -A single sample collected at a particular time and place that represents the composition of the water, air, or soil only at that time and place.

Great Waters Pollutants of Concern - The toxic pollutants of concern to the Great Waters program are mercury; cadmium and lead (and their compounds); dioxins; furans; polycyclic organic matter; polychlorinated biphenyls (PCBs); and the pesticides chlordane, DDT/DDE, dieldrin, hexachlorobenzene, alpha-hexachlorocyclohexane, lindane and toxaphene. Nitrogen compounds such as nitrogen oxides and ammonia are also pollutants of concern.

Greenhouse Effect - Trapping and build-up of heat in the atmosphere (troposphere) near the earth's surface. Some of the heat flowing back toward space from the earth's surface is absorbed by water vapor, carbon dioxide, ozone, and several other gases in the atmosphere and then re-radiated back toward the earth's surface. If the atmospheric concentrations of these greenhouse gases rise, the average temperature of the lower atmosphere will gradually increase.

Greenhouse Gas (GHG) - Any gas that absorbs infrared radiation in the atmosphere. Greenhouse gases include, but are not limited to, water vapor, carbon dioxide (CO_2), methane (CH_4), nitrous oxide (N_2O), hydrochlorofluorocarbons (HCFCs), ozone (O_3), hydrofluorocarbons (HFCs), perfluorocarbons (PFCs), and sulfur hexafluoride (SF_6).

Guidelines (human health and ecological risk assessment) - Official documentation stating current U.S. EPA methodology in assessing risk of harm from environmental pollutants to human populations and ecological receptors.

H

Half-Life - The time required for a reaction or process to proceed such that half of the original amount of the substance of interest has reacted or undergone the process. Examples include: (1) the time required for a pollutant to degrade to one-half of its original concentration; (2) the time required for half of the atoms of a radioactive element to undergo self-transmutation or decay (half-life of radium is 1620 years); (3) the time required for elimination from the body to half a total dose.

Hazard - In a general sense, "hazard" is anything that has a potential to cause harm. In risk assessment, the likelihood of experiencing a noncancer health effect is called hazard (not risk).

Hazard Identification - The process of determining whether exposure to an agent can cause a particular adverse health effect (e.g., cancer, birth defect) and whether the adverse health effect is likely to occur in humans at environmentally relevant doses.

Hazard Index (HI) -The sum of more than one hazard quotient for multiple substances and/or multiple exposure pathways. The HI is calculated separately for chronic, subchronic, and shorter-term duration exposures.

Hazardous Air Pollutants (HAP) - Defined under the Clean Air Act as pollutants that cause or may cause cancer or other serious health effects, such as reproductive effects or birth defects, or adverse environmental and ecological effects. Currently, the Clean Air Act regulates 188 chemicals and chemical categories as HAPs.

Hazard Quotient (HQ) - The ratio of a single substance exposure level over a specified time period (e.g., chronic) to a reference value (e.g., an RfC) for that substance derived from a similar exposure period.

Health Effects Assessment Tables (HEAST) - An older listing of (usually) interim toxicity values for chemicals of interest to Superfund, the Resource Conservation and Recovery Act (RCRA), and the EPA in general. HEAST values are generally placed low on the hierarchy of Agency recommended toxicity data sources and the compilation will eventually be phased out altogether.

Health Endpoint - An observable or measurable biological event used as an index to determine when a deviation in the normal function of the human body occurs.

Health Outcome Data (in Public Health Assessment) - Community-specific health information such as morbidity and mortality data, birth statistics, medical records, tumor and disease registries, surveillance data, and previously conducted health studies that may be collected at the local, state, and national levels by governments, private health care organizations, and professional institutions and associations.

Health Outcomes Study (in Public Health Assessment) - An investigation of exposed persons designed to assist in identifying exposure or effects on public health. Health studies also define the health problems that require further inquiry by means of, for example, a health surveillance or epidemiologic study.

Health Education (in Public Health Assessment) - Programs designed with a community to help it know about health risks and how to reduce these risks.

Health Consultation (in Public Health Assessment) - A review of available information or collection of new data to respond to a specific health question or request for information about a potential environmental hazard. Health consultations are focused on a specific exposure issue. Health consultations are therefore more limited than a public health assessment, which reviews the exposure potential of each pathway and chemical.

Henry's Law Constant - The ratio at equilibrium of the gas phase concentration to the liquid phase concentration of the gas.

High-End Exposure Estimate - A plausible estimate of individual exposure or dose for those persons at the upper end of an exposure or dose distribution, conceptually above the 90th percentile, but not higher than the individual in the population who has the highest exposure or dose.

Human Exposure Model (HEM) - An EPA model combining the Industrial Source Complex Short Term air dispersion model (ISCST) with a national set of meteorology files, U.S. census data, and a risk calculation component that can be used to estimate individual and population risks.

Hydrolysis - The decomposition of organic compounds by interaction with water.

I

Impervious Surface - A surface that cannot be penetrated by water (e.g., pavement).

Indirect Exposure Pathway - An indirect exposure pathway is one in which a receptor contacts a chemical in a medium that is different from the one to which the chemical was originally released (an example occurs with dioxin, which is emitted into the air, deposited on soil and accumulated in plants and animals which are then consumed by humans).

Individual Risk or Hazard - The risk or hazard to an individual in a population rather than to the population as a whole.

Indoor Source - Objects or places within buildings or other enclosed spaces that emit air pollutants.

Industrial Source Complex (ISC) Model - A steady-state Gaussian plume model which can be used to assess pollutant concentrations from a wide variety of sources associated with an industrial complex. This model can account for the following: settling and dry deposition of particles; downwash; point, area, line, and volume sources; plume rise as a function of downwind distance; separation of point sources; and limited terrain adjustment. ISC3 operates in both long-term (ISCLT) and short-term (ISCST) modes.

Influential Information - Scientific, financial, or statistical information that will have or does have a clear and substantial impact on important public policies or important private sector decisions.

Ingestion - Swallowing (such as eating or drinking).

Ingestion Exposure - Exposure to a chemical by swallowing it (such as eating or drinking).

Inhalation - Breathing.

Inhalation Exposure - Exposure to a chemical by breathing it in.

Inhalation Unit Risk (IUR) - The upper-bound excess lifetime cancer risk estimated to result from continuous exposure to an agent at a concentration of 1 $\mu g/m^3$ in air. The interpretation of unit risk would be as follows: if unit risk = 2×10^{-6} $\mu g/m^3$, 2 excess tumors may develop per 1,000,000 people if exposed daily for a lifetime to a concentration of 1 µg of the chemical in 1 m^3 of air.

Intake - The process by which a substance crosses the outer boundary of an organism without passing an absorption barrier, e.g., through ingestion or inhalation.

Intake Rate - Rate of inhalation, ingestion, and dermal contact depending on the route of exposure.

Integrated Risk Information System (IRIS) - An EPA database which contains information on human health effects that may result from exposure to various chemicals in the environment. IRIS was initially developed for EPA staff in response to a growing demand for consistent information on chemical substances for use in risk assessments, decision-making and regulatory activities. The information in IRIS is intended for those without extensive training in toxicology, but with some knowledge of health sciences.

Internal Dose - In exposure assessment, the amount of a substance penetrating the absorption barriers (e.g., skin, lung tissue, gastrointestinal tract) of an organism through either physical or biological processes.

Inversion - Subsidence Inversion - A temperature inversion that develops aloft as a result of air gradually sinking over a wide area and being warmed by adiabatic compression, usually associated with subtropical high pressure areas.

Inversion - Advection Inversion - Associated with the horizontal flow of warm air. Warm air moves over a cold surface, and the air nearest the surface cools, causing a surface-based inversion.

Inversion - Radiation Inversion - A thermally produced, surface-based inversion formed by rapid radiational cooling of the Earth's surface at night. It does not usually extend above the lower few hundred feet. Conditions which are favorable for this type of inversion are long nights, clear skies, dry air, little or no wind, and a cold or snow covered surface. It is also called a Nocturnal Inversion.

Iterative Process - Replication of a series of actions to produce successively better results, or to accommodate new and different critical information or scientific inferences.

Isopleths - A delineated line or area on a map that represent equal values of a variable.

L

Laboratory Studies - Research carried out in a laboratory (e.g., testing chemical substances, growing tissues in cultures, or performing microbiological, biochemical, hematological, microscopical, immunological, parasitological tests).

Leaching - The process by which soluble constituents are dissolved and filtered through the soil by a percolating fluid (usually rainwater).

Life Stage - A phase in the life cycle of an organism.

Line Source - A theoretical one-dimensional source from which releases may occur (e.g., roadways are often modeled as a one-dimensional line).

Lofting - In pollution studies, a pattern of flow that occurs when the top of a plume from a chimney stack disperses into slightly turbulent or neutral airflow conditions, while the lower part of the plume is prevented from dispersing down toward the surface by a stable boundary layer, especially at night. [Smith, J. [ed], 2001: The Facts on File Dictionary of Weather and Climate.]

Low-dose Extrapolation - An estimation of the dose-response relationship at doses less than the lowest dose studied experimentally.

Lowest Observed Adverse Effect Level (LOAEL) - The lowest exposure level in a study or group of studies at which there are statistically or biologically significant increases in frequency or severity of adverse effects between the exposed population and its appropriate control group. Also referred to as lowest-effect level (LEL).

M

Major Source - Under the Clean Air Act, a stationary source that emits more than 10 tons or more per year of a single hazardous air pollutant (HAP) or 25 or more tons per year of all HAPs.

Margin of exposure (MOE) - The point of departure divided by the actual or projected environmental exposure of interest.

Mass-Balance Estimate - An estimate of release of a chemical based on, generally, a comparison of the amount of chemical in raw materials entering a process versus the amount of chemical going out in products.

Maximum Achievable Control Technology (MACT) - Under the Clean Air Act, a group of technology based standards, applicable to both major and some area sources of air toxics, that are aimed at reducing releases of air toxics to the environment. MACT standards are established on a source category by source category basis.

Maximum Exposed Individual (MEI) - The MEI represents the highest estimated risk to an exposed individual, regardless of whether people are expected to occupy that area.

Maximum Individual Risk (MIR) - An MIR represents the highest estimated risk to an exposed individual in areas that people are believed to occupy.

Metric (or Measure) of Exposure - The quantitative outcome of the exposure assessment. For air toxics risk assessments, personal air concentration (or adjusted exposure concentration) is the metric of exposure for the inhalation route of exposure and intake rate is the metric of exposure for the ingestion route of exposure.

Measurement - In air toxics assessment, a physical assessment (usually of the concentration of a pollutant) taken in an environmental or biological medium, normally with the intent of relating the measured value to the exposure of an organism.

Measurement Endpoint - A measurable ecological characteristic that is related to the valued characteristic chosen as the assessment endpoint. Also known as "measure of effect."

Mechanical Turbulence - Random irregularities of fluid motion in air caused by buildings or other nonthermal, processes.

Mechanistic Model - A model that uses information about a chemical or other agent's mechanism(s) of action – how it interacts with and harms the target organs – to predict the dose-response curve or other applications.

Media Concentrations - The amount of a given substance in a specific amount of environmental medium. For air, the concentration is usually given as micrograms (µg) of substance per cubic meter (m^3) of air; in water as µg of substance per L of water; and in soil as mg of substance per kg of soil.

Metabolism - Generally, the biochemical reactions by which energy is made available for the use of an organism. Metabolism includes all chemical transformations occurring in an organism from the time a substance enters, until it has been utilized and the waste products eliminated. In toxicology, metabolism of a toxicant consists of a series of chemical transformations that take place within an organism. A wide range of enzymes act on toxicants, that may increase water solubility, and facilitate elimination from the organism. In some cases, however, metabolites may be more toxic than their parent compound.

Meteorology - The science of the atmosphere, including weather.

Microcosm Studies - Studies of the effects of stressors on multiple species found in multiple media which are conducted in enclosed experimental systems.

Microscale Assessment - An air monitoring network designed to assess concentrations in air volumes associated with area dimensions ranging from several meters up to about 100 meters.

Microenvironment - A small 3-dimensional space (e.g., an office, a room in a home) that can be treated as homogeneous (or well characterized) with regard to exposure concentration of a chemical.

Middle Scale Assessment - An air monitoring network designed to assess concentrations typical of areas up to several city blocks in size with dimensions ranging from about 100 meters to 0.5 kilometer.

Minimal Risk Levels (MRL) - Derived by ATSDR, an MRL is defined as an estimate of daily human exposure to a substance that is likely to be without an appreciable risk of adverse effects (noncancer) over a specified duration of exposure. MRLs can be derived for acute, intermediate, and chronic duration exposures by the inhalation and oral routes.

Mixed (Mixing) Layer - In the atmosphere, that part of the turbulent boundary layer that is dominated by turbulent diffusion caused by eddies generated by friction with the surface and thermals arising from surface heat sources. Surface heating during the day and the absence of temperature inversions allow components of the air within the planetary boundary layer to exhibit mainly random vertical movements. Such movements may become more organized into gusts of wind and dust devils during the afternoon. Despite being random, the turbulent movements allow the transfer of atmospheric properties, such as heat, water vapor, momentum, and air pollutants, from the near surface up through the planetary boundary layer.

Mixing Height - The depth through which atmospheric pollutants are typically mixed by dispersive processes.

Mixtures - Any set of multiple chemical substances occurring together in an environmental medium.

Mobile Source Air Toxics - Air toxics that are emitted from non-stationary objects that release pollution. Mobile sources include cars, trucks, buses, planes, trains, motorcycles and gasoline-powered lawn mowers. Another example is a portable generator.

Model - A mathematical representation of a natural system intended to mimic the behavior of the real system, allowing description of empirical data, and predictions about untested states of the system.

Model Uncertainty - Uncertainty due to necessary simplification of real-world processes, mis-specification of the model structure, model misuse, or use of inappropriate surrogate variables or inputs.

Modeling - An investigative technique using a mathematical or physical representation of a system or theory that accounts for all or some of its known properties.

Modeling Node - In air quality modeling, the location where impacts are predicted.

Monitoring - Periodic or continuous physical surveillance or testing to determine pollutant levels in various environmental media or in humans, plants, and animals.

Monte Carlo Technique- A repeated random sampling from the distribution of values for each of the parameters in a generic exposure or risk equation to derive an estimate of the distribution of exposures or risks in the population.

Multipathway Assessment - An assessment that considers more than one exposure pathway. For example, evaluation of exposure through both inhalation and ingestion would be a multipathway assessment. Another example would be evaluation of ingestion of contaminated soil and ingestion of contaminated food.

Multipathway Exposure - When an organism is exposed to pollutants through more than one exposure pathway. One example would be exposure through both inhalation and ingestion. Another example would be ingestion of contaminated soil and ingestion of contaminated food.

Multipathway Risk - The risk resulting from exposure to pollutants through more than one pathway.

Multistage Model - A mathematical function used to extrapolate the probability of cancer from animal bioassay data, using the form:

$$P(d) = 1 - e^{-\left(q_0 + q_1 d + q_2 d^2 + \ldots + q_k d^k\right)}$$

where:

P(d)	=	probability of cancer from a continuous, lifetime exposure rate d;
q_i	=	fitted dose coefficients of model; i = 0, 1, . . ., k; and
k	=	number of stages selected through best fit of the model, no greater than one less than the number of available dose groups.

Mutagen - A chemical that causes a permanent genetic change in a cell other than that which occurs during normal growth.

Mutagenicity - The capacity of a chemical or physical agent to cause permanent genetic change in a cell other than that which occurs during normal growth.

N

National Ambient Air Quality Standards (NAAQS) - Maximum air pollutant standards that EPA has set under the Clean Air Act for attainment by each state. Standards are set for each of the criteria pollutants.

National Air Toxics Assessment (NATA) - EPA's ongoing comprehensive evaluation of air toxics in the U.S. Activities include expansion of air toxics monitoring, improving and periodically updating emission inventories, improving national- and local-scale modeling and risk characterization, continued research on health effects and exposures to both ambient and indoor air, and improvement of assessment tools.

National Emissions Inventory (NEI) - EPA's primary emissions inventory of HAPs.

National Emissions Standards for Hazardous Air Pollutants (NESHAPs) - Emissions standards set by EPA for hazardous air pollutants. Also commonly referred to as the MACT standards.

National Emissions Trends (NET) Database - The NET database is an emission inventory that contains data on stationary and mobile sources that emit criteria air pollutants and their precursors. The database also includes estimates of annual emissions of these pollutants from point, area, and mobile sources. The NET is developed every three years (e.g., 1996 and 1999) by EPA, and includes emission estimates for all 50 States, the District of Columbia, Puerto Rico, and the Virgin Islands.

Natural Source - Non-manmade emission sources, including biological (biogenic sources such as plants) and geological sources (such as volcanoes), and windblown dust.

Neighborhood Scale Assessment - An air monitoring network designed to assess concentrations within some extended area of the city that has relatively uniform land use with dimensions in the 0.5 to 4.0 kilometers range.

Neurotoxicity - Ability to damage nervous system tissue or adversely effect nervous system function.

New Source Review - A Clean Air Act requirement that State Implementation Plans must include a permit review that applies to the construction and operation of new and modified stationary sources in nonattainment areas to ensure attainment of national ambient air quality standards.

New Source Performance Standards - Uniform national EPA air emission standards which limit the amount of pollution allowed from new sources or from modified existing sources.

Noncarcinogenic Effect - Any health effect other than cancer. Note that, while not all noncancer toxicants cause cancer, all carcinogens exhibit noncarcinogenic effects.

No Observable Adverse Effect Level (NOAEL) - An highest exposure level at which there are no statistically or biologically significant increases in the frequency or severity of adverse effect between the exposed population and its appropriate control; some effects may be produced at this level, but they are not considered adverse, nor precursors to adverse effects.

Nonpoint Source (NEI sense) - Diffuse pollution sources that are not assigned a single point of origin (e.g., multiple dry cleaners in a county which are only described in an inventory in the aggregate).

Nonroad Mobile Sources - Sources such as farm and construction equipment, gasoline-powered lawn and garden equipment, and power boats and outdoor motors that emit pollutants.

Non-Threshold Effect - An effect (usually an adverse health effect) for which there is no exposure level below which the effect is not expected to occur.

Non-Threshold Toxicant - A chemical for which there is no exposure level below which an adverse health outcome is not expected to occur. Such substances are considered to pose some risk of harm at any level of exposure.

Non Steady-state Model - A dynamic model; a mathematical formulation describing and simulating the physical behavior of a system or a process and its temporal variability.

North American Industry Classification System (NAICS) - NAICS replaced the Standard Industrial Classification (SIC) beginning in 1997. This industry-wide classification system has been designed as the index for statistical reporting of all economic activities of the U.S., Canada, and Mexico. NAICS industries are identified by a 6-digit code. The international NAICS agreement fixes only the first five digits of the code. The sixth digit, where used, identifies subdivisions of NAICS industries that accommodate user needs in individual countries.

O

Octanol/Water Partition Coefficient (K_{ow}) - The ratio of a chemical's solubility in n-octanol to its solubility in water at equilibrium. This measure is often used as an indication of a chemical's ability to bioconcentrate in organisms.

Office of Air and Radiation (OAR) - EPA's Office responsible for providing information about air pollution, clean air, air quality and radiation. OAR develops national programs, technical policies, and regulations for controlling air pollution and radiation exposure. OAR is concerned with pollution prevention, indoor and outdoor air quality, industrial air pollution, pollution from vehicles and engines, radon, acid rain, stratospheric ozone depletion, and radiation protection.

Office of Air Quality, Planning, and Standards (OAQPS) - An EPA Office within OAR whose primary mission is to preserve and improve air quality in the United States. As part of this goal, OAQPS monitors and reports on air quality, air toxics, and emissions. They also respond to visibility issues, as they relate to the level of air pollution. In addition, OAQPS is tasked by the EPA with providing technical information for professionals involved with monitoring and controlling air pollution, creating governmental policies, rules, and guidance (especially for stationary sources), and educating the public about air pollution and what can be done to control and prevent it.

OAQPS Toxicity Table - The EPA Office of Air and Radiation recommended default chronic toxicity values for hazardous air pollutants. They are generally appropriate for screening-level risk assessments, including assessments of select contaminants, exposure routes, or emission sources of potential concern, or to help set priorities for further research. For more complex, refined risk assessments developed to support regulatory decisions for single sources or substances, dose-response data may be evaluated in detail for each "risk driver" to incorporate appropriate new toxicological data. (http://www.epa.gov/ttn/atw/toxsource/summary.html)

Office of Radiation and Indoor Air (ORIA) - An EPA Office within OAR whose mission is to protect the public and the environment from the risks of radiation and indoor air pollution. The Office develops protection criteria, standards, and policies; works with other programs within EPA and other agencies to control radiation and indoor air pollution exposures; provides technical assistance to states through EPA's regional offices, and to other agencies having radiation and indoor air protection programs; directs an environmental radiation monitoring program; responds to radiological emergencies; and evaluates and assesses the overall risk and impact of radiation and indoor air pollution.

Office of Transportation and Air Quality (OTAQ) - An EPA Office within OAR whose mission is to reconcile the transportation sector with the environment by advancing clean fuels and technology, and working to promote more liveable communities. OTAQ is responsible for carrying out laws to control air pollution from motor vehicles, engines, and their fuels. Mobile sources include: cars and light trucks, large trucks and buses, farm and construction equipment, lawn and garden equipment, marine engines, aircraft, and locomotives.

Onroad Mobile Source - Any mobile source of air pollution such as cars, trucks, motorcycles, and buses that travels on roads and highways.

Open Pit Source - Large, open pits, such as surface coal mines and rock quarries.

Operating Permit Program - A program required by the Clean Air Act; requires existing industrial sources to obtain an"operating permit". The operating permit program is a national permitting system that consolidates all of the air pollution control requirements into a single, comprehensive "operating permit" that covers all aspects of a source's year-to-year air pollution activities.

P

Particle-bound - Reversibly absorbed or condensed onto the surface of particles.

Particulates/Particulate Matter (PM) - Solid particles or liquid droplets suspended or carried in the air.

Partitioning - The separation or division of a substance into two or more compartments. Environmental partitioning refers to the distribution of a chemical into various media (soil, air, water, and biota).

Partitioning Model - Models consisting of mathematical equations that estimate how chemicals will divide (i.e., partition) among abiotic and biotic media in a given environment based on chemical- and site- specific characteristics.

Passive Monitor - A type of air toxics monitor that collects airborne pollutants by absorption onto a reactive material (for example, sorbent tube, filter) for subsequent laboratory analysis. No pump is used to draw the air across the reactive material. This type of monitor is usually used for personal exposure monitoring or work space monitoring.

Pathway Specific Risk - The risk associated with exposure to a chemical agent or a mixture of chemicals via a specific pathway (e.g., inhalation of outdoor air).

Persistent, Bioaccumulative, and Toxic (PBT) Chemicals - Highly toxic, long-lasting substances that can build up in the food chain to levels that are harmful to human and ecosystem health. They are associated with a range of adverse health effects, including effects on the nervous system, reproductive and developmental problems, cancer, and genetic impacts.

Percentile - Any one of the points dividing a distribution of values into parts each of which contain 1/100 of the values. For example, the 75th percentile is a value such that 75 percent of the values are less than or equal to it.

Persistence - Refers to the length of time a compound stays in the environment, once introduced. A compound may persist for very short amounts of time (e.g., fractions of a second) or for long periods of time (e.g., hundreds of years).

Persistent Organic Pollutants (POPs) - Highly stable organic compounds used as pesticides or in industry. They are also generated unintentionally as the byproduct of combustion and industrial processes. POPs are a special problem because they persist in the environment, accumulate in the tissues of living organisms, and are toxic to humans and wildlife. POPs with these characteristics are typically semi-volatile, enabling them to move long distances and condense over colder regions of the earth. These properties lead to increased concern for the toxic effects that they can exert on a range of biota, in particular on top-of-the-food chain species, even at extremely low levels in the ambient environment.

Personal Air Monitoring Device - Unlike a passive air toxics monitor, this device uses a pump to draw the air sample through to measure exposure in the immediate vicinity of an individual. The air sample can be drawn across a reactive material (to be analayzed in a laboratory), or it can be drawn through a direct-read detector.

Personal Monitoring - A measurement collected from an individual's immediate environment using active or passive devices to collect the samples.

Pervious Surface - A surface that can be penetrated (usually in reference to water; e.g., crop land).

Pharmacodynamics - Process of interaction of pharmacologically active substances with target sites, and the biochemical and physiological consequences leading to therapeutic or adverse effects.

Pharmacokinetics - The study of the absorption, distribution, metabolism, and excretion of chemicals in living organisms and the genetic, nutritional, behavioral, and environmental factors that modify these parameters.

Photolysis - The breakdown of a material by sunlight; an important mechanism for the degradation of contaminants in air, surface water, and the terrestrial environment.

Physical Factors - Manmade and/or natural characteristics or features that influence the movement of pollutants in the environment (e.g., settling velocity, terrain effects).

Physiologically Based Pharmacokinetic (PBPK) Model - A computer model that describes what happens to a chemical in the body of a human or laboratory animal. It describes how the chemical gets into the body, where it goes in the body, how it is changed by the body, and how it leaves the body.

Piscivorous - A species feeding preferably on fish.

Planning and Scoping - The process of determining the purpose, scope, players, expected outcomes, analytical approach, schedule, deliverables, QA/QC, resources, and document requirements for the risk assessment.

Plume - The visible or measurable presence of a contaminant in the atmosphere, once released from a given point of origin (e.g., a plume of smoke from a forest fire).

Plume Height - The elevation to which a plume travels (i.e., the sum of the release height and plume rise).

Plume Rise - The height to which a plume rises in the atmosphere from the point of release.

Plume Transport - The movement of a plume through the atmosphere and across land and water features.

Plume Washout - The removal of a substance from the atmosphere via a precipitation event.

PM-10/PM-2.5. PM-10 or PM_{10} refers to particles in the atmosphere with a diameter of less than ten or equal to 10 micrometers. PM-2.5 or $PM_{2.5}$ refers to smaller particles in the air (i.e., less than or equal to 2.5 micrometers in diameter).

Point of Departure (PoD) - The dose-response point that marks the beginning of a low-dose extrapolation. This point can be the lower bound on dose for an estimated incidence or a change in response level from a dose-response model (BMD), or a NOAEL or LOAEL for an observed incidence, or change in level of response.

Point of Exposure - The location of potential contact between an organism and a chemical or physical agent.

Point of Release - Location of release to the environment.

Point Source (NEI sense) - A source of air pollution which can be physically located on a map.

Point Source (non-NEI sense) - A stack, vent, duct, pipe or other confined air stream from which chemicals may be released to the air.

Pollutant Release and Transfer Registries (PRTRs) - The international equivalent to the Toxics Release Inventory (TRI). PRTRs are data banks of recorded information of the releases

and transfers of toxic chemicals from industries, such as manufacturers, mining facilities, processors, or government-owned and operated facilities.

Population Risk or Hazard - Population risk refers to an estimate of the extent of harm for the population or population segment being addressed. It often refers to an analysis of the number of people living at a particular risk or hazard level.

Potential Risk - Estimated likelihood, or probability, of injury, disease, or death resulting from exposure to a potential environmental hazard.

Potential Dose - The amount of a compound contained in material swallowed, breathed, or applied to the skin.

Practical Quantitation Limit - The lowest level of quantitation that can be reliably achieved within specified limits of precision and accuracy during routine laboratory operating conditions.

Precision - A measure of the reproducibility of a measured value under a given set of circumstances.

Present Scenario - Risk characterizations using present scenarios to estimate risks to individuals (or populations) that currently reside in areas where potential exposures may occur (e.g., using an existing population within some specified area).

Prevailing Wind - Direction from which the wind blows most frequently.

Prevention of Significant Deterioration (PSD) - An EPA program in which state and/or federal permits are required in order to restrict emissions from new or modified sources in places where air quality already meets or exceeds primary and secondary ambient air quality standards.

Primary Standard - A pollution limit based on health effects. Primary standards are set for criteria air pollutants.

Probabilistic - A type of statistical modeling approach used to assess the expected frequency and magnitude of a parameter by running repetitive simulations using statistically selected inputs for the determinants of that parameter (e.g., rainfall, pollutants, flows, temperature).

Probabilistic Risk Assessment/Analysis - Calculation and expression of health risks using multiple risk descriptors to provide the likelihood of various risk levels. Probabilistic risk results approximate a full range of possible outcomes and the likelihood of each, which often is presented as a frequency distribution graph, thus allowing uncertainty or variability to be expressed quantitatively.

Probability Density Function (PDF) - The PDF is alternatively referred to in the literature as the probability function or the frequency function. For continuous random variables, that is, the random variables which can assume any value within some defined range (either finite or infinite), the probability density function expresses the probability that the random variable falls within some very small interval. For discrete random variables, that is, random variables which can only assume certain isolated or fixed values, the term probability mass function (PMF) is preferred over the term probability density function. PMF expresses the probability that the random variable takes on a specific value.

Problem Formulation (in Ecological Risk Assessment) - The initial stage of a risk assessment where the purpose of the assessment is articulated, assessment endpoints and a conceptual model are developed, and a plan for analyzing and characterizing risk is determined.

Problem Statement - A statement of the perceived problem to be studied by the risk assessment. Problem statements often also include statements about how the problem is going to be studied.

Public Health Consultation (Public Health Assessment) - See health consultation.

Public Health Assessment (PHA) - An evaluation of hazardous substances, health outcomes, and community concerns at a hazardous waste site or other potential source of pollutants to determine whether people could be harmed from coming into contact with those substances. The PHA also lists actions that need to be taken to protect public health.

Public Health Advisory (in Public Health Assessment) - A statement made by a regulatory agency that a release of hazardous substances or contamination by microbial pathogens poses an immediate threat to human health. The advisory includes recommended measures to reduce exposure and reduce the threat to human health.

Public Health Hazard Category (in Public Health Assessment) - Statements about whether people could be harmed by conditions present at the site in the past, present, or future. One or more hazard categories might be appropriate for each site. ATSDR's five public health hazard categories are no public health hazard, no apparent public health hazard, indeterminate public health hazard, public health hazard, and urgent public health hazard.

Q

Qualitative Uncertainty Estimate - A detailed examination, using qualitative information, of the systematic and random errors of a measurement or estimate.

Quality Assurance Project Plan - A document describing in comprehensive detail the necessary quality assurance, quality control, and other technical activities that must be implemented to ensure that the results of the work performed will satisfy the stated performance criteria.

Quality Assurance - An integrated system of activities involving planning, quality control, quality assessment, reporting and quality improvement to ensure that a product or service meets defined standards of quality with a stated level of confidence.

Quality Control - The overall system of technical activities whose purpose is to measure and control the quality of a product or service so that it meets the needs of its users. The aim is to provide data quality that is satisfactory, adequate, and dependable.

R

Random Variable - A quantity which can take on any number of values but whose exact value cannot be known before a direct observation is made. For example, the outcome of the toss of a pair of dice is a random variable, as is the height or weight of a person selected at random from a city phone book.

Receptor (modeling sense) - In fate/transport modeling, the location where impacts are predicted.

Receptor (non-modeling sense) - The entity which is exposed to an environmental stressor.

Red Book - 1983 NRC publication entitled *Risk Assessment in the Federal Government: Managing the Process*.

Reference Concentration (RfC) - An estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime.

Reference Dose (RfD) - An estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime.

Reference Media Evaluation Guides (RMEG) - A type of comparison value derived by ATSDR to protect the most sensitive populations. They do not consider carcinogenic effects, chemical interactions, multiple route exposure, or other media-specific routes of exposure, and are very conservative concentration values designed to protect sensitive members of the population.

Regional/National Scale Assessment - An air monitoring network designed to assess from tens to hundreds of kilometers, up to the entire nation.

Relative Potency Factor - The ratio of the toxic potency of a given chemical to that of an index chemical.

Release Parameters - The specific physical characteristics of the release (e.g., stack diameter, stack height, release flow rate, temperature).

Representativeness - The degree to which one or a few samples are characteristic of a larger population about which the analyst is attempting to make an inference.

Reproductive Toxicity - The occurrence of biologically adverse effects on the reproductive systems of females or males that may result from exposure to environmental agents. The toxicity may be expressed as alterations to the female or male reproductive organs, the related endocrine system, or pregnancy outcomes. The manifestation of such toxicity may include, but not be limited to, adverse effects on onset of puberty, gamete production and transport, reproductive cycle normality, sexual behavior, fertility, gestation, parturition, lactation, developmental toxicity, premature reproductive senescence, or modifications in other functions that are dependent on the integrity of the reproductive systems.

Residual Risk - The extent of health risk from air pollutants remaining after application of the Maximum Achievable Control Technology (MACT).

Resources - Money, time, equipment, and personnel available to perform the assessment.

Risk (in the context of human health) - The probability of injury, disease, or death from exposure to a chemical agent or a mixture of chemicals. In quantitative terms, risk is expressed in values ranging from zero (representing the certainty that harm will not occur) to one (representing the certainty that harm will occur). (Compare with hazard.)

Risk Assessor(s) - The person or group of people responsible for conducting a qualitative and quantitative evaluation of the risk posed to human health and/or the environment by environmental pollutants.

Risk Assessment - For air toxics, the scientific activity of evaluating the toxic properties of a chemical and the conditions of human or ecological exposure to it in order both to ascertain the likelihood that exposed humans or ecological receptors will be adversely affected, and to characterize the nature of the effects they may experience.

Risk Assessment Forum - A standing committee of senior EPA scientists which was established to promote Agency-wide consensus on difficult and controversial risk assessment issues and to ensure that this consensus is incorporated into appropriate Agency risk assessment guidance.

Risk Assessment Work Plan - A document that outlines the specific methods to be used to assess risk, and the protocol for presenting risk results. The risk assessment workplan may consist of one document or the compilation of several workplans that, together, constitute the overall risk assessment workplan.

Risk Characterization - The last phase of the risk assessment process in which the information from the toxicity and exposure assessment steps are integrated and an overall conclusion about risk is synthesized that is complete, informative and useful for decision-makers. In all cases, major issues and uncertainty and variability associated with determining the nature and extent of the risk should be identified and discussed. The risk characterization should be prepared in a manner that is clear, transparent, reasonable and consistent.

Risk Communication - The exchange of information about health or environmental risks among risk assessors and managers, the general public, news media, and other stakeholders.

Risk Management - The decision-making process that uses the results of risk assessment to produce a decision about environmental action. Risk management includes consideration of technical, scientific, social, economic, and political information.

Risk Manager(s) - The person or group responsible for evaluating and selecting alternative regulatory and non-regulatory responses to risk.

Root Uptake - The uptake of compounds available in the soil and their transfer to the above ground portions of the plant.

Route-to-Route Extrapolation - Calculations to estimate the dose-response relationship of an exposure route for which experimental data do not exist or are inadequate, and which are based on existing experimental data for other route(s) of exposure.

Runoff - That part of precipitation, snow melt, or irrigation water that runs off the land into streams or other surface water. It can carry pollutants from the air and land into receiving waters.

S

Sample - A small portion of something designed to evaluate the nature or quality of the whole (for example, one or several samples of air used to evaluate air quality generally).

Sampling and Analysis Plan - An established set of procedures specifying how a sample is to be collected, handled, analyzed, and the data validated and reported.

Sampling Frequency - The time interval between the collection of successive samples.

Science Advisory Board (SAB) - A group of recognized, non-EPA experts who advise EPA on science and science policy.

Scenario Uncertainty - Uncertainty due to descriptive errors, aggregation errors, errors in professional judgment, or incomplete analysis.

SCREEN3 - An air dispersion model developed to obtain conservative estimates of air concentration for use in screening level assessments through the use of conservative algorithms and meteorology.

Screening-level Risk Assessment - A risk assessment performed with few data and many conservative assumptions to identify exposures that should be evaluated more carefully for potential risk.

Secondary Production/Pollutant - Formation of pollutants in the atmosphere by chemical transformation of precursor compounds.

Secondary Standard - A pollution limit based on environmental effects (e.g., damage to property, plants, visibility). Secondary standards are set for criteria air pollutants.

Sensitive Subgroups - Identifiable subsets of the general population that, due to differential exposure or susceptibility, are at greater risk than the general population to the toxic effects of a specific air pollutant (e.g., depending on the pollutant and the exposure circumstances, these may be groups such as subsistence fishers, infants, asthmatics, or the elderly).

Settling Velocity/Rate - The maximum speed at which a particle will fall in still air. It is a function of its size, density, and shape.

Silage - Stored vegetation used as feed for cattle.

Simulation - A representation of a problem, situation in mathematical terms, especially using a computer.

Soil Volumetric Water Content - The soil-water content expressed as the volume of water per unit bulk volume of soil.

Soil Dry Bulk Density - The mass of dry soil per unit bulk volume.

Soil Erosion - Detachment and movement of topsoil or soil material from the upper part of the soil profile, by the action of wind or running water, especially as a result of changes brought about by human activity, such as unsuitable or mismanaged agriculture.

Solar Radiation - Energy from the sun. Of importance to the climate system, solar radiation includes ultra-violet radiation, visible radiation, and infrared radiation.

Solubility - The amount of mass of a compound that will dissolve in a unit volume of solution. Aqueous solubility is the maximum concentration of a chemical that will dissolve in pure water at a reference temperature.

Source - Any place or object from which pollutants are released.

Source Category - A group of similar industrial processes or industries that are contributors to releases of hazardous air pollutants. The 1990 amendments to the Clean Air Act (CAA) requires that the EPA publish and regularly update a listing of all categories and subcategories of major and area sources that emit hazardous air pollutants.

Source Characterization - The detailed description of the source (e.g., location, source of pollutant releases, pollutants released, release parameters).

Spatial Variability - The magnitude of difference in contaminant concentrations in samples separated by a known distance.

SPECIATE - EPA's repository of Total Organic Compound (TOC) and Particulate Matter (PM) speciated profiles for a variety of sources for use in source apportionment studies. The profiles in the system are provided as a library of available profiles for source-receptor and source apportionment type models, such as Chemical Mass Balance 8 (CMB8).

Stable Conditions (in the Atmosphere) - Air with little or no tendency to rise, that is usually accompanied by clear dry weather. Stable air holds, instead of dispersing, pollutants. [National Weather Service, Southern Region Headquarters' Jetstream Weather School, http://www.srh.weather.gov/jetstream/append/glossary.htm and EPA, 1997: Terms of Environment, http://www.epa.gov/OCEPAterms/.]

Stack - A chimney, smokestack, or vertical pipe that discharges used air.

Stack Release - The release of a chemical through a stack.

Stack Testing - The monitoring, by testing, of chemicals released from a stack.

Stakeholder(s) - Any organization, governmental entity, or individual that has a stake in or may be impacted by a given approach to environmental regulation, pollution prevention, energy conservation, etc.

Standard Industrial Classification (SIC) - A method of grouping industries with similar products or services and assigning codes to these groups.

Standard Operating Procedure (SOP) - A established set of written procedures adopted and used to guide the work of for a specific project. For example, an air monitoring study would include SOPs on sample collection and handling and SOPs on analytical requirements and data validation and reporting.

Standing Crop - The quantity of plant biomass in a given area, usually expressed as density (dry mass per unit area) or energy content per unit area.

Stationary Source - A source of pollution that is fixed in space.

Steady-state Model - Mathematical model of fate and transport that uses constant values of input variables to predict constant values of receiving media concentrations.

Stochastic - Involving or containing a random variable; involving probability or chance.

Stressor - Any physical, chemical, or biological entity that can induce adverse effects on ecosystems or human health.

Stressor-response Profile or Relationship (in Ecological Risk Assessment] - The product of characterization of ecological effects in the analysis phase of ecological risk assessment. The stressor-response profile/relationship summarizes the data on the effects of a stressor and the relationship of the data to the assessment endpoint.

Structure-activity Relationship (SAR) - Mathematical or qualitative expression of the relationships between biological activity or toxicity of a chemical to its chemical structure or substructure. Ideally, such relationships can be formulated as Quantitative Structure Activity Relationships (QSARs), in which some degree of predictive capability is present. [Air Risk Information Support Center, OAQPS, March 1989: Glossary of Terms Related to Health, Exposure, and Risk Assessment. EPA/450/3-88/016.]

Support Center for Regulatory Models (SCRAM) - An EPA website that is a source of information on atmospheric dispersion models (e.g., ISCST3, SCREEN 3, and ASPEN) that support regulatory programs required by the Clean Air Act. Documentation and guidance for these computerized models are a major feature of this website. This site also contains computer code, data, and technical documents that deal with mathematical modeling for the dispersion of air pollutants.

Synergistic Effect - A situation in which the overall effect of two chemicals acting together is greater than the simple sum of their individual effects.

T

Target Organ - The biological organ(s) most adversely affected by exposure to a chemical substance (e.g., the site of the critical effect).

Target Organ Specific Hazard Index (TOSHI) - The sum of hazard quotients for individual air toxics that affect the same organ/organ system or act by similar toxicologic processes

Temporal Variability - The difference in contaminant concentrations observed in samples taken at different times.

Teratogenesis - The introduction of nonhereditary birth defects in a developing fetus by exogenous factors such as physical or chemical agents acting in the womb to interfere with normal embryonic development.

Terrain Effects - The impact on the airflow as it passes over complex land features such as mountains.

Terrestrial Radiation - The total infrared radiation emitted by the earth and its atmosphere in the temperature range of approximately 200 to 300 Kelvin. Terrestrial radiation provides a major part of the potential energy changes necessary to drive the atmospheric wind system and is responsible for maintaining the surface air temperature within limits of livability.

Thermal Turbulence - Turbulent vertical motions that result from surface heating and the subsequent rising and sinking of air.

Threshold Dose/Threshold - The lowest dose of a chemical at which a specified measurable effect is observed and below which it is not observed.

Threshold Effect - An effect (usually an adverse health effect) for which there is an exposure level below which the effect is not expected to occur.

Threshold Toxicant - A chemical for which there is an exposure level below which an adverse health outcome is not expected to occur.

Tiered Analysis - An analysis arranged in layers/steps. Risk assessments/analyses are often conducted in consecutive layers/steps that begin with a reliance on conservative assumptions and little data (resulting in less certain, but generally conservative answers) and move to more study-area specific data and less reliance on assumptions (resulting in more realistic answers). The level of effort and resources also increases with the development of more realistic data.

Time-integrated Sample - Samples are collected over a period of time. Only the total pollutant collected is measured, and so only the average concentration during the sampling period can be determined.

Time-trend Study - Samples spaced in time to capture systematic temporal trends (e.g., a facility might change its production methods or products over time).

Time-weighted Sum of Exposures - Used in inhalation exposure modeling. Provides a total exposure from all different microenvironments in which a person spends time.

Toxic Air Pollutants - see hazardous air pollutant.

Toxicity - The degree to which a substance or mixture of substances can harm humans or environmental receptors.

Toxicity Assessment - Characterization of the toxicological properties and effects of a chemical, with special emphasis on establishment of dose-response characteristics.

Toxicity Test - Biological testing (usually with an cell system, invertebrate, fish, or small mammal) to determine the adverse effects of a compound.

Toxicology - The study of harmful interactions between chemicals and biological systems.

Toxic Release Inventory (TRI) - Annual database of releases to air, land, and water, and information on waste management in the United States of over 650 chemicals and chemical compounds. This data is collected under Section 313 of the Emergency Planning and Community Right to Know Act.

Trajectory - The track taken by a parcel of air as it moves within the atmosphere over a given period.

Transformation - The change of a chemical from one form to another.

Transparency - Conducting a risk assessment in such a manner that all of the scientific analyses, uncertainties, assumptions, and science policies which underlie the decisions made throughout the risk assessment are clearly stated (i.e., made readily apparent).

Turbulence - Irregular motion of the atmosphere, as indicated by gusts and lulls in the wind.

U

Uncertainty - Uncertainty represents a lack of knowledge about factors affecting exposure/toxicity assessments and risk characterization and can lead to inaccurate or biased estimates of risk and hazard. Some of the types of uncertainty include scenario uncertainty, parameter uncertainty, and model uncertainty.

Uncertainty analysis - A detailed examination of the systematic and random errors of a measurement or estimate (in this case a risk or hazard estimate); an analytical process to provide information regarding the uncertainty.

Uncertainty Factor (UF) - One of several, generally 10-fold factors, used in operationally deriving the RfD and RfC from experimental data. UFs are intended to account for (1) the variation in sensitivity among the members of the human population; (2) the uncertainty in extrapolating animal data to humans, i.e., interspecies variability; (3) the uncertainty in extrapolating from data obtained in a study with less-than-lifetime exposure to lifetime exposure, i.e., extrapolating from subchronic to chronic exposure; (4) the uncertainty in extrapolating from a LOAEL rather than from a NOAEL; and (5) the uncertainty associated with extrapolation from animal data when the data base is incomplete.

Universal Soil Loss Equation - An equation used to predict the average annual soil loss per unit area per year.

Unit Risk Estimate (URE) - The upper-bound excess lifetime cancer risk estimated to result from continuous exposure to an agent at a concentration of 1µ g/L in water, or 1 $\mu g/m^3$ in air. The interpretation of unit risk would be as follows: if the water unit risk = 2×10^{-6} µg/L, 2 excess tumors may develop per 1,000,000 people if exposed daily for a lifetime to 1 µg of the chemical in 1 liter of drinking water.

Unstable Conditions (in the Atmosphere) - An atmospheric state in which warm air is below cold air. Since warm air naturally rises above cold air (due to warm air being less dense than cold air), vertical movement and mixing of air layers can occur.

Uptake - The process by which a substance crosses an absorption barrier and is absorbed into the body.

Urban Scale Assessment - An air monitoring network designed to assess the overall, citywide conditions with dimensions on the order of 4 to 50 kilometers. This scale would usually require more than one site for definition.

V

Vapor - The gas given off by substances that are solids or liquids at ordinary atmospheric pressure and temperatures.

Variability - Refers to the observed differences attributable to true heterogeneity or diversity in a population or exposure parameter. Examples include human physiological variation (e.g., natural variation in body weight, height, breathing rate, drinking water intake rate), weather variability, variation in soil types and differences in contaminant concentrations in the environment. Variability is usually not reducible by further measurement of study, but it can be better characterized.

Volatilization/Vapor Release - The conversion of a liquid or solid into vapors.

Volume Source - In air dispersion modeling, a three dimensional volume from which a release may occur (e.g., a gas station modeled as a box from which chemicals are emitted).

W

Watershed - The land area that drains into a stream; the watershed for a major river may encompass a number of smaller watersheds that ultimately combine at a common point.

Weight-of-Evidence (WOE) - A system for characterizing the extent to which the available data support the hypothesis that an agent causes an adverse health effect in humans. For example, under EPA's 1986 cancer risk assessment guidelines, the WOE was described by categories "A through E," Group A for known human carcinogens through Group E for agents with evidence of noncarcinogenicity. The approach outlined in EPA's proposed guidelines for carcinogen risk assessment (1996 and updates) considers all scientific information in determining whether and under what conditions an agent may cause cancer in humans, and provides a narrative approach to characterize carcinogenicity rather than categories.

White Book - 1996 Presidential Commission on Risk Assessment and Risk Management (CRARM) publication entitled *Risk Assessment and Risk Management in Regulatory Decision-Making*.

Wind Rose - A graphical display showing the frequency and strength of winds from different directions over some period of time.

Appendix A Listing of All HAPs

Appendix A. Listing of HAPs

CAS Number	Chemical Name	Common Name	CAA HAP	TRI Chemical	Urban HAP	Mobile Source Air Toxic
75-07-0	Acetaldehyde		X	X	X	X
60-35-5	Acetamide		X	X		
75-05-8	Acetonitrile		X	X		
98-86-2	Acetophenone		X	X		
53-96-3	2-Acetylaminofluorene		X	X		
107-02-8	Acrolein		X	X	X	X
79-06-1	Acrylamide		X	X		
79-10-7	Acrylic acid		X	X		
107-13-1	Acrylonitrile		X	X	X	
107-05-1	Allyl chloride		X	X		
92-67-1	4-Aminobiphenyl		X	X		
62-53-3	Aniline		X	X		
90-04-0	o-Anisidine		X	X		
1332-21-4	Asbestos		X	X		
71-43-2	Benzene (including benzene from gasoline)		X	X	X	X
92-87-5	Benzidine		X	X		
98-07-7	Benzotrichloride		X	X		
100-44-7	Benzylchloride		X	X		
92-52-4	Biphenyl		X	X		
117-81-7	Bis (2-ethylhexyl) phthalate	DEHP	X	X		
542-88-1	Bis(chloromethyl)ether		X	X		
75-25-2	Bromoform		X	X		
106-99-0	1,3-Butadiene		X	X	X	X
156-62-7	Calcium cyanamide		X	X		
133-06-2	Captan		X	X		
63-25-2	Carbaryl		X	X		
75-15-0	Carbon disulfide		X	X		
56-23-5	Carbon tetrachloride		X	X	X	
463-58-1	Carbonyl sulfide		X	X		
120-80-9	Catechol		X	X		
133-90-4	Chloramben		X	X		
57-74-9	Chlordane		X	X		
7782-50-5	Chlorine		X	X		
79-11-8	Chloroacetic acid		X	X		
532-27-4	2-Chloroacetophenone		X	X		
108-90-7	Chlorobenzene		X	X		
510-15-6	Chlorobenzilate		X	X		
67-66-3	Chloroform		X	X	X	
107-30-2	Chloromethyl methyl ether		X	X		
126-99-8	Chloroprene		X	X		
1319-77-3	Cresol/Cresylic acid (mixed isomers)		X	X		

Appendix A. Listing of HAPs

CAS Number	Chemical Name	Common Name	CAA HAP	TRI Chemical	Urban HAP	Mobile Source Air Toxic
95-48-7	o-Cresol		X	X		
108-39-4	m-Cresol		X	X		
106-44-5	p-Cresol		X	X		
98-82-8	Cumene		X	X		
N/A	2,4-Dichlorophenoxyacetic Acid (including salts and esters)	2-4-D	X			
72-55-9	1,1-dichloro-2,2-bis(p-chlorophenyl)ethylene	DDE	X			
334-88-3	Diazomethane		X	X		
132-64-9	Dibenzofuran		X	X		
96-12-8	1,2-Dibromo-3-chloropropane		X	X		
84-74-2	Dibutyl phthalate		X	X		
106-46-7	1,4-Dichlorobenzene		X	X		
91-94-1	3,3'-Dichlorobenzidine		X	X		
111-44-4	Dichloroethylether	Bis[2-chloroethyl]ether	X	X		
542-75-6	1,3-Dichloropropene		X	X	X	
62-73-7	Dichlorvos		X	X		
111-42-2	Diethanolamine		X	X		
64-67-5	Diethyl sulfate		X	X		
119-90-4	3,3'-Dimethoxybenzidine		X	X		
60-11-7	4-Dimethylaminoazobenzene		X	X		
121-69-7	N,N-Dimethylaniline		X	X		
119-93-7	3,3'-Dimethylbenzidine		X	X		
79-44-7	Dimethylcarbamoyl chloride		X	X		
68-12-2	N,N-Dimethylformamide		X	X		
57-14-7	1,1-Dimethylhydrazine		X	X		
131-11-3	Dimethyl phthalate		X	X		
77-78-1	Dimethyl sulfate		X	X		
N/A	4,6-Dinitro-o-cresol (including salts)		X			
51-28-5	2,4-Dinitrophenol		X	X		
121-14-2	2-4-Dinitrotoluene		X	X		
123-91-1	1,4-Dioxane	1,4-Diethyleneoxide	X	X		
122-66-7	1,2-Diphenylhydrazine		X	X		
106-89-8	Epichlorohydrin	l-Chloro-2,3-epoxypropane	X	X		
106-88-7	1,2-Epoxybutane		X	X		
140-88-5	Ethyl acrylate		X	X		
100-41-4	Ethylbenzene		X	X		X
51-79-6	Ethyl carbamate	Urethane	X	X		
75-00-3	Ethyl chloride	Chloroethane	X	X		
106-93-4	Ethylene dibromide	Dibromoethane	X	X	X	
107-06-2	Ethylene dichloride	1,2-Dichloroethane	X	X	X	
107-21-1	Ethylene glycol		X	X		
151-56-4	Ethyleneimine	Aziridine	X	X		

Appendix A. Listing of HAPs

CAS Number	Chemical Name	Common Name	CAA HAP	TRI Chemical	Urban HAP	Mobile Source Air Toxic
75-21-8	Ethylene oxide		X	X	X	
96-45-7	Ethylene thiourea		X	X		
75-34-3	Ethylidene dichloride	1-1-Dichloroethane	X	X		
50-00-0	Formaldehyde		X	X	X	X
76-44-8	Heptachlor		X	X		
118-74-1	Hexachlorobenzene		X	X	X	
87-68-3	Hexachlorobutadiene		X	X		
N/A	1,2,3,4,5,6-Hexachlorocyclohexane (all stereoisomers-including lindane)		X			
77-47-4	Hexachlorocyclopentadiene		X	X		
67-72-1	Hexachloroethane		X	X		
822-06-0	Hexamethylene diisocyanate		X			
680-31-9	Hexamethylphosphoramide		X	X		
110-54-3	Hexane		X	X		X
302-01-2	Hydrazine		X	X	X	
7647-01-0	Hydrochloric acid	Hydrogen Chloride	X	X		
7664-39-3	Hydrogen fluoride	Hydrofluoric acid	X	X		
123-31-9	Hydroquinone		X	X		
78-59-1	Isophorone		X			
108-31-6	Maleic anhydride		X	X		
67-56-1	Methanol		X	X		
72-43-5	Methoxychlor		X	X		
74-83-9	Methyl bromide	Bromomethane	X	X		
74-87-3	Methyl chloride	Chloromethane	X	X		
71-55-6	Methyl chloroform	1-1-1-Trichloroethane	X	X		
78-93-3	Methyl ethyl ketone	2-Butanone	X	X		
60-34-4	Methyl hydrazine		X	X		
74-88-4	Methyl iodide	Iodomethane	X	X		
108-10-1	Methyl isobutyl ketone	Hexone	X	X		
624-83-9	Methyl isocyanate		X	X		
80-62-6	Methyl methacrylate		X	X		
1634-04-4	Methyl tert-butyl ether	MTBE	X	X		X
101-14-4	4,4'-Methylenebis	2-chloroaniline	X	X		
75-09-2	Methylene chloride	Dichloromethane	X	X	X	
101-68-8	4-4'-Methylenediphenyl diisocyanate	MDI	X			
101-77-9	4-4'-Methylenedianiline		X	X		
91-20-3	Naphthalene		X	X		X
98-95-3	Nitrobenzene		X	X		
92-93-3	4-Nitrobiphenyl		X	X		
100-02-7	4-Nitrophenol		X	X		
79-46-9	2-Nitropropane		X	X		
684-93-5	N-Nitroso-N-methylurea		X	X		

Appendix A. Listing of HAPs

CAS Number	Chemical Name	Common Name	CAA HAP	TRI Chemical	Urban HAP	Mobile Source Air Toxic
62-75-9	N-Nitrosodimethylamine		X	X		
59-89-2	N-Nitrosomorpholine		X	X		
56-38-2	Parathion		X	X		
82-68-8	Pentachloronitrobenzene	Quintobenzene	X	X		
87-86-5	Pentachlorophenol		X	X		
108-95-2	Phenol		X	X		
106-50-3	p-Phenylenediamine		X	X		
75-44-5	Phosgene		X	X		
7803-51-2	Phosphine		X	X		
7723-14-0	Phosphorus		X	X		
85-44-9	Phthalic anhydride		X	X		
1336-36-3	Polychlorinated biphenyls	Aroclors	X	X	X	
1120-71-4	1-3-Propane sultone		X	X		
57-57-8	beta-Propiolactone		X	X		
123-38-6	Propionaldehyde		X	X		
114-26-1	Propoxur	Baygon	X	X		
78-87-5	Propylene dichloride	1,2-Dichloropropane	X	X	X	
75-56-9	Propylene oxide		X	X		
75-55-8	1-2-Propylenimine	2-Methylaziridine	X	X		
91-22-5	Quinoline		X	X	X	
106-51-4	Quinone	p-Benzoquinone	X	X		
100-42-5	Styrene		X	X		X
96-09-3	Styrene oxide		X	X		
1746-01-6	2,3,7,8-Tetrachlorodibenzo-p-dioxin		X		X	X
79-34-5	1,1,2,2-Tetrachloroethane		X	X	X	
127-18-4	Tetrachloroethylene	Perchloroethylene	X	X	X	
7550-45-0	Titanium tetrachloride		X	X		
108-88-3	Toluene		X	X		X
95-80-7	Toluene-2,4-diamine		X	X		
584-84-9	2,4-Toluene diisocyanate		X	X		
95-53-4	o-Toluidine		X	X		
8001-35-2	Toxaphene	chlorinated camphene	X	X		
120-82-1	1,2,4-Trichlorobenzene		X	X		
79-00-5	1,1,2-Trichloroethane		X	X		
79-01-6	Trichloroethylene		X	X	X	
95-95-4	2-4-5-Trichlorophenol		X	X		
88-06-2	2-4-6-Trichlorophenol		X	X		
121-44-8	Triethylamine		X	X		
1582-09-8	Trifluralin		X	X		
540-84-1	2,2,4-Trimethylpentane		X			
108-05-4	Vinyl acetate		X	X		

Appendix A. Listing of HAPs

CAS Number	Chemical Name	Common Name	CAA HAP	TRI Chemical	Urban HAP	Mobile Source Air Toxic
593-60-2	Vinyl bromide		X	X		
75-01-4	Vinyl chloride		X	X	X	
75-35-4	Vinylidene chloride	1,1-Dichloroethylene	X	X		
1330-20-7	Xylenes (mixed isomers)		X	X		X
95-47-6	o-Xylene		X	X		
108-38-3	m-Xylene		X	X		
106-42-3	p-Xylene		X	X		
	Antimony Compounds		X	X		
	Arsenic Compounds (inorganic including arsine)		X	X	X	X
	Beryllium Compounds		X	X	X	
	Cadmium Compounds		X	X	X	
	Chromium Compounds		X	X	X	X
	Cobalt Compounds		X	X		
	Coke Oven Emissions		X		X	
	Cyanide Compounds **1**		X	X		
	Glycol ethers **2**		X			
	Lead Compounds		X	X	X	X
	Manganese Compounds		X	X	X	X
	Mercury Compounds		X	X	X	X
	Fine mineral fibers **3**		X			
	Nickel Compounds		X	X	X	X
	Polycyclic Organic Matter **4**		X	X	X	X
	Radionuclides (including radon) **5**		X			
	Selenium Compounds		X	X		

1 X'CN where X=H or any other group where a formal dissociation may occur. For example KCN or CA(CN)2.

2 Includes mono- and di- ethers of ethylene glycol, diethylene glycol, triethylene glycol R-(OCH2CH2)n-OR where n= 1,2, or 3; R= alkyl or aryl groups; R' = R, H or groups which, when removed, yield glycol ethers with the structure: R-(OCH2CH)n-OH. Polymers are excluded from the glycol category.

3 Includes mineral fiber emissions from facilities manufacturing or processing glass, rock or slag fibers (or other mineral derived fibers) or average diameter 1micrometer or less.

4 Includes organic compounds with more than one benzene ring, and which have a boiling point greater than or equal to 100 degrees C.

5 A type of atom which spontaneously undergoes radioactive decay.

Appendix B Guide to Federal Agencies that Oversee Air Toxics

This appendix contains descriptions and contacts of the primary EPA organizations that routinely deal with air toxics risk related regulations and information. Additional governmental offices that also deal with air toxics information are also listed. This listing is not meant to be either comprehensive or static and updates and suggestions for additions are welcome (email to mitchell.ken@epa.gov).

The listing is arranged first by EPA headquarters offices and contacts that deal specifically with air toxics risk related issues. EPA Regional air toxics contacts and other governmental agencies that provide health and risk assessment information complete the listing.

1. EPA Headquarters Offices that Work Directly on Air Toxics Issues

a. Office of Air and Radiation. The Office of Air and Radiation (OAR) develops national programs, technical policies, and regulations for controlling air pollution and radiation exposure. OAR is concerned with energy conservation and pollution prevention, indoor and outdoor air quality, industrial air pollution, pollution from vehicles and engines, radon, acid rain, stratospheric ozone depletion, and radiation protection. http://www.epa.gov/air

There are three main offices within OAR that work on air toxics issues - OAQPS, OTAQ, and ORIA.

i. Office of Air Quality Planning and Standards (OAQPS). OAQPS primary mission is to preserve and improve air quality in the United States. OAQPS, as part of this goal, monitors and reports on air quality, air toxics, and emissions. They also watch for visibility issues, as they relate to the level of air pollution. In addition, OAQPS is tasked by the EPA with providing technical information for professionals involved with monitoring and controlling air pollution, creating governmental policies, rules, and guidance for professionals and government, and educating the public about air pollution and what can be done to control and prevent it. http://www.epa.gov/air/oaqps/index.html

ii. Office of Transportation and Air Quality (OTAQ). OTAQ protects public health and the environment by controlling air pollution from motor vehicles, engines, and the fuels used to operate them, and by encouraging travel choices that minimize emissions. These “mobile sources” include cars and light trucks, large trucks and buses, nonroad recreational vehicles (such as dirt bikes and snowmobiles), farm and construction equipment, lawn and garden equipment, marine engines, aircraft, and locomotives. http://www.epa.gov/otaq/

iii. Office of Radiation and Indoor Air (ORIA). The mission of ORIA is to protect the public and the environment from the risks of radiation and indoor air pollution. The

programs within EPA and other agencies to control radiation and indoor air pollution exposures; provides technical assistance to states through EPA's regional offices, and to other agencies having radiation and indoor air protection programs; directs an environmental radiation monitoring program; responds to radiological emergencies; and evaluates and assesses the overall risk and impact of radiation and indoor air pollution. http://www.epa.gov/air/oria.html

b. **Office of Pollution Prevention and Toxics (OPPT).** OPPT has the primary responsibility for administering the Toxic Substances Control Act (TSCA) and the Pollution Prevention Act of 1990. It also manages the Chemical Right-to-Know Initiative and the New and Existing Chemicals programs; the Design for the Environment (DFE), Green Chemistry, and Environmentally Preferable Products (EPP) programs; and the Lead, Asbestos, and Polychlorinated Biphenyls (PCBs) program. http://www.epa.gov/opptintr/.

c. **Office of Research and Development (ORD).** The U.S. Environmental Protection Agency (EPA) relies on sound science to safeguard both human health and the environment. The Office of Research and Development (ORD) is the scientific research arm of EPA. ORD's leading-edge research helps provide the solid underpinning of science and technology for the Agency. ORD conducts research on ways to prevent pollution, protect human health, and reduce risk. The work at ORD laboratories, research centers, and offices across the country helps improve the quality of air, water, soil, and the way we use resources. Applied science at ORD builds our understanding of how to protect and enhance the relationship between humans and the ecosystems of Earth. www.epa.gov/ord

i. **Office of Science Policy (OSP)**. The OSP integrates and communicates scientific information generated by or for ORD's laboratories and centers, as well as ORD's expert advice on the use of scientific information. EPA and the scientific community at large use this information to ensure that EPA's decisions and environmental policies are informed by sound science. http://www.epa.gov/osp/

ii. **The National Center for Environmental Assessment (NCEA).** NCEA is EPA's national resource center for human health and ecological risk assessment. NCEA conducts risk assessments, carries out research to improve the state-of-the-science of risk assessment, and provides guidance and support to risk assessors. www.epa.gov/ncea

iii. **National Exposure Research Laboratory (NERL).** NERL is comprised of several divisions with diversified research specialties. NERL conducts research and development that leads to improved methods, measurements and models to assess and predict exposures of humans and ecosystems to harmful pollutants and other conditions in air, water, soil, and food. www.epa.gov/nerl/

iv. **National Health and Environmental Effects Research Laboratory (NHEERL).** NHEERL is the Agency's focal point for scientific research on the effects of contaminants and environmental stressors on human health and ecosystem integrity. Its research mission and goals help the Agency to identify and understand the

processes that affect our health and environment, and helps the Agency to evaluate the risks that pollution poses to humans and ecosystems. The impact of NHEERL's efforts can be felt far beyond the EPA, by enabling state and local governments to implement effective environmental programs, assisting industry in setting and achieving environmental goals, and collaborating with international governments and organizations on issues of environmental importance. http://www.epa.gov/nheerl/

v. **National Risk Management Research Laboratory (NRMRL).** NRMRL conducts research into ways to prevent and reduce pollution risks that threaten human health and the environment. The laboratory investigates methods to prevent and control pollution of air, land, and water, and to restore ecosystems. The goals of this research are to develop and promote technologies that protect and improve the environment; develop scientific and engineering information to support regulatory and policy decisions; and provide technical support and information transfer to ensure implementation of environmental regulations and strategies at the national and community levels. In addition, NRMRL collaborates with both public and private sector partners to anticipate emerging problems and to foster technologies that reduce the cost of compliance. http://www.epa.gov/ORD/NRMRL/

2. EPA Headquarters Offices that Work on Specific Air Toxics Risk Issues

a. OAQPS Risk and Exposure Assessment Group (REAG). The REAG maintains the scientific and analytical expertise necessary to conduct human and ecological air toxics risk assessments and develop new assessment methodologies, guidelines, and policies for air toxics risk assessments, risk characteristics, and risk communication. The Group also serves as a center of air toxics health risk information for Regional, State, and local agencies. http://www.epa.gov/oar/oaqps/organization/esd/reag.html

b. OAQPS Air Quality Modeling Group (AQMG). The Air Quality Modeling Group is responsible for providing leadership and direction on the full range of atmospheric dispersion models and other mathematical simulation techniques used in assessing source impacts and control strategies. The Group serves as the focal point on modeling techniques for other EPA headquarters staff, Regional Offices, and State and local agencies. It coordinates with ORD on the development of new models and techniques, as well as wider issues of atmospheric research. Finally, the Group conducts modeling analyses to support policy/regulatory decisions in OAQPS. http://www.epa.gov/air/oaqps/organization/emad/aqmg.html

c. OAQPS Emission Factors and Inventories Group (EFIG). Emission inventories are the basis for numerous efforts including trends analysis, regional, and local scale air quality modeling, regulatory impact assessments, and human exposure modeling. These inventories are used in analyses by EPA, State and local agencies, as well as the public. As a central depository for emission facts, inventory data and factor and inventory development references, the EFIG is responsible for providing technical assistance to Regional, State, and local clients. Through this working relationship, inventories are

developed to meet the emerging needs of all their users.
http://www.epa.gov/air/oaqps/organization/emad/efig.html

d. OAQPS Monitoring and Quality Assurance Group (MQAG). MQAG is responsible for identifying ambient monitoring needs based on OAQPS' data requirements, and for developing the monitoring program and quality assurance infrastructure to support these requirements with the highest quality ambient air data.
http://www.epa.gov/air/oaqps/organization/emad/mqag.html

e. **OAQPS Policy, Planning, and Standards Group (PPSG).** The PPSG, which is in the Emissions Standards Division of OAQPS, facilitates planning and development of Division activities and integration of Division programs with other OAQPS and EPA programs. The group is responsible for developing and implementing national emission standards, new source performance standards, control techniques guidelines, regulatory review programs, and other technical documents for specific categories of stationary sources of hazardous and criteria air pollutants. Finally, the Group performs comprehensive analyses of hazardous and criteria air pollutant emissions and control measures for the specified categories of stationary sources. Such analyses typically form the basis for national emission standards or technical guidance documents.
http://www.epa.gov/oar/oaqps/organization/esd/ppsg.html

f. **OTAQ Air Toxics Center.** The Air Toxics Center is OTAQ's resource on mobile source air toxics and other mobile source-related human health and welfare issues. The Center provides expertise on mobile source air toxic emissions, exposure and risk to the Agency. It helps regulators and the public understand the risk from mobile source air toxics to human health and welfare. It also develops mobile source-related air toxics regulations, and addresses air toxics impacts of all mobile source control programs. In addition, it develops information, tools and resources to empower states, communities and individuals to make and implement their own decisions about air toxics. Finally, the Center works to influence the toxics research agenda and strategies of parties internal and external to EPA in order to advance OTAQ's mission. www.epa.gov/otaq/toxics.htm

3. EPA Regional Air Toxics Contacts

Region 1		
FUNCTION	**NAME**	**TELEPHONE**
Maximum Achievable Control Technology (MACT)	Susan Lancey	617-918-1656
Toxics Emissions Inventory	Bob McConnell	617-918-1046
Air Deposition	Ian Cohen	617-918-1655
Air Dispersion/ Deposition Modeling	Brian Hennessey	617-918-1654
Monitoring	Peter Kahn	781-860-4392
Community Assessments	Marybeth Smuts	617-918-1512
Risk Assessment	Marybeth Smuts	617-918-1512
Mobile Sources	Robert Judge	617-918-1045
Indoor Air	Eugene Benoit	617-918-1639

Region 2		
FUNCTION	**NAME**	**TELEPHONE**
Maximum Achievable Control Technology (MACT)	Umesh Dholakia	212-637-4023
Toxics Emissions Inventory	Raymond Forde	212-637-3716
Air Deposition	Bob Kelly	212-637-3709
Air Dispersion/ Deposition Modeling	Bob Kelly	212-637-3709
Monitoring	Mazeeda Khan Avi Teitz	212-637-3715 732-906-6160
Community Assessments	Carol Bellizzi Marlon Gonzales	212-637-3712 212-637-3769
Risk Assessment	Gina Ferreira Carol Bellizzi	212-637-3768 212-637-3712
Mobile Sources	Reema Persaud	212-637-3760
Indoor Air	Larainne Koehler	212-637-4005

Region 3		
FUNCTION	**NAME**	**TELEPHONE**
Maximum Achievable Control Technology (MACT)	Ray Chalmers	215-814-2061
Air Deposition	Al Cimorelli	215-814-2189
Air Dispersion/Deposition Modeling	Al Cimorelli	215-814-2189
Monitoring	Ted Erdman	215-814-2766
Community Assessments	Helene Drago	215-814-5796
Risk Assessment	Alvaro Alvarado	215-814-2109
Mobile Sources	Brian Rehn	215-814-2176
Indoor Air	Fran Dougherty Cristina Schulingkamp	215-814-2083 215-814-2086

Region 4		
FUNCTION	**NAME**	**TELEPHONE**
Maximum Achievable Control Technology (MACT)	Lee Page	404-562-9131
Toxics Emissions Inventory	Leonardo Ceron	404-562-9129
Air Deposition	Dr. John Ackermann Latoya Miller	404-562-9063 404-562-9885
Air Dispersion/Deposition Modeling	Stan Krivo Rick Gillam	404-562-9123 404-562-9049
Monitoring	Van Shrieves Danny France	404-562-9089 706-355-8738
Community Assessments	Paul Wagner	404-562-9100
Risk Assessment	Dr. Kenneth Mitchell (human health/ecological) Dr. Solomon Pollard (human health) Ofia Hodoh (human health) Dr. John Ackermann (ecological) Latoya Miller (ecological)	404-562-9065 404-562-9180 404-562-9176 404-562-9063 404-562-9885
Mobile Sources	Dale Aspy	404-562-9041
Indoor Air	Henry Slack	404-562-9143

Region 5		
FUNCTION	**NAME**	**TELEPHONE**
Maximum Achievable Control Technology (MACT)	Bruce Varner	312-886-6793
Toxics Emissions Inventory	Suzanne King	312-886-6054
Air Deposition	Erin Newman	312-886-4587
Air Dispersion/ Deposition Modeling	Randy Robinson Phuong Nguyen	312-353-6713 312-886-6701
Monitoring	Motria Caudill	312-886-0267
Community Assessments	Jackie Nwia Michele Palmer	312-886-6081 312-886-0387
Risk Assessment	George Bollweg Margaret Sieffert Jaime Julian	312-353-5598 312-353-1151 312-886-9402
Mobile Sources	Suzanne King	312-886-6054
Indoor Air	Jack Barnette Sheila Batka	312-886-6175 312-886-6053

Region 6		
FUNCTION	**NAME**	**TELEPHONE**
Maximum Achievable Control Technology (MACT)	Jeff Robinson	214-665-6435
Toxics Emissions Inventory	Herb Sherrow	214-665-7237
Air Deposition	Phil Crocker	214-665-7373
Air Dispersion/ Deposition Modeling	Quang Nguyen	214-665-7238
Monitoring	Kuenja Chung	214-665-8345
Community Assessments	Ruben Casso	214-665-6763
Risk Assessment	Jeff Yurk	214-665-8309
Mobile Sources	Sandra Rennie	214-665-7367
Indoor Air	Mike Miller	214-665-7550

Region 7		
FUNCTION	**NAME**	**TELEPHONE**
Maximum Achievable Control Technology (MACT)	Richard Tripp	913-551-7566
Toxics Emissions Inventory	Michael Jay	913-551-7460
Air Deposition	Michael Jay	913-551-7460
Air Dispersion/ Deposition Modeling	Richard Daye	913-551-7619
Monitoring	Michael Davis	913-551-7096
Community Assessments	Marcus Rivas	913-551-7669
Risk Assessment	James Hirtz	913-551-7472
Mobile Sources	James Hirtz	913-551-7472
Indoor Air	Robert Dye	913-551-7605

Region 8		
FUNCTION	**NAME**	**TELEPHONE**
Maximum Achievable Control Technology (MACT)	Deldi Reyes	303-312-6055
Toxics Emissions Inventory	Daniel Webster	303-312-6446
Air Deposition	Anne-Marie Patrie	303-312-6524
Air Dispersion/ Deposition Modeling	Victoria Parker-Christensen	303-312-6441
Monitoring	Michael Copeland	303-312-6010
Community Assessments	Victoria Parker-Christensen Anne-Marie Patrie	303-312-6441 303-312-6524
Risk Assessment	Victoria Parker-Christensen Anne-Marie Patrie	303-312-6441 303-312-6524
Mobile Sources	Jeff Kimes	303-312-6445
Indoor Air	Ron Schiller	303-312-6017

Region 9		
FUNCTION	**NAME**	**TELEPHONE**
Maximum Achievable Control Technology (MACT)	Mae Wang John Brock	415-947-4124 415-947-3999
Toxics Emissions Inventory	Larry Biland	415-947-4132
Air Deposition	Pam Tsai Barbara Toole-O'Neil	415-947-4196 415-972-3991
Air Dispersion/ Deposition Modeling	Carol Bohnenkamp Scott Bohning	415-947-4130 415-947-4127
Monitoring	Catherine Brown	415-947-4137
Community Assessments	Mike Bandrowski	415-947-4194
Risk Assessment	Pam Tsai Arnold Den	415-947-4196 415-947-4191
Indoor Air	Barbara Spark	415-947-4189
Mobile Sources	Sylvia Dugre David Jesson	415-947-4149 415-947-4150

Region 10		
FUNCTION	**NAME**	**TELEPHONE**
Maximum Achievable Control Technology (MACT)	Lucita Valiere	206-553-8087
Toxics Emissions Inventory	Madonna Narvaez	206-553-2117
Air Deposition	Madonna Narvaez	206-553-2117
Air Dispersion/ Deposition Modeling	Mahbubul Islam	206-553-6985
Monitoring	Keith Rose	206-553-1949
Community Assessments	Peter Murchie Lisa McArthur	503-326-6554 206-553-1814
Risk Assessment	Julie Wroble	206-553-1079
Mobile Sources	Wayne Elson	206-553-1463
Indoor Air	Ann Wawrukiewicz	206-553-2589

4. Other Federal Agencies

a. **Agency for Toxics Substances and Disease Registry (ATSDR).** The mission of the Agency for Toxic Substances and Disease Registry (ATSDR), as an agency of the U.S. Department of Health and Human Services, is to serve the public by using the best science, taking responsive public health actions, and providing trusted health information to prevent harmful exposures and disease related to toxic substances. ATSDR is directed by congressional mandate to perform specific functions concerning the effect on public health of hazardous substances in the environment. These functions include public health assessments of waste sites, health consultations concerning specific hazardous substances, health surveillance and registries, response to emergency releases of hazardous substances, applied research in support of public health assessments, information development and dissemination, and education and training concerning hazardous substances. http://www.atsdr.cdc.gov/about.html

b. **National Center for Environmental Health (NCEH).** CDC's National Center for Environmental Health (NCEH) strives to promote health and quality of life by preventing or controlling those diseases or deaths that result from interactions between people and their environment. http://www.cdc.gov/nceh/

c. **National Cancer Institute (NCI).** The NCI is a component of the National Institutes of Health (NIH), one of eight agencies that compose the Public Health Service (PHS) in the Department of Health and Human Services (DHHS). The NCI, established under the National Cancer Act of 1937, is the Federal Government's principal agency for cancer research and training. The National Cancer Act of 1971 broadened the scope and responsibilities of the NCI and created the National Cancer Program. Over the years, legislative amendments have maintained the NCI authorities and responsibilities and added new information dissemination mandates as well as a requirement to assess the incorporation of state-of-the-art cancer treatments into clinical practice. The National Cancer Institute coordinates the National Cancer Program, which conducts and supports research, training, health information dissemination, and other programs with respect to the cause, diagnosis, prevention, and treatment of cancer, rehabilitation from cancer, and the continuing care of cancer patients and the families of cancer patients. www.cancer.gov

d. **National Library of Medicine (NLM).** The National Library of Medicine (NLM), on the campus of the National Institutes of Health in Bethesda, Maryland, is the world's largest medical library. The Library collects materials in all areas of biomedicine and health care, as well as works on biomedical aspects of technology, the humanities, and the physical, life, and social sciences. The collections stand at more than 6 million items--books, journals, technical reports, manuscripts, microfilms, photographs and images. Housed within the Library is one of the world's finest medical history collections of old and rare medical works. The Library's collection may be consulted in the reading room or requested on interlibrary loan. NLM is a national resource for all U.S. health science libraries through a National Network of Libraries of Medicine®. http://www.nlm.nih.gov/nlmhome.html

e. **National Institute of Environmental Health Sciences (NIEHS).** Human health and human disease result from three interactive elements: environmental factors, individual susceptibility and age. The mission of the National Institute of Environmental Health Sciences (NIEHS) is to reduce the burden of human illness and dysfunction from environmental causes by understanding each of these elements and how they interrelate. The NIEHS achieves its mission through multidisciplinary biomedical research programs, prevention and intervention efforts, and communication strategies that encompass training, education, technology transfer, and community outreach. http://www.niehs.nih.gov/external/welcome.htm

Appendix C Recommended Dose-Response Values for HAPs

This appendix presents tabulated dose-response assessments that the Office of Air Quality Planning and Standards (OAQPS) uses for risk assessments of hazardous air pollutants. A description of the derivation of these values, along with any updates can be found at the following website: http://www.epa.gov/ttn/atw/toxsource/summary.html.

Table 1. Prioritized Dose-Response Values (10/28/03)			WOE[3] for Cancer		CHRONIC INHALATION				CHRONIC ORAL			
					NONCANCER		CANCER		NONCANCER		CANCER	
CHEMICAL NAME	CAS NO.[1]	HAP NO.[2]	EPA	IARC	mg/m3	SOURCE	1/(ug/m3)	SOURCE	mg/kg/d	SOURCE	1/(mg/kg/d)	SOURCE
Acetaldehyde	75-07-0	1	B2	2B	0.009	IRIS	2.2E-06	IRIS				
Acetamide	60-35-5	2		2B			2.0E-05	CAL				
Acetonitrile	75-05-8	3	D		0.06	IRIS						
Acetophenone	98-86-2	4	D									
Acrolein	107-02-8	6		3	0.00002	IRIS						
Acrylamide	79-06-1	7	B2	2A	0.0007	P-CAL	1.3E-03	IRIS				
Acrylic acid	79-10-7	8			0.001	IRIS						
Acrylonitrile	107-13-1	9	B1	2A	0.002	IRIS	6.8E-05	IRIS				
Allyl chloride	107-05-1	10	C	3	0.001	IRIS	6.0E-06	CAL				
Aniline	62-53-3	12	B2	3	0.001	IRIS	1.6E-06	CAL				
Antimony compounds	7440-36-0	173										
Antimony pentoxide	1314-60-9	173										
Antimony potassium tartrate	304-61-0	173										
Antimony tetroxide	1332-81-6	173										
Antimony trioxide	1309-64-4	173		2B	0.0002	IRIS						
Arsenic compounds	7440-38-2	174	A	1	0.00003	CAL	4.3E-03	IRIS				
Arsine	7784-42-1	174			0.00005	IRIS						
Benzene	71-43-2	15	A	1	0.03	IRIS	7.8E-06	IRIS				
Benzidine	92-87-5	16	A		0.01	P-CAL	6.7E-02	IRIS				
Benzotrichloride	98-07-7	17	B2	2B			3.7E-03	Conv. Oral				
Benzyl chloride	100-44-7	18	B2	2B			4.9E-05	CAL				
Beryllium compounds	7440-41-7	175	B1	1	0.00002	IRIS	2.4E-03	IRIS				
Biphenyl	92-52-4	19	D									
Bis(2-ethylhexyl)phthalate	117-81-7	20	B2	2B	0.01	CAL	2.4E-06	CAL				
Bis(chloromethyl)ether	542-88-1	21	A	1			6.2E-02	IRIS				
Bromoform	75-25-2	22	B2	3			1.1E-06	IRIS				
1,3-Butadiene	106-99-0	23	A	2A	0.002	IRIS	3.0E-05	IRIS				
Cadmium compounds	7440-43-9	176	B1	1	0.00002	CAL	1.8E-03	IRIS	0.0005	IRIS		
Captan	133-06-2	26	B2	3			1.0E-06	Conv. Oral				
Carbaryl	63-25-2	27										
Carbon disulfide	75-15-0	28			0.7	IRIS						
Carbon tetrachloride	56-23-5	29	B2	2B	0.04	CAL	1.5E-05	IRIS				
Chloramben	133-90-4	32										
Chlordane	57-74-9	33	B2	2B	0.0007	IRIS	1.0E-04	IRIS	0.0005	IRIS	3.5E-01	IRIS
Chlorine	7782-50-5	34			0.0002	CAL						
Chloroacetic acid	79-11-8	35										
2-Chloroacetophenone	532-27-4	36			0.00003	IRIS						
Chlorobenzene	108-90-7	37	D		1	CAL						
Chlorobenzilate	510-15-6	38	B2				7.8E-05	HEAST				
Chloroform	67-66-3	39	B2	2B	0.098	ATSDR						
Chloroprene	126-99-8	41			0.007	HEAST						
Chromium (III) compounds	16065-83-1	177	D									
Chromium (VI) compounds	18540-29-9	177	A	1	0.0001	IRIS	1.2E-02	IRIS				

Table 1. Prioritized Dose-Response Values (10/28/03)			WOE[3] for Cancer		CHRONIC INHALATION				CHRONIC ORAL			
					NONCANCER		CANCER		NONCANCER		CANCER	
CHEMICAL NAME	CAS NO.[1]	HAP NO.[2]	EPA	IARC	mg/m3	SOURCE	1/(ug/m3)	SOURCE	mg/kg/d	SOURCE	1/(mg/kg/d)	SOURCE
Chromium (VI) trioxide, chromic acid mist	11115-74-5	177	A	1	0.000008	IRIS						
Cobalt compounds	7440-48-4	178			0.0001	ATSDR						
Coke Oven Emissions	8007-45-2	179	A				6.2E-04	IRIS				
m-Cresol	108-39-4	44	C									
o-Cresol	95-48-7	43	C									
p-Cresol	106-44-5	45	C									
Cresols (mixed)	1319-77-3	42	C		0.6	CAL						
Cumene	98-82-8	46	D		0.4	IRIS						
Cyanazine	21725-46-2	180	C				2.4E-04	Conv. Oral				
Cyanide compounds	57-12-5	180	D									
Acetone cyanohydrin	75-86-5	180			0.01	HEAST						
Calcium cyanide	592-01-8	180										
Copper cyanide	544-92-3	180										
Cyanogen	460-19-5	180										
Cyanogen bromide	506-68-3	180										
Cyanogen chloride	506-77-4	180										
Ethylene cyanohydrin	109-78-4	180										
Hydrogen cyanide	74-90-8	180			0.003	IRIS						
Potassium cyanide	151-50-8	180										
Potassium silver cyanide	506-61-6	180										
Silver cyanide	506-64-9	180										
Sodium cyanide	143-33-9	180										
Thiocyanic acid, 2-(benzothiazolylthio) methyl est	21564-17-0	180										
Zinc cyanide	557-21-1	180										
2,4-D, salts and esters	94-75-7	47										
DDE	72-55-9	48	B2				9.7E-05	Conv. Oral			3.4E-01	IRIS
1,2-Dibromo-3-chloropropane	96-12-8	51	B2		0.0002	IRIS	2.0E-03	CAL				
Dibutylphthalate	84-74-2	52	D									
p-Dichlorobenzene	106-46-7	53	C	2B	0.8	IRIS	1.1E-05	CAL				
3,3'-Dichlorobenzidine	91-94-1	54	B2	2B			3.4E-04	CAL				
Dichloroethyl ether	111-44-4	55	B2				3.3E-04	IRIS				
1,3-dichloropropene	542-75-6	56	B2	2B	0.02	IRIS	4.0E-06	IRIS				
Dichlorvos	62-73-7	57	B2	2B	0.0005	IRIS	8.3E-05	Conv. Oral				
Diesel engine emissions	DIESEL EMIS.	190	B1		0.005	IRIS						
Diethanolamine	111-42-2	58			0.003	CAL						
3,3'-Dimethoxybenzidine	119-90-4	61	B2	2B			4.0E-06	Conv. Oral				
p-Dimethylaminoazobenzene	60-11-7	62		2B			1.3E-03	CAL				
3,3'-Dimethylbenzidine	119-93-7	63	B2				2.6E-03	Conv. Oral				
Dimethyl formamide	68-12-2	65		2B	0.03	IRIS						
N,N-dimethylaniline	121-69-7	59		3								
1,1-Dimethylhydrazine	57-14-7	66	B2	2B								
2,4-dinitrophenol	51-28-5	70										
2,4-Dinitrotoluene	121-14-2	71	B2	2B	0.007	P-CAL	8.9E-05	CAL				

Table 1. Prioritized Dose-Response Values (10/28/03)			WOE[3] for Cancer		CHRONIC INHALATION				CHRONIC ORAL			
					NONCANCER		CANCER		NONCANCER		CANCER	
CHEMICAL NAME	CAS NO.[1]	HAP NO.[2]	EPA	IARC	mg/m3	SOURCE	1/(ug/m3)	SOURCE	mg/kg/d	SOURCE	1/(mg/kg/d)	SOURCE
2,4/2,6-Dinitrotoluene (mixture)	25321-14-6	71	B2	2B			1.9E-04	Conv. Oral				
1,4-Dioxane	123-91-1	72	B2	2B	3	CAL	3.1E-06	Conv. Oral				
1,2-Diphenylhydrazine	122-66-7	73	B2				2.2E-04	IRIS				
Epichlorohydrin	106-89-8	74	B2	2A	0.001	IRIS	1.2E-06	IRIS				
1,2-Epoxybutane	106-88-7	75			0.02	IRIS						
Ethyl acrylate	140-88-5	76	B2	2B			1.4E-05	Conv. Oral				
Ethyl benzene	100-41-4	77	D		1	IRIS						
Ethyl carbamate	51-79-6	78		2B			2.9E-04	CAL				
Ethyl chloride	75-00-3	79			10	IRIS						
Ethylene dibromide	106-93-4	80	B2	2A	0.0008	CAL	2.2E-04	IRIS				
Ethylene dichloride	107-06-2	81	B2	2B	2.4	ATSDR	2.6E-05	IRIS				
Ethylene glycol	107-21-1	82			0.4	CAL						
Ethylene oxide	75-21-8	84	B1	1	0.03	CAL	8.8E-05	CAL				
Ethylene thiourea	96-45-7	85	B2	2B	0.003	P-CAL	1.3E-05	CAL				
Ethylidene dichloride	75-34-3	86	C		0.5	HEAST	1.6E-06	CAL				
Formaldehyde	50-00-0	87	B1	2A	0.0098	ATSDR	5.5E-09	EPA OAQPS				
Diethylene glycol monobutyl ether	112-34-5	181			0.02	HEAST						
Diethylene glycol monoethyl ether	111-90-0	181										
Ethylene glycol butyl ether	111-76-2	181	C		13	IRIS						
Ethylene glycol ethyl ether	110-80-5	181			0.2	IRIS						
Ethylene glycol ethyl ether acetate	111-15-9	181			0.3	CAL						
Ethylene glycol methyl ether	109-86-4	181			0.02	IRIS						
Ethylene glycol methyl ether acetate	110-49-6	181			0.09	CAL						
Heptachlor	76-44-8	88	B2	2B			1.3E-03	IRIS	0.0005	IRIS	4.5E+00	IRIS
Hexachlorobenzene	118-74-1	89	B2	2B	0.003	P-CAL	4.6E-04	IRIS	0.0008	IRIS	1.6E+00	IRIS
Hexachlorobutadiene	87-68-3	90	C	3	0.09	P-CAL	2.2E-05	IRIS				
Hexachlorocyclopentadiene	77-47-4	91	E		0.0002	IRIS						
Hexachlorodibenzo-p-dioxin, mixture	19408-74-3	187	B2				1.3E+00	IRIS			6.2E+03	IRIS
Hexachloroethane	67-72-1	92	C	3	0.08	P-CAL	4.0E-06	IRIS				
Hexamethylene-1,6-diisocyanate	822-06-0	93			0.00001	IRIS						
n-Hexane	110-54-3	95			0.2	IRIS						
Hydrazine	302-01-2	96	B2	2B	0.0002	CAL	4.9E-03	IRIS				
Hydrochloric acid	7647-01-0	97			0.02	IRIS						
Hydrofluoric acid	7664-39-3	98			0.03	CAL						
Hydroquinone	123-31-9	99										
Isophorone	78-59-1	100	C		2	CAL	2.7E-07	Conv. Oral				
Lead compounds	7439-92-1	182	B2	2B	0.0015	EPA OAQPS						
Tetraethyl lead	78-00-2	182							0.0000001	IRIS		
Lindane (gamma-HCH)	58-89-9	101		2B	0.0003	P-CAL	3.1E-04	CAL	0.0003	IRIS	1.1E+00	CAL
alpha-Hexachlorocyclohexane (a-HCH)	319-84-6	101	B2	2B	0.02	P-CAL	1.8E-03	IRIS	0.008	ATSDR	6.3E+00	IRIS
beta-Hexachlorocyclohexane (b-HCH)	319-85-7	101	C	2B	0.002	P-CAL	5.3E-04	IRIS			1.8E+00	IRIS
technical Hexachlorocyclohexane (HCH)	608-73-1	101	B2	2B			5.1E-04	IRIS			1.8E+00	IRIS
Maleic anhydride	108-31-6	102			0.0007	CAL						

Table 1. Prioritized Dose-Response Values (10/28/03)			WOE[3] for Cancer		CHRONIC INHALATION				CHRONIC ORAL			
					NONCANCER		CANCER		NONCANCER		CANCER	
CHEMICAL NAME	CAS NO.[1]	HAP NO.[2]	EPA	IARC	mg/m3	SOURCE	1/(ug/m3)	SOURCE	mg/kg/d	SOURCE	1/(mg/kg/d)	SOURCE
Manganese compounds	7439-96-5	183	D		0.00005	IRIS						
Mercuric chloride	7487-94-7	184	C		0.00009	CAL			0.0003	IRIS		
Mercury (elemental)	7439-97-6	184	D		0.0003	IRIS						
Methyl mercury	22967-92-6	184	C						0.0001	IRIS		
Phenylmercuric acetate	62-38-4	184							0.00008	IRIS		
Methanol	67-56-1	103			4	CAL						
Methoxychlor	72-43-5	104	D	3					0.005	IRIS		
Methyl bromide	74-83-9	105	D		0.005	IRIS						
Methyl chloride	74-87-3	106	D		0.09	IRIS						
Methyl ethyl ketone	78-93-3	108			5	IRIS						
Methyl isobutyl ketone	108-10-1	111			3	IRIS						
Methyl isocyanate	624-83-9	112			0.001	CAL						
Methyl methacrylate	80-62-6	113	E		0.7	IRIS						
Methyl tert-butyl ether	1634-04-4	114			3	IRIS						
4,4'-Methylene bis(2-chloroaniline)	101-14-4	115	B2	2A			4.3E-04	CAL				
Methylene chloride	75-09-2	116	B2	2B	1	ATSDR	4.7E-07	IRIS				
Methylene diphenyl diisocyanate	101-68-8	117	D		0.0006	IRIS						
4,4'-Methylenedianiline	101-77-9	118		2B	0.02	CAL	4.6E-04	CAL				
Naphthalene	91-20-3	119	C		0.003	IRIS						
Nickel compounds	7440-02-0	186	A	2B	0.0002	ATSDR						
Nickel oxide	1313-99-1	186			0.0001	CAL						
Nickel refinery dust	NI_DUST	186	A				2.4E-04	IRIS				
Nickel subsulfide	12035-72-2	186	A				4.8E-04	IRIS				
Nitrobenzene	98-95-3	120	D	2B	0.03	P-CAL						
2-Nitropropane	79-46-9	123	B2	2B	0.02	IRIS	5.6E-06	EPA OAQPS				
Nitrosodimethylamine	62-75-9	125	B2	2A			1.4E-02	IRIS				
N-Nitrosomorpholine	59-89-2	126		2B			1.9E-03	CAL				
Parathion	56-38-2	127	C	3								
Polychlorinated biphenyls	1336-36-3	136	B2	2A			1.0E-04	IRIS			2.0E+00	IRIS
Aroclor 1016	12674-11-2	136							0.00007	IRIS		
Aroclor 1254	11097-69-1	136							0.00002	IRIS		
Pentachloronitrobenzene	82-68-8	128	C	3			7.4E-05	Conv. Oral				
Pentachlorophenol	87-86-5	129	B2	2B	0.1	P-CAL	5.1E-06	CAL				
Phenol	108-95-2	130	D	3	0.2	CAL						
p-Phenylenediamine	106-50-3	131										
Phosgene	75-44-5	132			0.0003	P-CAL						
Phosphine	7803-51-2	133	D		0.0003	IRIS						
Phosphorus, white	7723-14-0	134	D		0.00007	P-CAL						
Phthalic anhydride	85-44-9	135			0.02	CAL						
Polybrominated Diphenyl Ethers	PBDE	187							0.007	ATSDR		
Acenaphthene	83-32-9	187	D						0.06	IRIS		
Acenaphthylene	206-96-8	187	D									
Anthracene	120-12-7	187	D	3					0.3	IRIS		

Table 1. Prioritized Dose-Response Values (10/28/03)			WOE[3] for Cancer		CHRONIC INHALATION				CHRONIC ORAL			
					NONCANCER		CANCER		NONCANCER		CANCER	
CHEMICAL NAME	CAS NO.[1]	HAP NO.[2]	EPA	IARC	mg/m3	SOURCE	1/(ug/m3)	SOURCE	mg/kg/d	SOURCE	1/(mg/kg/d)	SOURCE
Benzo(a)anthracene	56-55-3	187	B2	2A			1.1E-04	CAL			1.2E+00	CAL
Benzo(b)fluoranthene	205-99-2	187	B2	2B			1.1E-04	CAL			1.2E+00	CAL
Benzo[j]fluoranthene	205-82-3	187		2B			1.1E-04	CAL			1.2E+00	CAL
Benzo(k)fluoranthene	207-08-9	187	B2	2B			1.1E-04	CAL			1.2E+00	CAL
Benzo(g,h,i)perylene	191-24-2	187	D	3								
Benzo(a)pyrene	50-32-8	187	B2	2A			1.1E-03	CAL			7.3E+00	IRIS
Benzo(e)pyrene	192-97-2	187		3								
Carbazole	86-74-8	187	B2	3			5.7E-06	Conv. Oral			2.0E-02	HEAST
beta-Chloronaphthalene	91-58-7	187							0.08	IRIS		
Chrysene	218-01-9	187	B2	3			1.1E-05	CAL			1.2E-01	CAL
Dibenz[a,h]acridine	226-36-8	187		2B			1.1E-04	CAL			1.2E+00	CAL
Dibenz[a,j]acridine	224-42-0	187		2B			1.1E-04	CAL			1.2E+00	CAL
Dibenz(a,h)anthracene	53-70-3	187	B2	2A			1.2E-03	CAL			4.1E+00	CAL
7H-Dibenzo[c,g]carbazole	194-59-2	187		2B			1.1E-03	CAL			1.2E+01	CAL
Dibenzo[a,e]pyrene	192-65-4	187		2B			1.1E-03	CAL			1.2E+01	CAL
Dibenzo[a,h]pyrene	189-64-0	187		2B			1.1E-02	CAL			1.2E+02	CAL
Dibenzo[a,i]pyrene	189-55-9	187		2B			1.1E-02	CAL			1.2E+02	CAL
Dibenzo[a,l]pyrene	191-30-0	187		2B			1.1E-02	CAL			1.2E+02	CAL
7,12-Dimethylbenz(a)anthracene	57-97-6	187					7.1E-02	CAL			2.5E+02	CAL
1,6-Dinitropyrene	42397-64-8	187		2B			1.1E-02	CAL			1.2E+02	CAL
1,8-Dinitropyrene	42397-65-9	187		2B			1.1E-03	CAL			1.2E+01	CAL
Fluoranthene	206-44-0	187	D	3					0.04	IRIS		
Fluorene	86-73-7	187	D	3					0.04	IRIS		
Indeno(1,2,3-cd)pyrene	193-39-5	187	B2	2B			1.1E-04	CAL			1.2E+00	CAL
3-Methylcholanthrene	56-49-5	187					6.3E-03	CAL			2.2E+01	CAL
5-Methylchrysene	3697-24-3	187		2B			1.1E-03	CAL			1.2E+01	CAL
1-Methylnaphthalene	90-12-0	187							0.07	ATSDR		
5-Nitroacenaphthene	602-87-9	187		2B			3.7E-05	CAL			1.3E-01	CAL
6-Nitrochrysene	7496-02-8	187		2B			1.1E-02	CAL			1.2E+02	CAL
2-Nitrofluorene	607-57-8	187		2B			1.1E-05	CAL			1.2E-01	CAL
1-Nitropyrene	5522-43-0	187		2B			1.1E-04	CAL			1.2E+00	CAL
4-Nitropyrene	57835-92-4	187		2B			1.1E-04	CAL			1.2E+00	CAL
Phenanthrene	85-01-8	187	D									
Pyrene	129-00-0	187	D						0.03	IRIS		
1,3-Propane sultone	1120-71-4	137		2B			6.9E-04	CAL				
Propoxur	114-26-1	140	B2									
Propylene dichloride	78-87-5	141	B2		0.004	IRIS	1.9E-05	Conv. Oral				
Propylene oxide	75-56-9	142	B2	2B	0.03	IRIS	3.7E-06	IRIS				
Quinoline	91-22-5	144	B2									
Selenium compounds	7782-49-2	189	D		0.02	CAL						
Hydrogen selenide	7783-07-5	189			0.00008	CAL						
Selenious acid	7783-00-8	189	D									
Selenourea	630-10-4	189										

Table 1. Prioritized Dose-Response Values (10/28/03)			WOE[3] for Cancer		CHRONIC INHALATION				CHRONIC ORAL			
					NONCANCER		CANCER		NONCANCER		CANCER	
CHEMICAL NAME	CAS NO.[1]	HAP NO.[2]	EPA	IARC	mg/m3	SOURCE	1/(ug/m3)	SOURCE	mg/kg/d	SOURCE	1/(mg/kg/d)	SOURCE
Styrene	100-42-5	146		2B	1	IRIS						
Styrene oxide	96-09-3	147		2A	0.006	P-CAL						
2,3,7,8-Tetrachlorodibenzo-p-dioxin	1746-01-6	148	B2		4E-08	CAL	3.3E+01	EPA ORD	1E-09	ATSDR	1.5E+05	EPA ORD
1,1,2,2-Tetrachloroethane	79-34-5	149	C	3			5.8E-05	IRIS				
Tetrachloroethene	127-18-4	150	B2-C	2A	0.27	ATSDR	5.9E-06	CAL				
Titanium tetrachloride	7550-45-0	151			0.0001	ATSDR						
Toluene	108-88-3	152	D	3	0.4	IRIS						
2,4-Toluene diamine	95-80-7	153	B2				1.1E-03	CAL				
2,4/2,6-Toluene diisocyanate mixture (TDI)	26471-62-5	154		2B	0.00007	IRIS	1.1E-05	CAL				
o-Toluidine	95-53-4	155	B2	2B			5.1E-05	CAL				
Toxaphene	8001-35-2	156	B2	2B			3.2E-04	IRIS			1.1E+00	IRIS
1,2,4-Trichlorobenzene	120-82-1	157	D		0.2	HEAST						
1,1,2-Trichloroethane	79-00-5	158	C	3	0.4	P-CAL	1.6E-05	IRIS				
1,1,1-Trichloroethane	71-55-6	107	D		1	CAL						
Trichloroethylene	79-01-6	159	B2-C	2A	0.6	CAL	2.0E-06	CAL				
2,4,5-Trichlorophenol	95-95-4	160										
2,4,6-Trichlorophenol	88-06-2	161	B2				3.1E-06	IRIS				
Triethylamine	121-44-8	162			0.007	IRIS						
Trifluralin	1582-09-8	163	C	3			2.2E-06	Conv. Oral	0.0075	IRIS	7.7E-03	IRIS
Uranium compounds	7440-61-1	188			0.0003	ATSDR						
Uranium, soluble salts	URANSOLS	188										
Vinyl acetate	108-05-4	165		2B	0.2	IRIS						
Vinyl bromide	593-60-2	166	B2	2A	0.003	IRIS	3.2E-05	HEAST				
Vinyl chloride	75-01-4	167	A	1	0.1	IRIS	8.8E-06	IRIS				
Vinylidene chloride	75-35-4	168	C		0.2	IRIS						
m-Xylene	108-38-3	171										
o-Xylene	95-47-6	170										
Xylenes (mixed)	1330-20-7	169			0.1	IRIS						

1Chemical Abstracts Services number for the compound.
2Position of the compound on the HAP list in the Clean Air Act (112[b][2])
3Weight-of-evidence. See http://www.epa/iris/carcino.htm, http://193.51.164.11/monoeval/grlist.html.

Table 2. Acute Dose-Response Values (10/22/03)			AEGL-1	AEGL-2	AEGL-3	ERPG-1	ERPG-2	ERPG-3	IDLH/10	MRL	REL
CHEMICAL NAME	CAS NO.	HAP NO.	mg/m3	mg/m3	mg/m3	mg/m3	mg/m3	mg/m3	mg/m3	mg/m3	mg/m3
Acetaldehyde	75-07-0	1				18	360	1800	360		
Acetonitrile	75-05-8	3							84		
Acrolein	107-02-8	6	0.069 [p]	0.23 [p]	3.2 [p]	0.23	1.1	6.9	0.46	0.00011	0.00019
Acrylamide	79-06-1	7							6		
Acrylic acid	79-10-7	8	2.9 [i]	140 [i]	530 [i]	5.9	150	2200			6
Acrylonitrile	107-13-1	9				22	77	170	19	0.22	
Allyl chloride	107-05-1	10				9.4	130	940	78		
Aniline	62-53-3	12	30 [f]	46 [f]	57 [f]				38		
Anisidine	90-04-0	13							5		
Antimony compounds	7440-36-0	173							5		
Arsenic compounds	7440-38-2	174							0.5		0.00019
Arsine	7784-42-1	174		0.54 [f]	1.6 [f]		1.6	4.8	0.96		0.16
Benzene	71-43-2	15		2600 [p]	13000 [p]	160	480	3200	160	0.16	1.3
Benzyl chloride	100-44-7	18				5.2	52	130	52		0.24
Beryllium compounds	7440-41-7	175					0.025	0.1	0.4		
Bis(chloromethyl)ether	542-88-1	21					0.47	2.4			
Bromoform	75-25-2	22							880		
1,3-Butadiene	106-99-0	23				22	440	11000	440		
Cadmium compounds	7440-43-9	176							9		
Carbaryl	63-25-2	27							10		
Carbon disulfide	75-15-0	28	12 [p]	500 [p]	1500 [p]	3.1	160	1600	160		6.2
Carbon tetrachloride	56-23-5	29	75 [i]	350 [i]	1100 [i]	130	630	4700	130	1.3	1.9
Chlordane	57-74-9	33							10		
Chlorine	7782-50-5	34	1.5 [i]	5.8 [i]	58 [i]	2.9	8.7	58	2.9		0.21
Chloroacetic acid	79-11-8	35		26 [p]							
Chlorobenzene	108-90-7	37							460		
Chloroform	67-66-3	39		430 [p]	8300 [p]		240	24000	240	0.49	0.15
Chloromethyl methyl ether	107-30-2	40		0.2 [i]	3.1 [i]		3.3	33			
Chloroprene	126-99-8	41							110		
Chromium (VI) compounds	18540-29-9	177							1.5		
Chromium (VI) trioxide, chromic acid mist	11115-74-5	177							1.5		
Cobalt compounds	7440-48-4	178							2		
m-Cresol	108-39-4	44							110		
o-Cresol	95-48-7	43							110		
p-Cresol	106-44-5	45							110		
Cresols (mixed)	1319-77-3	42							110		
Cumene	98-82-8	46							440		
Cyanide compounds	57-12-5	180							2.5		
Acetone cyanohydrin	75-86-5	180	2.9 [p]	19 [p]	52 [p]						
Cyanogen chloride	506-77-4	180					1	10			
Hydrogen cyanide	74-90-8	180	2.2 [i]	7.8 [f]	17 [f]		11	28	5.5		0.34
2,4-D, salts and esters	94-75-7	47							10		

Table 2. Acute Dose-Response Values (10/22/03)			AEGL-1	AEGL-2	AEGL-3	ERPG-1	ERPG-2	ERPG-3	IDLH/10	MRL	REL
CHEMICAL NAME	CAS NO.	HAP NO.	mg/m3	mg/m3	mg/m3	mg/m3	mg/m3	mg/m3	mg/m3	mg/m3	mg/m3
Dibutylphthalate	84-74-2	52							400		
p-Dichlorobenzene	106-46-7	53							90	4.8	
Dichloroethyl ether	111-44-4	55							58		
Dichlorvos	62-73-7	57							10	0.018	
Dimethyl formamide	68-12-2	65		270 [p]	540 [p]	6	300	600	150		
Dimethyl phthalate	131-11-3	67							200		
Dimethyl sulfate	77-78-1	68							3.6		
N,N-dimethylaniline	121-69-7	59							50		
1,1-Dimethylhydrazine	57-14-7	66		7.4 [f]	27 [f]				3.7		
4,6-Dinitro-o-cresol	534-52-1	69							0.5		
2,4-Dinitrotoluene	121-14-2	71							5		
1,4-Dioxane	123-91-1	72	61 [p]	1200 [p]	2700 [p]				180		3
Epichlorohydrin	106-89-8	74	19 [p]	91 [p]	270 [p]	7.6	76	380	28		1.3
Ethyl acrylate	140-88-5	76				0.041	120	1200	1400		
Ethyl benzene	100-41-4	77							350		
Ethyl chloride	75-00-3	79							1000	40	
Ethylene dibromide	106-93-4	80							77		
Ethylene dichloride	107-06-2	81				200	810	810	20		
Ethylene glycol	107-21-1	82								1.3	
Ethylene imine (aziridine)	151-56-4	83		8.1 [i]	17 [i]						
Ethylene oxide	75-21-8	84		81 [i]	360 [i]		90	900	140		
Ethylidene dichloride	75-34-3	86							1200		
Formaldehyde	50-00-0	87	0.49 [p]	17 [p]	61 [p]	1.2	12	31	2.5	0.049	0.094
Ethylene glycol butyl ether	111-76-2	181							340	29	14
Ethylene glycol ethyl ether	110-80-5	181							180		0.37
Ethylene glycol ethyl ether acetate	111-15-9	181									0.14
Ethylene glycol methyl ether	109-86-4	181									0.093
Heptachlor	76-44-8	88							3.5		
Hexachlorobutadiene	87-68-3	90				32	110	320			
Hexachloroethane	67-72-1	92								58	
n-Hexane	110-54-3	95							390		
Hydrazine	302-01-2	96	0.13 [i]	17 [i]	46 [i]	0.65	6.5	39	6.5		
Hydrochloric acid	7647-01-0	97	2.7 [i]	33 [i]	150 [i]	4.5	30	220	7.5		2.1
Hydrofluoric acid	7664-39-3	98	0.82 [i]	20 [i]	36 [i]	1.6	16	41	2.5	0.025	0.24
Hydroquinone	123-31-9	99							5		
Lead compounds	7439-92-1	182							10		
Tetraethyl lead	78-00-2	182							4		
Tetramethyl lead	75-74-1	182							4		
Lindane (gamma-HCH)	58-89-9	101							5		
Maleic anhydride	108-31-6	102							1		
Manganese compounds	7439-96-5	183							50		
Mercury (elemental)	7439-97-6	184					1.6	16			0.0018

Table 2. Acute Dose-Response Values (10/22/03)			AEGL-1	AEGL-2	AEGL-3	ERPG-1	ERPG-2	ERPG-3	IDLH/10	MRL	REL
CHEMICAL NAME	CAS NO.	HAP NO.	mg/m3	mg/m3	mg/m3	mg/m3	mg/m3	mg/m3	mg/m3	mg/m3	mg/m3
Mercury compounds	HG_CMPDS	184							1		
Methyl mercury	22967-92-6	184							0.2		
Methanol	67-56-1	103	690 [i]	2700 [i]	10000 [i]	260	1300	6500	790		28
Methoxychlor	72-43-5	104							500		
Methyl bromide	74-83-9	105					190	780	97	0.19	3.9
Methyl chloride	74-87-3	106					830	2100	410	1	
Methyl ethyl ketone	78-93-3	108	290 [p]	5000 [p]	12000 [p]						13
Methyl hydrazine	60-34-4	109		3.6 [f]	11 [f]				7.2		
Methyl iodide	74-88-4	110				150	290	730	58		
Methyl isocyanate	624-83-9	112		0.16 [i]	0.47 [i]	0.058	1.2	12	0.7		
Methyl methacrylate	80-62-6	113							410		
Methyl tert-butyl ether	1634-04-4	114								7.2	
Methylene chloride	75-09-2	116				690	2600	14000	800	2.1	14
Methylene diphenyl diisocyanate	101-68-8	117				0.2	2	25	7.5		
Naphthalene	91-20-3	119							130		
Nickel carbonyl	13463-39-3	186		0.25 [i]	1.1 [i]				1.4		
Nickel compounds	7440-02-0	186							1		0.006
Nitrobenzene	98-95-3	120							100		
2-Nitropropane	79-46-9	123							36		
Parathion	56-38-2	127							1		
Pentachlorophenol	87-86-5	129							0.25		
Phenol	108-95-2	130	17 [i]	58 [i]	180 [i]	38	190	770	96		5.8
Phosgene	75-44-5	132		1.2 [f]	3 [f]		0.81	4	0.81		0.004
Phosphine	7803-51-2	133		2.8 [i]	5 [i]		0.7	7			
Phosphorus, white	7723-14-0	134								0.02	
Phthalic anhydride	85-44-9	135							6		
Propylene dichloride	78-87-5	141							180	0.23	
Propylene oxide	75-56-9	142	140 [i]	690 [i]	1400 [i]	120	590	1800	95		3.1
1,2-Propyleneimine	75-55-8	143		28 [i]	54 [i]						
Quinone	106-51-4	145							10		
Selenium compounds	7782-49-2	189							0.1		
Hydrogen selenide	7783-07-5	189		2.4 [p]	7.3 [p]		0.66	6.6	0.33		0.005
Styrene	100-42-5	146				210	1100	4300	300		21
1,1,2,2-Tetrachloroethane	79-34-5	149							69		
Tetrachloroethene	127-18-4	150	240 [i]	1600 [i]	8100 [i]	680	1400	6800	100	1.4	20
Titanium tetrachloride	7550-45-0	151	0.54 [p]	7.8 [p]	44 [p]	5	20	100			
Toluene	108-88-3	152	750 [i]	1900 [i]	11000 [i]	190	1100	3800	190	3.8	37
2,4-Toluene diisocyanate	584-84-9	154	0.14 [i]	0.59 [i]	3.6 [i]	0.071			1.8		
o-Toluidine	95-53-4	155							22		
1,1,2-Trichloroethane	79-00-5	158							55		
1,1,1-Trichloroethane	71-55-6	107	1300 [i]	3300 [i]	21000 [i]	1900	3800	19000	380	11	68
Trichloroethylene	79-01-6	159	700 [p]	2400 [p]	20000 [p]	540	2700	27000		11	

Table 2. Acute Dose-Response Values (10/22/03)			AEGL-1	AEGL-2	AEGL-3	ERPG-1	ERPG-2	ERPG-3	IDLH/10	MRL	REL
CHEMICAL NAME	CAS NO.	HAP NO.	mg/m3	mg/m3	mg/m3	mg/m3	mg/m3	mg/m3	mg/m3	mg/m3	mg/m3
Triethylamine	121-44-8	162									2.8
Uranium compounds	7440-61-1	188							1		
Uranium hexafluoride	7783-81-5	188	52 [i]	140 [i]	520 [i]	5	15	30			
Vinyl acetate	108-05-4	165				18	260	1800			
Vinyl chloride	75-01-4	167	640 [p]	3100 [p]	12000 [p]					1.3	180
m-Xylene	108-38-3	171							390		
o-Xylene	95-47-6	170							390		
p-Xylene	106-42-3	172							390		
Xylenes (mixed)	1330-20-7	169	560 [p]	1900 [p]	4000 [p]				390	4.3	22
AEGLs: f = final, I = interim, p = proposed											

Appendix D Methodology for Identifying PB-HAP Compounds

This Appendix provides and justifies a list of hazardous air pollutants that have sufficient persistence and bioaccumulation potential to make them candidates for multipathway risk assessments. The list was selected in two stages.

The first stage was to determine which HAPs are already listed as persistent, bioaccumulative, and toxic (PBT) substances by the following EPA programs:

1. Priority PBT Profiles (Pollution Prevention program): http://www.epa.gov/pbt/cheminfo.htm.

2. Great Waters Pollutants of Concern: http://www.epa.gov/oar/oaqps/gr8water/3rdrpt/execsum.html.

3. Toxics Release Inventory: http://www.epa.gov/tri/chemical/pbt_chem_list.htm.

All substances that are both HAPs pursuant to the CAA and listed by at least one of these programs are shown in Exhibit 1.

The second stage was to determine if, based on their toxicity and bioaccumulation potential, any additional substances should be assessed for multipathway risk by the air toxics program. This determination was made by calculating two indexes for all HAPs for which data could be obtained. One index (intended to estimate relative carcinogenic potential by oral exposure) was the product of the oral carcinogenic potency slope and the bioconcentration factor (obtained from the EPA PBT Profiler, http://www.pbtprofiler.net/). The other index (intended to estimate relative noncarcinogenic hazard by oral exposure) was the ratio of the same bioconcentration factor to the oral reference dose. The cancer and noncancer indexes were normalized to a scale of 1 and combined by averaging (with chemicals with no data not averaged, rather than averaged as zero).

The HAPs were then ranked in descending order of the combined index, and the substances that comprised 99.9999% of the total of all substances were selected as potential candidates for multipathway risk assessment. Results of the ranking exercise are shown in Exhibit 1.

Of the 26 substances that comprised 99.9999% of the aggregate index for all HAPs, 19 are classified as polycyclic organic matter under the Clean Air Act. These were combined into a single category in the table. Metals could not be ranked because the PBT Profiler does not contain data for inorganic pollutants, but were included in the table because of their presence on the other lists. Three other substances shown as “NA” fell outside the 99.9999% aggregate limit.

In summary, no substance not already on at least one existing list emerged in this analysis as a significant potential PBT substance. Therefore, based on our current estimates of toxicity and bioaccumulation potential, the 14 substances in the table represent a conservative list for multipathway risk assessments in the air toxics program.

Exhibit 1. Identity and Ranking of Potential PB-HAP Compounds				
PB-HAP Compound	**OAQPS Rank**	**Pollution Prevention Priority PBTs**	**Great Waters Pollutants of Concern**	**TRI PBT Chemicals**
Cadmium compounds	NA[1]		X	
Chlordane	7	X	X	X
Chlorinated dibenzodioxins and furans	1	X[2]	X	X[3]
DDE	8	X	X	
Heptachlor	4			X
Hexachlorobenzene	6	X	X	X
Hexachlorocyclohexane (all isomers)	NA[4]		X	
Lead compounds	NA[1]	X[5]	X	X
Mercury compounds	NA[1]	X	X	X
Methoxychlor	NA[4]			X
Polychlorinated biphenyls	3	X	X	X
Polycyclic organic matter	2[6]	X[7]	X	X[8]
Toxaphene	5	X	X	X
Trifluralin	NA[4]			X

(1) Not ranked because the PBT Profiler lacks data for inorganic compounds
(2) "Dioxins and furans" (denotes the phraseology of the source list)
(3) "Dioxin and dioxin-like compounds"
(4) Did not fall within 99.9999% of cumulative index
(5) Alkyl lead
(6) 19 POM compounds that fell within the top 26 substances were assigned the rank of 7,12-dimethylbenz(a)anthracene, the highest-ranked compound
(7) Benzo[a]pyrene
(8) "Polycyclic aromatic compounds" and benzo[g,h,i]perylene

Appendix E Overview of Air Toxics Emission Sources

This appendix provides general information on the types of air toxics commonly associated with various types of sources. The table begins with the regulated major source categories and is followed by mobile sources, indoor sources, and miscellaneous sources. This table is not meant to be a comprehensive listing of all chemicals that may be emitted from a given source or group of sources in a particular location.

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Commercial / Industrial Sources				
Halogenated Solvent Cleaners (1614)	methylene chloride; perchloroethylene; trichloroethylene; 1,1,1-trichloroethane; carbon tetrachloride; chloroform[c]	SIC: 33, 34, 36, 37 NAICS: 332, 333, 334, 335, 336, 447	MACT/GACT, see 40 CFR Part 63 Subpart T	U.S. EPA. 1995. *Profile of the Iron and Steel Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., September 1995. EPA/310-R-95-005. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/iron.html
Acetal Resins Production (1301)		SIC: 2869 NAICS: 325199	MACT, see 40 CFR Part 63 YY (General MACT)	U.S. EPA. 1997. *Profile of the Plastic Resins and Man-made Fibers Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., September 1997. EPA/310-R-97-008. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/plastic.html
Acrylic/Modacrylic Fibers Production (1001)		SIC: 2869 NAICS: 325199	MACT, see 40 CFR Part 63 YY	U.S. EPA. 1997. *Profile of the Plastic Resins and Man-made Fibers Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., September 1997. EPA/310-R-97-008. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/plastic.html

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Acrylonitrile-Butadiene-Styrene Production (1302)	styrene; acrylonitrile; butadiene; ethylene glycol; methanol; acetaldehyde; dioxane	SIC: 2821, 2822 NAICS: 325211, 325212	MACT, see 40 CFR Part 63 JJJ	U.S. EPA. 2001. *Polymers and Resins IV Inspection Tool*. Adopt-a-MACT Compliance Tool, Washington, D.C., September 2001. Available at: http://www.epa.gov/ttn/atw/pr4/privinspect.html USEPA. 1997. *Profile of the Plastic Resins and Man-made Fibers Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., September 1997. EPA/310-R-97-008. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/plastic.html
Aerospace Industries (0701)	chromium; cadmium; methylene; chloride; toluene; xylene; methyl ethyl ketone; ethylene glycol; glycol ethers	SIC: 3720, 3721, 3724, 3728, 3760, 3761, 3764, 3769 NAICS: 336411, 336412, 336413, 336414, 336419, 481111, 481112	MACT, see 40 CFR Part 63 Subpart GG	U.S. EPA. 1998. *Profile of the Aerospace Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., November 1998. EPA/310-R-98-001. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/aerospace.html
Amino/Phenolic Resins Production (1347)	formaldehyde, methanol, phenol, xylene, toluene	SIC: 2821 NAICS: 325211	MACT, see 40 CFR Part 63 Subpart OOO	U.S. EPA. 1997. *Profile of the Plastic Resins and Man-made Fibers Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., September 1997. EPA/310-R-97-006. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/plastic.html U.S. EPA. 1998. *Hazardous Air Pollutant Emissions from the Manufacture of Amino and Phenolic Resins: Basis and Purpose Document for Proposed Standards*. Emission Standards Division, Washington, D.C., May 1998. Available at: http://www.epa.gov/ttn/atw/amino/p_r3bpd.wpd

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Ammonium Sulfate - Caprolactam By-Product Plants (1401)	toluene; methanol; xylene; methyl ethyl ketone; ethyl benzene; methyl isobutyl ketone; hydrogen chloride; vinyl acetate	NAICS: 3251, 3252, 3253, 3254, 3255, 3256, 3259	MACT, see 40 CFR Part 63 Subpart FFFF	U.S. EPA. 2002. *Profile of the Organic Chemical Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., November 2002. EPA/310-R-02-001. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/organic.html
Asphalt Roofing and Processing (0418)	formaldehyde; hexane; hydrogen chloride; phenol; polycyclic organic matter; toluene	SIC: 2911, 2952 NAICS: 32411, 324122	MACT, see 40 CFR Part 63 Subpart LLLLL	U.S. EPA. 1995. *Profile of the Stone, Clay, Glass and Concrete Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., September 1995. EPA/310-R-95-017. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/stone.html

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Asphalt/Coal Tar Application - Metal Pipes (0402)	xylenes; toluene; methyl ethyl ketone; phenol; cresols/cresylic acid; glycol ethers (including ethylene glycol monobutyl ether); styrene; methyl isobutyl ketone; ethyl benzene	NAICS: 335312, 336111, 336211, 336312, 33632, 33633, 33634, 33637, 336399, 331316, 331524, 332321, 332323, 33312, 333611, 333618, 332312, 332722, 332813, 332991, 332999, 334119, 336413, 339999, 33612, 336211, 331319, 331422, 335929, 332311, 33242, 81131, 322214, 326199, 331513, 332439, 331111, 331513, 33121, 331221, 331511, 33651, 336611, 482111, 3369, 331316, 336991, 336211, 336112, 336213, 336214, 336399, 326291, 326299, 332311, 332312, 336212, 336999, 33635, 56121, 8111, 56211	MACT, see 40 CFR Part 63 Subpart MMMM	U.S. EPA. 1995. *Profile of the Fabricated Metal Products Industry*. Office of the Compliance Sector Notebook Project, Washington, D.C., September 1995. EPA/310-R-95-007. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/fabric.html U.S. EPA. 1995. *Profile of the Stone, Clay, Glass and Concrete Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., September 1995. EPA/310-R-95-017. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/stone.html

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Auto & Light Duty Truck (Surface Coating) (0702)	toluene; xylene; glycol ethers; methyl ethyl ketone; methyl isobutyl ketone; ethylbenzene; methanol	NAICS: 336111, 336112, 336211	MACT, see 40 CFR Part 63 Subpart IIII	U.S. EPA. 1995. *Profile of the Motor Vehicle Assembly Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., September 1995. EPA/310-R-95-009. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/motor.html U.S. EPA. 2002. *Regulatory Impact Analysis for the Proposed Automobile and Light Duty Truck Coating NESHAP*. Final Report, Washington, D.C., October 2002. EPA-452/R-01-013. Available at: http://www.epa.gov/ttn/atw/auto/autoriap.pdf U.S. EPA. 1997. *U.S. Auto Assembly Plants and Their Communities -- Environmental, Economic, and Demographic Profile*. Common Sense Initiative Automobile Manufacturing Sector. Washington, D.C., December 1997. Available at: http://www.epa.gov/oar/opar/auto/
Boat Manufacturing (1305)	styrene; methyl methacrylate; methylene chloride (dichloromethane); toluene; xylene; n-hexane; methyl ethyl ketone; methyl isobutyl ketone; methyl chloroform (1,1,1-trichloroethane)	SIC: 3731, 3732 NAICS: 336612	MACT, see 40 CFR Part 63 Subpart VVVV	U.S. EPA. 1997. *Profile of the Shipbuilding and Repair Industry*. Office of Compliance Sector Notebook Project. Washington, D.C., November 1997. EPA/310-R-97-008. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/ship.html

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Brick and Structural Clay Products Manufacturing (0414)	hydrogen fluoride; hydrogen chloride; antimony; arsenic; beryllium; cadmium; chromium; cobalt; mercury; manganese; nickel; lead; selenium	SIC: 3251, 3253, 3259 NAICS: 327121, 327122, 327123	MACT, see 40 CFR Part 63 Subpart JJJJJ	U.S. EPA. 1995. *Profile of the Stone, Clay, Glass and Concrete Industry.* Office of Compliance Sector Notebook Project. Washington, D.C., September 1995. EPA/310-R-95-017. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/stone.html
Butyl Rubber Production (1307)	n-hexane; 1,3-butadiene; acrylonitrile; methyl chloride; hydrogen chloride; carbon tetrachloride; chloroprene; toluene	SIC: 2821, 2822 NAICS: 325211, 325212	MACT, see 40 CFR Part 63 Subpart U	U.S. EPA. 1995. *Profile of the Rubber and Plastics Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., September 1995. EPA/310-R-95-016. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/rubber.html
Carbon Black Production (1415)	cyanide compounds; acrylonitrile; acetonitrile; carbonyl sulfide; carbon disulfide; benzene; 1,3 butadiene; toluene; 2,4 toluene diisocyanate	SIC: 2895 NAICS: 325182	General MACT, see 40 CFR Part 63 YY	U.S. EPA. 2002. *Profile of the Organic Chemical Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., November 2002. EPA/310-R-02-001. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/organic.html
Carbonyl Sulfide (COS) Production (1604)	toluene; methanol; xylene; hydrogen chloride; methylene chloride	NAICS: 3251, 3252, 3253, 3254, 3255, 3256, 3259	MACT, see 40 CFR Part 63 FFFF (General MACT)	U.S. EPA. 2002. *Profile of the Organic Chemical Industry, Second Edition (2002).* Office of Compliance Sector Notebook Project, Washington, D.C., November 2002. EPA/310-R-02-001. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/organic.html

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Cellulose Products Manufacturing (1349)	carbon disulfide; carbonyl sulfide; ethylene oxide; methanol; methyl chloride; propylene oxide; toluene	SIC: 2819, 2821, 2823, 2869, 3089 NAICS: 325188, 325199, 325211, 325221, 326121, 326199	MACT, see 40 CFR Part 63 UUUU	U.S. EPA. 2002. Profile of the Pulp and Paper Industry, 2nd Edition. Office of Compliance Sector Notebook Project, Washington, D.C., November 2002. EPA/310-R-02-002. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/pulp.html U.S. EPA. 1997. *Profile of the Plastic Resins and Man-made Fibers Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., September 1997. EPA/310-R-97-008. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/plastic.html
Mercury Cell Chlor-Alkali Plants (Formerly Chlorine Production)		SIC: chlorine 2812	EPA proposes not to regulate chlorine and hydrochloric acid (HCl) emissions for the Chlorine Production source category.	U.S. EPA. 1995. *Profile of the Inorganic Chemical Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., September 1995. EPA/310-R-95-004. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/inorganic.html
Chromic Acid Anodizing (1607)	chromium	NAICS: 332, 333, 334, 335, 336	MACT, see 40 CFR Part 63 Subpart N	U.S. EPA. 1993. *Chromium Emissions from Chromium Electroplating and Chromic Acid Anodizing Operations*. Background Information for Proposed Standards, Washington, D.C., July 1993. EPA 453/R-93-030a and EPA 453/r-93-030b, Volumes 1 and 2. Available at: http://www.epa.gov/ttn/atw/chrome/chromepg.html

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Clay Ceramics Manufacturing (0415)	hydrogen flouride; hydrogen chloride; antimony; arsenic; beryllium; cadmium; chromium; cobalt; mercury; manganese; nickel; lead; selenium	SIC: 3253, 3261 NAICS: 327122, 327111	MACT, see 40 CFR Part 63 Subpart KKKKK	U.S. EPA. 1995. *Profile of the Stone, Clay, Glass and Concrete Industry.* Office of Compliance Sector Notebook Project. Washington, D.C., September 1995. EPA/310-R-95-017. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/stone.html
Coke Ovens: Charging, Top Side, and Door Leaks (0302)	coal tar (benzene, toluene, and xylene); creosote; coal tar pitch; polycyclic aromatic hydrocarbons (benzo(a)pyrene, benzanthracene, chrysene, phenanthrene)	NAICS: 331111, 324199	MACT, see 40 CFR Part 63 Subpart L	U.S. EPA. 1995. *Profile of the Petroleum Refining Industry*. Office of Compliance, Sector Notebook Project. Washington, D.C., September 1995. EPA/310-R-95-013. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/petroleum.html
Coke Ovens: Pushing, Quenching, & Battery Stacks (0303)	polycyclic organic matter; polynuclear aromatic hydrocarbons; benzene; toluene; xylene	NAICS: 331111, 324199	MACT, see 40 CFR Part 63 Subpart CCCCC	U.S. EPA. 1995. *Profile of the Petroleum Refining Industry*. Office of Compliance, Sector Notebook Project. Washington, D.C., September 1995. EPA/310-R-95-013. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/petroleum.html
Commercial Sterilization Facilities (1609)	ethylene oxide	NAICS: 3391	MACT, see 40 CFR Part 63 Subpart O	U.S. EPA. 1997. *Ethylene Oxide Commercial Sterilization and Fumigation Operations.* NESHAP Implementation Document. Washington, D.C., September, 1997. EPA-456/R-004. Available at: http://www.epa.gov/ttn/atw/eo/eoguide.pdf

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Cyanide Chemicals Manufacturing (1405)	cyanide compounds; acrylonitrile; acetonitrile; carbonyl sulfide; carbon disulfide; benzene; 1,3 butadiene; toluene; 2,4 toluene diisocyanate	SIC: 2819, 2869 NAICS: 325188, 325199	MACT, see 40 CFR Part 63 YY (General MACT)	
Decorative Chromium Electroplating (1610)	chromium	NAICS: 332, 333, 334, 335, 336	MACT, see 40 CFR Part 63 Subpart N	
Dry Cleaning: Perchloroethylene (1643)	perchloroethylene	NAICS: 8123	MACT, see 40 CFR Part 63 Subpart M	U.S. EPA. 1995. *Profile of the Dry Cleaning Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., September 1995. EPA/310-R-95-001. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/dry.html

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Engine Test Facilities (0101)	toluene; benzene; mixed xylenes; 1,3-butadiene	SIC: 3511, 3519, 3523, 3524, 3531, 3559, 3566, 3599, 3621, 3711, 3714, 3721, 3724, 3761, 3764, 4226, 4512, 4581, 5541, 7538, 7539, 7699, 8299, 8711, 8731, 8734, 8741, 9661, 9711 NAICS: 54171, 92711, 92811, 332212 333111, 333112, 333120, 333319, 333611, 333612, 333618, 335312, 336111, 336112, 336120, 336312, 336350, 336399, 336411, 336412, 336414, 336415, 336992, 481111, 488190, 541380, 611692, 811111, 811118, 811310, 811411	MACT, see 40 CFR Part 63 Subpart PPPPP	
Epichlorohydrin Elastomers Production (1311)	n-hexane; 1,3-butadiene; acrylonitrile; methyl chloride; hydrogen chloride; carbon tetrachloride; chloroprene; toluene	SIC: 2821, 2822 NAICS: 325211, 325212	MACT, see 40 CFR Part 63 Subpart U	

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Epoxy Resins Production (1312)	epichlorohydrin, methanol, hydrochloric acid	SIC: 2821, 2823, 2824	MACT, see 40 CFR Part 63 Subpart W	U.S. EPA. 1997. *Profile of the Plastic Resins and Man-made Fibers Industry*. Office of Compliance Sector Notebook Project, Washington, DC, September 1997. EPA/310-R-97-008. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/plastic.html
Ethylene Processes (1635)	cyanide compounds; acrylonitrile; acetonitrile; carbonyl sulfide; carbon disulfide; benzene; 1,3 butadiene; toluene; 2,4 toluene diisocyanate	SIC: 2869 NAICS: 325110	MACT, see 40 CFR Part 63 YY	U.S. EPA. 1997. *Profile of the Plastic Resins and Man-made Fibers Industry*. Office of Compliance Sector Notebook Project, Washington, DC. EPA/310-R-97-006. http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/plastic.html
Ethylene-Propylene Rubber Production (1313)	n-hexane; 1,3-butadiene; acrylonitrile; methyl chloride; hydrogen chloride; carbon tetrachloride; chloroprene; toluene	SIC: 2821, 2822 NAICS: 325211, 325212	MACT, see 40 CFR Part 63 Subpart U	U.S. EPA. 1995. *Profile of the Rubber and Plastics Industry*. Office of Compliance Sector Notebook Project, Washington, DC, September 1995. EPA/310-R-95-016. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/rubber.html
Ferroalloys Production (0304)	ferromanganese; silicomanganese; nickel compounds	SIC: 3313	MACT, see 40 CFR Part 63 Subpart XXX	
Flexible Polyurethane Foam Fabrication Operations (1341)	hydrochloric acid; 2,4-toluene diisocyanate; hydrogen cyanide; methylene chloride	SIC: 3086 NAICS: 32615	MACT, see 40 CFR Part 63 Subpart MMMMM	U.S. EPA. 1995. *Profile of the Wood Furniture and Fixtures Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., September 1995. EPA/310-R-95-003. http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/wood.html

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Flexible Polyurethane Foam Production (1314)	methylene chloride; 2,4-toluene diisocyanate; methyl chloroform; methylene diphenyl diisocyanate; propylene oxide; diethanolamine; methyl ethyl ketone; methanol; toluene	SIC: 3086 NAICS: 32615	MACT, see 40 CFR Part 63 Subpart III	U.S. EPA. 1995. Profile of the Wood Furniture and Fixtures Industry. Office of Compliance Sector Notebook Project, Washington, D.C., September 1995. EPA/310-R-95-003. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/wood.html
Friction Products Manufacturing (1636)	n-hexane; toluene; trichloroethylene	NAICS: 33634, 327999, 333613	MACT, see 40 CFR Part 63 QQQQQ	
Fumed Silica Production (1406)	hydrochloric acid; chlorine	SIC: 2819, 2821, 2869 NAICS: 325188, 325211, 325199	MACT, see 40 CFR Part 63 Subpart NNNNN	
Gasoline Distribution (Stage I) (0601)	benzene; toluene; hexane; ethyl benzene; naphthalene; cumene; xylenes; n-hexane; 2, 2, 4-trimethylpentane; methyl tert-butyl ether	SIC: 2911, 4226, 4613, 5171 NAICS: 324110, 493190, 486910, 422710	MACT, see 40 CFR Part 63 Subpart R	U.S. EPA. 1995. *Profile of the Petroleum Refining Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., September 1995. EPA/310-R-95-013. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/petroleum.html.
Hard Chromium Electroplating (1615)	chromium	NAICS: 332, 333, 334, 335, 336	MACT, see 40 CFR Part 63 Subpart N	

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Hazardous Waste Incineration (0801)	chlorinated dioxins and furans; particulate matter (as a surrogate for antimony, cobalt, manganese, nickel, and selenium); carbon monoxide; mercury; lead; cadmium; arsenic; beryllium; chromium; hydrogen chloride and chlorine gas (combined); hydrocarbons		MACT, see 40 CFR Parts 63, 261 and 270	U.S. EPA. *Hazardous Waste Combustion NESHAP Toolkit.* Available at: http://www.epa.gov/epaoswer/hazwaste/combust/toolkit/index.htm
Hydrochloric Acid Production (1407)	hydrochloric acid; chlorine	SIC: 2819, 2821, 2869 NAICS: 325188, 325211, 325199	MACT, see 40 CFR Part 63 Subpart NNNNN	None found at this writing.
Hydrogen Fluoride Production (1409)		SIC: 2819 NAICS: 325188	MACT, see 40 CFR Part 63 YY (General MACT)	U.S. EPA. *Profile of the Plastic Resins and Man-made Fibers Industry.* Office of Compliance Sector Notebook Project, Washington, D.C., September 1997. EPA/310-R-97-006 . Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/plastic.html
Hypalon (TM) Production (1315)	n-hexane; 1,3-butadiene; acrylonitrile; methyl chloride; hydrogen chloride; carbon tetrachloride; chloroprene; toluene	SIC: 2821, 2822 NAICS: 325211, 325212	MACT, see 40 CFR Part 63 Subpart U	None found at this writing.

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Industrial/Commercial/ Institutional Boilers & Process Heaters (0107)	arsenic; cadmium; chromium; hydrogen chloride; hydrogen fluoride; lead; manganese; mercury; nickel	SIC: 13, 24, 26, 28, 29, 30, 33, 34, 37, 49, 80, 82 NAICS: 211, 221, 316, 321, 322, 324, 325, 326, 331, 332, 336, 339, 611, 622	MACT, see 40 CFR Part 63 Subpart DDDDD	U.S. EPA. 1999. *Profile of Oil and Gas Extraction Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., October 2000. EPA/310-R-99-006. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/oil.html. Also see *Profile of Lumber and Wood Products Industry, Profile of Organic and Inorganic Chemical Manufacturing Industry, Profile of Petroleum Refining Industry, and Profile of Rubber and Plastic Industry*. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/index.html
Industrial Cooling Towers (1619)	chromium compounds		MACT, see 40 CFR Part 63 Subpart Q	U.S. EPA. 1995. *Profile of the Petroleum Refining Industry*. Office of Compliance, Sector Notebook Project. Washington, D.C., September 1995. EPA/310-R-95-013. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/petroleum.html U.S. EPA. 2001. *Profile of the Organic Chemical Industry*. Office of Compliance Assistance and Sector Programs Division. Washington, D.C., September 2001. EPA/310/R-02-001. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/organic.html

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Integrated Iron & Steel Manufacturing (0305)	metals (primarily manganese and lead); polycyclic organic matter; benzene; carbon disulfide	SIC: 3312 NAICS: 331111	MACT, see 40 CFR Part 63 Subpart FFFFF	U.S. EPA. 1995. *Profile of the Iron and Steel Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., September 1995. EPA/310-R-95-005. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/iron.html
Iron Foundries (0308)	lead; manganese; cadmium; chromium; nickel; acetophenone; benzene; cumene; dibenzofurans; dioxins; formaldehyde; methanol; naphthalene; phenol; pyrene; toluene; triethylamine; xylene	NAICS: 331511	MACT, see 40 CFR Part 63 Subpart EEEEE	U.S. EPA. 1995. *Profile of the Iron and Steel Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., September 1995. EPA/310-R-95-005. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/iron.html
Large Appliance (Surface Coating) (0704)	glycol ethers; methylene diphenyl diisocyanate; methyl ethyl ketone; toluene; xylene	NAICS: 333312, 333319, 333415, 335221, 335222, 335224, 335228	MACT, see 40 CFR Part 63 Subpart NNNN	U.S. EPA. 1995. *Profile of the Dry Cleaning Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., September 1995. EPA/310-R-95-001. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/dry.html.
Leather Tanning & Finishing Operations (1634)	glycol ethers; toluene; xylene	SIC: 3111 NAICS: 3161	MACT, see 40 CFR Part 63 Subpart TTTT	None found as of this writing

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Light Weight Aggregate Manufacturing (0417)	toluene; methanol; methyl ethyl ketone; xylenes; phenol; methylene chloride; ethylene glycol; glycol ethers; hexane; methyl isobutyl ketone; cresols and cresylic acid; dimethylformamide; vinyl acetate; formaldehyde; ethyl benzene	NAICS: 322211, 322212, 322221, 322222, 322223, 322224, 322225, 322226, 322299, 323111, 323116, 325992, 326111, 326112, 326113, 32613, 326192, 32791, 332999, 339944	See MACT in 40 CFR Part 63 Subpart JJJJ	None found as of this writing
Lime Manufacturing (0408)	hydrogen chloride; antimony; arsenic; beryllium; cadmium; chromium; lead; manganese; mercury; nickel; selenium	NAICS: 32741, 33111, 3314	MACT, see 40 CFR Part 63 Subpart AAAAA	None found at this writing.
Magnetic Tapes (Surface Coating) (0705)	methyl ethyl ketone; toluene; methyl isobutyl ketone; toluene diisocyanate; ethylene glycol; methanol; xylenes; ethyl benzene; acetaldehyde; chromium; cobalt	SIC: 3695, 2675	MACT, see 40 CFR Part 63 Subpart EE	
Manufacture of Nutritional Yeast (1101)	acetaldehyde	SIC: 2099 NAICS: 311999	MACT, see 40 CFR Part 63 Subpart CCCC	

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Marine Vessel Loading Operations (0603)	benzene; toluene; hexane		MACT, see 40 CFR Part 63 Subpart Y	U.S. EPA. 1997. *Profile of the Water Transportation Industry (Shipping and Barging).* Office of Compliance Sector Notebook Project, Washington, D.C., September 1997. EPA/310-R-97-003. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/water.html
Metal Can (Surface Coating) (0707)	ethylene glycol monobutyl ether; other glycol ethers; xylenes; hexane; methyl isobutyl ketone; methyl ethyl ketone	NAICS: 332115, 332116, 332431, 332812, 332999	MACT, see 40 CFR Part 63 Subpart KKKK	U.S. EPA. 1995. *Profile of the Metal Fabrication Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., September 1995. EPA/310-R-95-007. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/fabric.html
Metal Coil (Surface Coating) (0708)	methyl ethyl ketone; glycol ethers; xylenes (isomers and mixtures); toluene; isophorone.	SIC: 34 NAICS: 332812, 331319, 332312, 332322, 332323, 332311, 33637, 332813, 332999, 333293, 336399, 325992, 42183, 323122, 339991, 326113, 32613, 32614, 331112, 331221, 33121, 331312, 331314, 331315	MACT, see 40 CFR Part 63 Subpart SSSS	U.S. EPA. 1995. *Profile of the Metal Fabrication Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., September 1995. EPA/310-R-95-007. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/fabric.html

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Metal Furniture (Surface Coating) (0709)	xylene; toluene; ethylene glycol monobutyl ether; other glycol ethers; ethylbenzene; methyl ethyl ketone	NAICS: 81142, 337124, 337127, 337214, 337215, 339111	MACT, see 40 CFR Part 63 Subpart RRRR	U.S. EPA. 1995. *Profile of the Metal Fabrication Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., September 1995. EPA/310-R-95-007. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/fabric.html
Methyl Methacrylate-Acrylonitrile-Butadiene-Styrene Production (1317)	styrene; acrylonitrile; butadiene; ethylene glycol; methanol; acetaldehyde; dioxane	SIC: 2821, 2822 NAICS: 325211, 325212	MACT, see 40 CFR Part 63 JJJ	U.S. EPA. 2001. *Polymers and Resins IV Inspection Tool*. Adopt-a-MACT Compliance Tool, Washington, D.C., September 2001. Available at: http://www.epa.gov/ttn/atw/pr4/privinspect.html
Methyl Methacrylate-Butadiene-Styrene Terpolymers Production (1318)	styrene; acrylonitrile; butadiene; ethylene glycol; methanol; acetaldehyde; dioxane	SIC: 2821, 2822 NAICS: 325211, 325212	MACT, see 40 CFR Part 63 JJJ	U.S. EPA. 2001. *Polymers and Resins IV Inspection Tool*. Adopt-a-MACT Compliance Tool, Washington, D.C., September 2001. Available at: http://www.epa.gov/ttn/atw/pr4/privinspect.html
Mineral Wool Production (0409)	carbonyl sulfide; nine hazardous metals; formaldehyde; phenol	SIC: 3296	MACT, see 40 CFR Part 63 DDD	
Miscellaneous Coatings Manufacturing (1642)	toluene; xylene; glycol ethers; methyl ethyl ketone, and methyl isobutyl ketone	NAICS: 3255	MACT, see 40 CFR Part 63 Subpart HHHHH	U.S. EPA. 1995. *Profile of the Metal Fabrication Industry*. Office of Compliance Sector Notebook Project. Washington, D.C., September 1995. EPA/310-R-95-007. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/fabric.html

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Miscellaneous Metal Parts & Products (Surface Coating) (0710)	xylene; toluene; methyl ethyl ketone; phenol; cresols/cresylic acid; 2-butoxyethanol; styrene; methyl isobutyl ketone; ethyl benzene; glycol ethers	NAICS: 335312, 336111, 336211, 336312, 33632, 33633, 33634, 33637, 336399, 331316, 331524, 332321, 332323, 33312, 333611, 333618, 332312, 332722, 332813, 332991, 332999, 334119, 336413, 339999, 33612, 336211, 331319, 331422, 335929, 332311, 33242, 81131, 322214, 326199, 331513, 332439, 331111, 331513, 33121, 331221, 331511, 33651, 336611, 482111, 3369, 331316, 336991, 336211, 336112, 336213, 336214, 336399, 326291, 326299, 332311, 332312, 336212, 336999, 33635, 56121, 8111, 56211	MACT, see 40 CFR Part 63 Subpart MMMM	U.S. EPA. 1995. *Profile of the Metal Fabrication Industry*. Office of Compliance Sector Notebook Project. Washington, D.C., September 1995. EPA/310-R-95-007. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/fabric.html

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Miscellaneous Organic Chemical Products & Processes (1641)	toluene; methanol; xylene; methyl ethyl ketone; ethyl benzene; methyl isobutyl ketone; hydrogen chloride; vinyl acetate	NAICS: 3251, 3252, 3253, 3254, 3255, 3256, 3259	MACT, see 40 CFR Part 63 Subpart FFFF	U.S. EPA. 2002. *Profile of the Organic Chemical Industry, 2nd Edition.* Office of Compliance Sector Notebook Project. Washington, DC., November 2002. EPA/310-R-02-001. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/organic.html.
Municipal Landfills (0802)	vinyl chloride; ethyl benzene; toluene; benzene	SIC: 4953, 9511 NAICS: 562212, 924110	MACT, see 40 CFR Part 63 Subpart AAAA	None found as of this writing
Natural Gas Transmission & Storage (0504)	benzene; toluene; ethyl benzene; mixed xylenes; n-hexane	SIC: 40,42,46, 49, 1321 NAICS: 211112 Note: Condensate tank batteries, glycol dehydration units, natural gas processing plants, and natural gas transmission and storage facilities not included.	MACT, see 40 CFR Part 63 Subpart HHH	U.S. EPA. 1997. *Profile of the Ground Transportation Industry - Railroad, Trucking, and Pipeline.* Office of Compliance Sector Notebook Project, Washington, D.C., September 1997. EPA/310-R-97-002. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/ground.html
Neoprene Production (1320)	n-hexane; 1,3-butadiene; acrylonitrile; methyl chloride; hydrogen chloride; carbon tetrachloride; chloroprene; toluene	SIC: 2821, 2822 NAICS: 325211, 325212	MACT, see 40 CFR Part 63 Subpart U	U.S. EPA. 1997. *Profile of Plastic Resins and Man-Made Fibers Industry.* Office of Compliance Sector Notebook Project, Washington, D.C., September 1997. EPA/310-R-97-006. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/plastic.html

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Nitrile Butadiene Rubber Production (1321)	n-hexane; 1,3-butadiene; acrylonitrile; methyl chloride; hydrogen chloride; carbon tetrachloride; chloroprene; toluene	SIC: 2821, 2822 NAICS: 325211, 325212	MACT, see 40 CFR Part 63 Subpart U	U.S. EPA. 1995. *Profile of the Rubber and Plastic Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., September 1995. EPA/310-R-95-016. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/rubber.html
Nitrile Resins Production (1342)	Styrene, n-hexane, 1,3-butadiene, acrylonitrile, methyl chloride, hydrogen chloride, carbon tetrachloride, chloroprene, toluene		MACT, see 40 CFR Part 63 Subpart U	U.S. EPA. 1997. *Profile of Plastic Resins and Man-Made Fibers Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., September 1997. EPA/310-R-97-006. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/plastic.html.
Off-Site Waste and Recovery Operations (0806)	benzene, methylene chloride		MACT, see 40 CFR Part 63 Subpart DD	None found at this writing.
Oil & Natural Gas Production (0501)	benzene; toluene; ethyl benzene; mixed xylenes; n-hexane	SIC: 1311, 1321, 1381, 1382, 1389 NAICS: 211112 (Condensate tank batteries, glycol dehydration units, natural gas processing plants, and natural gas transmission and storage facilities.)	MACT, see 40 CFR Part 63 HH	U.S. EPA. 1999. *Profile of the Oil and Gas Extraction Industry*. Office of Compliance Sector Notebook Project, Washington, D.C. EPA/310-R-99-006. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/oil.html

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Organic Liquids Distribution (Non-Gasoline) (0602)	benzene; ethylbenzene; toluene; vinyl chloride; xylenes	SIC: 2821, 2865, 2869, 2911, 4226, 4612, 5169, 5171 NAICS: 325211, 325192, 325188, 32411, 49311, 49319, 48611, 42269, 42271	MACT, see 40 CFR Part 63 Subpart EEEE	U.S. EPA. *1997. Profile of the Ground Transportation Industry - Railroad, Trucking and Pipeline.* Office of Compliance Sector Notebook Project. Washington, D.C., September 1997. EPA/310-R-97-002. http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/ground.html U.S. EPA. 1995. *Profile of the Petroleum Refining Industry.* Office of Compliance Sector Notebook Project. Washington, D.C., September 1995. EPA/310-R-95-013. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/petroleum.html U.S. EPA. 2002. *Organic Chemical Manufacturing Industry.* Office of Compliance Sector Notebook Project. Washington, D.C., November 2002. EPA/310-R-02-001. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/organic.html

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Paper & Other Webs (Surface Coating) (0711)	toluene; methanol; methyl ethyl ketone; xylenes; phenol; methylene chloride; ethylene glycol; glycol ethers; hexane; methyl isobutyl ketone; cresols and cresylic acid; dimethylformamide; vinyl acetate; formaldehyde; ethyl benzene	NAICS: 322211, 322212, 322221, 322222, 322223, 322224, 322225, 322226, 322299, 323111, 323116, 325992, 326111, 326112, 326113, 32613, 326192, 32791, 332999, 339944	MACT, see 40 CFR Part 63 JJJJ	MACT Sources: Profile of the Pulp and Paper Industry, 2nd Edition (2002). http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/pulp.html
Pesticide Active Ingredient Production (0911)	toluene; methanol; methyl chloride; hydrogen chloride	SIC: 2869, 2879 NAICS: 32532, 325199	MACT, see 40 CFR Part 63 MMM	U.S. EPA. 2000. *Profile of the Agricultural Chemical, Pesticide, and Fertilizer Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., September 2000. EPA/310-R-00-003. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/chemical.html
Petroleum Refineries - Catalytic Cracking, Catalytic Reforming, & Sulfur Plant Units (0502)	hydrogen fluoride; hydrogen chloride; 2,2,4-trimethylpentane; methyl tert butyl ether; benzene; naphthalene; cresols/cresylic acid; phenol; ethylbenzene; toluene; hexane; xylenes; methyl ethyl ketone	SIC: 2911	MACT, see 40 CFR Part 63 Subpart CC	U.S. EPA. 1995. *Profile of the Petroleum Refining Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., September 1995. EPA/310-R-95-013. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/petroleum.html

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Petroleum Refineries - Other Sources Not Distinctly Listed (0503)	benzene, toluene, ethyl benzene, 2,2,4-trimethylpentane, cresols/cresylic acid, ethylbenzene, hexane, methyl ethyl ketone	SIC: 2911	MACT, see 40 CFR Part 63 Subpart CC	U.S. EPA. 1995. *Profile of the Petroleum Refining Industry*. Office of Compliance Sector Notebook Project. Washington, D.C., September 1995. EPA/310-R-95-013. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/petroleum.html.
Pharmaceuticals Production (1201)	methylene chloride; methanol; toluene; hydrogen chloride; dimethylformamide; hexane	SIC: 2833, 2834 NAICS: 32541, 325412	MACT, see 40 CFR Part 63 GGG	U.S. EPA. 1997. *Profile of the Pharmaceutical Industry*. Office of Compliance Sector Notebook Project. Washington, D.C., September 1997. EPA/310-R-97-005. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/pharmaceutical.html
Phosphate Fertilizers Production (1410)	hydrogen fluoride; arsenic; beryllium; cadmium; chromium; manganese; mercury; nickel; methyl isobutyl ketone	SIC: 2874 NAICS: 325314	MACT, see 40 CFR Part 63 BB	U.S. EPA. 2000. *Profile of the Agricultural Chemical, Pesticide, and Fertilizer Industry*. Office of Compliance Sector Notebook Project. Washington, D.C., September 2000. EPA/310-R-00-003. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/chemical.html
Phosphoric Acid Manufacturng (1411)	hydrogen fluoride; arsenic; beryllium; cadmium; chromium; manganese; mercury; nickel; methyl isobutyl ketone	SIC: 2874 NAICS: 325314	MACT, see 40 CFR Part 63 AA	U.S. EPA. 2000. *Profile of the Agricultural Chemical, Pesticide, and Fertilizer Industry*. Office of Compliance Sector Notebook Project. Washington, D.C., September 2000. EPA/310-R-00-003. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/chemical.html

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Plastic Parts & Products (Surface Coating) (0712)	methyl ethyl ketone; methyl isobutyl ketone; toluene; ethylene glycol monobutyl ether; other glycol ethers; xylenes	NAICS: 32615, 32614, 33422, 33992, 326199, 333313, 336211, 336212, 336213, 336214, 336399, 336999, 337214, 339111, 339112, 339999	MACT, see 40 CFR Part 63 Subpart PPPP	U.S. EPA. 1995. *Profile of the Motor Vehicle Assembly Industry*. Office of Compliance Sector Notebook Project. Washington, D.C., September 1995. EPA/310-R-95-009. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/motor.html
Plywood and Composite Wood Products (1624)	acetaldehyde; acrolein; formaldehyde; methanol; phenol; propionaldehyde	SIC: 2421, 2435, 2436, 2439, 2493 NAICS: 321211, 321212, 321213, 321219, 321999	MACT, see 40 CFR Part 63 Subpart DDDD	The Plywood and Composite Wood Products MACT was proposed on January 9, 2003. The comment period ended on March 10, 2003. The final rule will most likely be promulgated in March 2004, with a compliance date of March 2007.
Polybutadiene Rubber Production (1325)	styrene; n-hexane; 1,3- butadiene; acrylonitrile; methyl chloride; hydrogen chloride; carbon tetrachloride; chloroprene; toluene	SIC: 2821, 2822 NAICS: 325211, 325212	MACT, see 40 CFR Part 63 Subpart U	U.S. EPA. 1995. *Profile of the Rubber and Plastic Industry*. Office of Compliance Sector Notebook Project. Washington, D.C., September 1995. EPA/310-R-95-016. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/rubber.html
Polycarbonates Production (1326)	TOC, organic HAPs	SIC: 2869 NAICS: 325199	MACT, see 40 CFR Part 63 YY (Generic MACT)	U.S. EPA. 1997. *Profile of the Plastic Resins and Man-made Fibers Industry*. Office of Compliance Sector Notebook Project. Washington, D.C., September 1997. EPA/310-R-97-006 . Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/plastic.html
Polyether Polyols Production (1625)	ethylene oxide; propylene oxide; hexane; toluene	SIC: 2843, 2869 NAICS: 325199, 325613	MACT, see 40 CFR Part 63 PPP	None found at this writing.

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Polyethylene Terephthalate Production (1328)	styrene; acrylonitrile; butadiene; ethylene glycol; methanol; acetaldehyde; dioxane	SIC: 2821, 2822 NAICS: 325211, 325212	MACT, see 40 CFR Part 63 JJJ	U.S. EPA. 2001. *Polymers and Resins IV Inspection Tool*. Adopt-a-MACT Compliance Tool, Washington, D.C., September 2001. Available at: http://www.epa.gov/ttn/atw/pr4/privinspect.html
Polystyrene Production (1331)	styrene; acrylonitrile; butadiene; ethylene glycol; methanol; acetaldehyde; dioxane	SIC: 2821, 2822 NAICS: 325211, 325212	MACT, see 40 CFR Part 63 JJJ	U.S. EPA. 2001. *Polymers and Resins IV Inspection Tool*. Adopt-a-MACT Compliance Tool, Washington, D.C., September 2001. Available at: http://www.epa.gov/ttn/atw/pr4/privinspect.html
Polysulfide Rubber Production (1332)	styrene; n-hexane; 1,3- butadiene; acrylonitrile; methyl chloride; hydrogen chloride; carbon tetrachloride; chloroprene; toluene	SIC: 30	MACT, see 40 CFR Part 63 Subpart U	U.S. EPA. 1995. *Profile of the Rubber and Plastic Industry*. Office of Compliance Sector Notebook Project. Washington, D.C., September 1995. EPA/310-R-95-016. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/rubber.html
Polyvinyl Chloride & Copolymers Production (1336)	vinyl chloride; vinylidene chloride (1,1 dichloroethylene); vinyl acetate	SIC: 2821 NAICS: 325211	MACT, see 40 CFR Part 63 J	None found as of this writing

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Portland Cement Manufacturing (0410)	acetaldehyde; arsenic; benzene; cadmium; chromium; chlorobenzene; dibenzofurans; formaldehyde; hexane; hydrogen chloride; lead; manganese; mercury; naphthalene; nickel; phenol; polycyclic organic matter; selenium; styrene; 2,3,7,8-tetrachlorodibenzo-p-dioxin; toluene; xylenes	SIC: 3241 NAICS: 32731	MACT, see 40 CFR Part 63 Subpart LLL	U.S. EPA. 1995. *Profile of the Stone, Clay, Glass and Concrete Industry*. Office of Compliance Sector Notebook Project. Washington, D.C., September 1995. EPA/310-R-95-017. http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/stone.html
Primary Aluminum Production (0201)	hydrogen flouride; polycyclic aromatic hydrocarbons	NAICS: 331312	MACT, see 40 CFR Part 63 Subpart LL	U.S. EPA. 1995. *Profile of the Nonferrous Metals Industry*. Office of Compliance Sector Notebook Project. Washington, D.C., September 1995. EPA/310-R-95-010. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/nonferrous.html
Primary Copper Smelting (0203)	antimony; arsenic; beryllium; cadmium; cobalt; lead; manganese; nickel; selenium	SIC: 3339	MACT, see 40 CFR Part 63 Subpart QQQ	None found at this writing.
Primary Lead Smelting (0204)	arsenic; antimony; cadmium	SIC: 3339	MACT, see 40 CFR Part 63 Subpart TTT	None found at this writing.
Primary Magnesium Refining (0207)	chlorine; hydrochloric acid; dioxin/furan; trace amounts of several HAP metals	NAICS: 331419	MACT, see 40 CFR Part 63 Subpart TTTTT	None found at this writing.

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Printing, Coating & Dyeing Of Fabrics (0713)	toluene; methyl ethyl ketone; methanol; xylenes; methyl isobutyl ketone; methylene chloride; n-hexane; trichloroethylene; n,n-dimethyl formamide.; 1,1,1-trichloroethane; naphthalene; ethyl benzene; glycol ethers (ethylene glycol); biphenyl; styrene	NAICS: 31321, 31322, 313241, NAICS: 313311, 313312, 313320, 314110	MACT, see 40 CFR Part 63 Subpart OOOO	U.S. EPA. 1997. *Profile of the Textiles Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., September 1997. EPA/310-R-97-009. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/textiles.html
Printing/Publishing (Surface Coating) (0714)	xylene; toluene; ethylbenzene; methyl ethyl ketone; methyl isobutyl ketone; methanol; ethylene glycol; certain glycol ethers	SIC: 2671, 2711, 2721, 2754, 2759	MACT, see 40 CFR Part 63 Subpart KK	U.S. EPA. 1995. *Profile of the Printing Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., September 1995. EPA/310-R-95-014. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/printing.html
Publicly Owned Treatment Works (POTW) Emissions (0803)	xylenes; methylene chloride; toluene; ethyl benzene; chloroform; tetrachloroethylene; benzene; naphthalene	SIC: 4952 NAICS: 22132	MACT, see 40 CFR Part 63 Subpart VVV	None found at this writing.
Pulp & Paper Production - Combustion (Kraft, Soda, Sulfite, & Semi-Chemical) (1626-2)		SIC: 2611, 2621,2631 NAICS: 32211, 32212, 32213	MACT, see 40 CFR Part 63 Subpart S	U.S. EPA. 2002. *Profile of the Pulp and Paper Industry, 2nd Edition*. Office of Compliance Sector Notebook Project, Washington, D.C., November 2002. EPA/310-R-95-015. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/pulp.html

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Pulp & Paper Production - Non-Combustion (1626-1)		SIC 26	MACT, see 40 CFR Part 63 Subpart S	U.S. EPA. 2002. *Profile of the Pulp and Paper Industry, 2nd Edition*. Office of Compliance Sector Notebook Project, Washington, D.C., November 2002. EPA/310-R-95-015. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/pulp.html
Refractories Products Manufacturing (0406)	ethylene glycol; formaldehyde; hydrogen fluoride; hydrochloric acid; methanol; phenol; polycyclic organic matter	NAICS: 327124, 327125	MACT, see 40 CFR Part 63 Subpart SSSSS	None found at this writing.
Reinforced Plastic Composites Production (1337)	styrene; methyl methacrylate; methylene chloride (dichloromethane)	SIC: 2821, 3084, 3087, 3088, 3089, 3281, 3296, 3431, 3531, 3612, 3613, 3621, 3663, 3711, 3713, 3714, 3716, 3728, 3743, 3792, 3799 NAICS: 33312, 33612, 33651, 33653, 35313, 325211, 325991, 326122, 326191, 327991, 327993, 332998, 333422, 335311, 335312, 336112, 336211, 336213, 336214, 336399, 336413	MACT, see 40 CFR Part 63 Subpart WWWW	None found at this writing.

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Rocket Engine Test Firing (1627)	toluene, benzene, mixed xylenes, 1,3-butadiene	SIC: 3724, 3761, 3764, 9661, 9711 NAICS: 336412, 336414 , 336415, 54171, 92711, 92811	MACT, see 40 CFR Part 63 Subpart PPPPP	U.S. EPA. 1997. *Profile of the Air Transportation Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., February 1998. EPA/310-R-97-001. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/air.html
Rubber Tire Production (1631)	toluene; hexane	SIC: 2296, 3011, 7534 NAICS: 314992, 326211, 326212	MACT, see 40 CFR Part 63 Subpart XXXX	U.S. EPA. *Profile of the Rubber and Plastic Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., September 1995. EPA/310-R-95-016. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/rubber.html
Secondary Aluminum Production (0202)	hydrogen chloride, hydrogen fluoride, chlorine, 2,3,7,8-tetrachlorodibenzo-p-dioxin, organic HAPs, particulate HAP metals	SIC: 3341, 3334, 3353, 3354, 3355, 3363, 3365 NAICS: 331314, 331312, 331315, 331316, 331319, 331521, 331524	MACT, see 40 CFR Part 63 Subpart RRR	None found at this writing.
Secondary Lead Smelting (0205)	lead compounds; arsenic compounds; 1,3-butadiene	NAICS: 331492	MACT, see 40 CFR Part 63 Subpart X	None found at this writing.
Semiconductor Manufacturing (1629)	hydrochloric acid; hydrogen flouride; methanol; glycol ethers; xylene	SIC: 3674 NAICS: 334413	MACT, see 40 CFR Part 63 Subpart BBBBB	U.S. EPA. 2001. *National Emission Standards for Hazardous Air Pollutants: Semiconductor Manufacturing-Background Information for Proposed Standards*. Office of Air Quality Planning and Standards, Research Triangle Park, NC, February 2001. Available at: http://www.epa.gov/ttn/atw/semicon/smatr_bid.pdf

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Shipbuilding & Ship Repair (Surface Coating) (0715)	xylene; toluene; ethylbenzene; methyl ethyl ketone; methyl isobutyl ketone; ethylene glycol; glycol ethers	SIC: 3731	MACT, see 40 CFR Part 63 Subpart II	U.S. EPA. 1997. *Profile of the Shipbuilding and Repair Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., November 1997. EPA/310-R-97-008. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/ship.html
Site Remediation (0805)	benzene; ethyl benzene; toluene; vinyl chloride; xylenes; other volatile organic compounds	NAICS: 325211, 325192, 325188, 32411, 49311, 49319, 48611, 42269, 42271	MACT, see 40 CFR Part 63 Subpart GGGGG	
Solvent Extraction for Vegetable Oil Production (1103)	n-hexane	SIC: 2076, 2079 NAICS: 311223	MACT, see 40 CFR Part 63 Subpart GGGG	None found at this writing.
Spandex Production (1003)	cyanide compounds; acrylonitrile; acetonitrile; carbonyl sulfide; carbon disulfide; benzene; 1,3 butadiene; toluene; 2,4 toluene diisocyanate	SIC: 2824 NAICS: 325222	MACT, see 40 CFR Part 63 YY	U.S. EPA. 1997. *Profile of the Plastic Resins and Man-made Fibers Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., September 1997. EPA/310-R-97-006. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/plastic.html
Stationary Combustion Turbines (0108)	formaldehyde; toluene; benzene; acetaldehyde	SIC: 1311, 1321, 4911, 4922, 4931 NAICS: 221, 2211, 211111, 211112, 486210	MACT, see 40 CFR Part 63 YYYY	
Stationary Reciprocal Internal Combustion Engines (0105)	formaldehyde; acrolein; methanol; acetaldehyde	SIC: 1311, 1321, 4911, 4922, 9711 NAICS: 2211, 48621, 92811, 211111, 211112	MACT, see 40 CFR Part 63 Subpart ZZZZ	

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Steel Pickling - HCL Process (0310)	hydrochloric acid	SIC: 3312, 3315, 3317	MACT, see 40 CFR Part 63 Subpart CCC	
Styrene Acrylonitrile Production (1338)	styrene; acrylonitrile; butadiene; ethylene glycol; methanol; acetaldehyde; dioxane	SIC: 2821, 2822 NAICS: 325211, 325212	MACT, see 40 CFR Part 63 JJJ	U.S. EPA. 2001. *Polymers and Resins IV Inspection Tool*. Adopt-a-MACT Compliance Tool, Washington, D.C., September 2001. Available at: http://www.epa.gov/ttn/atw/pr4/privinspect.html
Styrene-Butadiene Rubber & Latex Production (1339)	styrene; n-hexane; 1,3- butadiene; acrylonitrile; methyl chloride; hydrogen chloride; carbon tetrachloride; chloroprene; toluene	SIC: 2821, 2822 NAICS: 325211, 325212	MACT, see 40 CFR Part 63 Subpart U	U.S. EPA. 1995. *Profile of the Rubber and Plastic Industry*. Office of Compliance Sector Notebook Program, Washington, D.C., September 1995. EPA/310-R-95-016. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/rubber.html
Synthetic Organic Chemical Manufacturing (HON) (1501)	toluene, methanol, xylene, hydrogen chloride, and methylene chloride	NAICS: 3251, 3252, 3253, 3254, 3255, 3256, 3259	MACT, see 40 CFR Part 63 Subpart FFFF and Miscellaneous Organic NESHAP	
Taconite Iron Ore Processing (0411)	metal compounds (such as manganese, arsenic, lead, nickel, chromium, and mercury); products of incomplete combustion (including formaldehyde); hydrogen chloride; hydrogen fluoride	NAICS: 21221	MACT, see 40 CFR Part 63 Subpart RRRRR	None found at this writing.

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Utility Boilers: Coal (1808-1)	arsenic; cadmium; chromium; hydrogen chloride; hydrogen fluoride; lead; manganese; mercury; nickel	SIC: 29 NAICS: 324	See 40 CFR Part 63 Subpart DDDDD	None found at this writing.
Utility Boilers: Natural Gas (1808-2)	arsenic; cadmium; chromium; hydrogen chloride; hydrogen fluoride; lead; manganese; mercury; nickel	SIC: 13, 49 NAICS: 211, 221	See 40 CFR Part 63 Subpart DDDDD	None found at this writing.
Utility Boilers: Oil (1808-3)	arsenic; cadmium; chromium; hydrogen chloride; hydrogen fluoride; lead; manganese; mercury; nickel	SIC: 24, 29 NAICS: 321, 324	See 40 CFR Part 63 Subpart DDDDD	None found at this writing.
Wet-Formed Fiberglass Mat Production (0413)	formaldehyde; methanol; vinyl acetate	SIC: 3229325 NAICS: 327212	MACT, see 40 CFR Part 63 Subpart HHHH	None found at this writing.
Wood Building Products (Surface Coating) (0703)	xylenes; toluene; ethyl benzene; ethylene glycol monobutyl ether; other glycol ethers; methyl ethyl ketone; methyl isobutyl ketone; methanol; styrene; formaldehyde	SIC: 2421, 2426, 2431, 2435, 2436, 2493, 2499 NAICS: 321211, 321212, 321219, 321911, 321918, 321999 Note: The subcategory of the SIC and NAICS code depends on the final end use of the product.	MACT, see 40 CFR Part 63 Subpart QQQQ	U.S. EPA. 1995. *Profile of the Wood Furniture and Fixtures Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., September 1995. EPA/310-R-95-003. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/wood.html

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Wood Furniture (Surface Coating) (0716)	toluene; xylene; methanol; methyl ethyl ketone; methyl isobutyl ketone; glycol ethers; formaldehyde	SIC: 2511, 2512, 2517, 2519, 2521, 2531, 2541	MACT, see 40 CFR Part 63 Subpart JJ	U.S. EPA. 1995. *Profile of the Wood Furniture and Fixtures Industry*. Office of Compliance Sector Notebook Project, Washington, D.C., September 1995. EPA/310-R-95-003. Available at: http://www.epa.gov/compliance/resources/publications/assistance/sectors/notebooks/wood.html
Wool Fiberglass Manufacturing (0412)	arsenic, chromium, lead, formaldehyde, phenol, and methanol	SIC: 3296	MACT, see 40 CFR Part 63 Subpart NNN	None found at this writing.
Mobile Sources				
Mobile sources	acetaldehyde, acrolein, arsenic compounds, benzene, 1,3-butadiene, chromium compounds, diesel particulate matter, diesel exhaust organic gases, dioxin/ furans, ethylbenzene, formaldehyde , n-hexane, lead compounds, manganese compounds, mercury compounds, MTBE, naphthalene, nickel compounds, polycyclic organic matter, styrene, toluene, xylene	N/A	Various, see http://www.epa.gov/otaq/	EPA's Office of Transportation Air Qualtiy provides information on mobile source air toxics at http://www.epa.gov/otaq/toxics.htm In-depth information on desiel engine exhaust can be found at http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=29060&CFID=12048081&CFTOKEN=92457493 The Health Effects Institute is an independent, nonprofit corporation chartered in 1980 to provide high-quality, impartial, and relevant science on the health effects of pollutants from motor vehicles and from other sources in the environment (see www.healtheffects.org).

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Indoor Sources				
Tobacco smoke	Many, including benzene, toluene, formaldehyde, acrolein, N-nitrosdimethyl-amine, polycyclic organic matter, methyl chloride, 1,3-butadiene, phenol, catechol, hydroquinone, aniline, o-toluidine, quinoline, polychlorinated dibenzo-p-dioxins, nickel, cadmium, polonium-210	N/A	Voluntary programs to protect children from the effects of secondhand smoke	U.S. EPA. 1992. *Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders*. Office of Research and Development and Office of Air and Radiation, Washington, D.C., December 1992. EPA/600/6-90/006F. Available at: http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=2835 Smoke-Free Homes Campaign: http://www.epa.gov/smokefree/
Consumer and commercial products	Many organic chemicals and metals, including benzene, toluene, xylenes, aldehydes and ketones, chlorinated solvents, ethylene glycol and glycol ethers, phthalates, pesticides	N/A	Voluntary programs to control exposures/risks	Sources of VOCs indoors: http://www.epa.gov/iaq/voc.html Pesticides: http://www.epa.gov/iaq/pesticid.html

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Building materials	Many, including formaldehyde from pressed wood products; chemicals (see consumer and commercial products) from caulks and sealants, paints and wall coverings, floor coverings, etc.; and asbestos and lead in older buildings	N/A	Voluntary programs to control exposures/risks	Formaldehyde: http://www.epa.gov/iaq/formalde.html Asbestos: http://www.epa.gov/asbestos/ashome.html Lead: http://www.epa.gov/iaq/lead.html
Natural Sources				
Forest fires	Various volatile and semivolatile organic compounds (e.g., dioxins, PAHs)	N/A	No federal programs currently exist	See tables 32-34 in: http://www.epa.gov/ttn/chief/ap42/ch13/related/firerept.pdf. Also see the documentation for the Preliminary 2002 National Emissions Inventory (NEI), pages A58-A70: ftp://ftp.epa.gov/pub/EmisInventory/prelim2002nei/nonpoint/documentation/2002prelimneinonpt_032004.pdf, and and the 1999 final NEI, (pages A56-A60: ftp://ftp.epa.gov/EmisInventory/finalnei99ver3/haps/documentation/nonpoint/nonpt99ver3_aug2003.pdf
Radon	radon	N/A	Voluntary programs to control exposures/risks	Radon in indoor air: http://www.epa.gov/radon

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
Other Sources				
Long-range transport	aldrin, chlordane, DDT, dieldrin, dioxins and furans, endrin, mirex, heptachlor, hexachlorobenzene, mercury, PCBs, toxaphene	N/A	N/A	Information on mercury as a global pollutant can be found on the United Nations Environment Programme website, which also provides in-depth information and assessment of the issue of global mercury (see http://www.chem.unep.ch/mercury/). General information about the health and environmental impacts of persistent organic pollutants (POPs) can be found at http://www.epa.gov/international/toxics/brochure.html. This site describes what actions the United States and some other countries have already taken to address these pollutants, and to describe the actions set into motion by the Stockholm Convention on POPs to address ths issue globally. More in-depth information on global POPs can be found in *The Foundation for Global Action on Persistent Organic Pollutants: a United States Perspective*, Office of Research and Development, U.S. EPA, Research Triangle Park, NC, EPA/600/P-01/003F, 2002 (http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=51746). General reference websites with information on the issue of long range transport are EPA's Great Lakes National Program Office (GLNPO; www.epa.gov/glnpo/), the Binational Toxics Strategy (www.epa.gov/glnpo/bns/), and the Artic Monitoring and Assessment Programme (http://www.amap.no/).

Source Name[a]	Typical Pollutants	Typical Industries (SIC)	Regulatory and Control Programs	References and Other Information
[a] HAP Source Category names are followed by MACT source category codes used for source classification in the National Toxics Inventory (see http://www.epa.gov/ttn/chief/codes/index.html#mact). Except for mobile and natural sources, and sources of indoor air toxics, the table does not include sources of criteria pollutants and TRI chemicals that are not also MACT HAP sources.				
(b) Very limited information is available about emissions and risks associated with the many non-HAP compounds used in solvent cleanings since the MACT rule was promulgated. These compounds are not listed in the table.				
[c]The estimate of air toxics emissions from halogenated solvent cleaning is from background analyses conducted for the MACT rule. The estimate is based on estimates and assumptions about the national number of cleaning machines, the types of cleaning machines and processes in use, control equipment and work practice standards in use before and after the MACT rule, solvents used and solvent use rates, and emissions factors for the various machine types and control equipment combinations. A sample of MACT compliance reports collected from states and EPA regions for a residual risk assessment suggest that (1) the population of cleaning machines estimated for the MACT rule may have been substantially overestimated and/or (2) many cleaning machines have been removed from service or changed to solvents not covered by the MACT.				

Appendix F Specific HAPs Included in the National Emissions Inventory (NEI)

Appendix F. Specific HAPs Included in the NEI

EPAChemRegistryName	NEI HAP Category	NEI Pollutant Name	CASRN
1,1,1-Trichloroethane	Methyl Chloroform (1,1,1-Trichloroethane)	Methyl Chloroform	71-55-6
1,1,2,2-Tetrachloroethane	1,1,2,2-Tetrachloroethane	1,1,2,2-Tetrachloroethane	79-34-5
1,1,2-Trichloroethane	1,1,2-Trichloroethane	1,1,2-Trichloroethane	79-00-5
1,1'-Biphenyl, chloro derivs.	Polychlorinated Biphenyls (Aroclors)	Polychlorinated Biphenyls	1336-36-3
1,1-Dichloroethane	Ethylidene Dichloride (1,1-Dichloroethane)	Ethylidene Dichloride (1,1-Dichloroethane)	75-34-3
1,1-Dichloroethylene	Vinylidene Chloride (1,1-Dichloroethylene)	Vinylidene Chloride	75-35-4
1,1-Dimethylhydrazine	1,1-Dimethylhydrazine	1,1-Dimethyl Hydrazine	57-14-7
1,2,3,4,6,7,8,9-Octachlorodibenzofuran	Dioxins/Furans as 2,3,7,8-TCDD TEQs	Octachlorodibenzofuran	39001-02-0
1,2,3,4,6,7,8,9-Octachlorodibenzo-p-dioxin	Dioxins/Furans as 2,3,7,8-TCDD TEQs	Octachlorodibenzo-p-Dioxin	3268-87-9
1,2,3,4,6,7,8-Heptachlorodibenzofuran	Dioxins/Furans as 2,3,7,8-TCDD TEQs	1,2,3,4,6,7,8-Heptachlorodibenzofuran	67562-39-4
1,2,3,4,6,7,8-Heptachlorodibenzo-p-dioxin	Dioxins/Furans as 2,3,7,8-TCDD TEQs	1,2,3,4,6,7,8-Heptachlorodibenzo-p-Dioxin	35822-46-9
1,2,3,4,7,8,9-Heptachlorodibenzofuran	Dioxins/Furans as 2,3,7,8-TCDD TEQs	1,2,3,4,7,8,9-Heptachlorodibenzofuran	55673-89-7
1,2,3,4,7,8-Hexachlorodibenzofuran	Dioxins/Furans as 2,3,7,8-TCDD TEQs	1,2,3,4,7,8-Hexachlorodibenzofuran	70648-26-9
1,2,3,4,7,8-Hexachlorodibenzo-p-dioxin	Dioxins/Furans as 2,3,7,8-TCDD TEQs	1,2,3,4,7,8-Hexachlorodibenzo-p-Dioxin	39227-28-6
1,2,3,6,7,8-Hexachlorodibenzofuran	Dioxins/Furans as 2,3,7,8-TCDD TEQs	1,2,3,6,7,8-Hexachlorodibenzofuran	57117-44-9
1,2,3,6,7,8-Hexachlorodibenzo-p-dioxin	Dioxins/Furans as 2,3,7,8-TCDD TEQs	1,2,3,6,7,8-Hexachlorodibenzo-p-Dioxin	57653-85-7
1,2,3,7,8,9-Hexachlorodibenzofuran	Dioxins/Furans as 2,3,7,8-TCDD TEQs	1,2,3,7,8,9-Hexachlorodibenzofuran	72918-21-9
1,2,3,7,8,9-Hexachlorodibenzo-p-dioxin	Dioxins/Furans as 2,3,7,8-TCDD TEQs	1,2,3,7,8,9-Hexachlorodibenzo-p-Dioxin	19408-74-3
1,2,3,7,8-Pentachlorodibenzofuran	Dioxins/Furans as 2,3,7,8-TCDD TEQs	1,2,3,7,8-Pentachlorodibenzofuran	57117-41-6
1,2,3,7,8-Pentachlorodibenzo-p-dioxin	Dioxins/Furans as 2,3,7,8-TCDD TEQs	1,2,3,7,8-Pentachlorodibenzo-p-Dioxin	40321-76-4

Appendix F. Specific HAPs Included in the NEI

EPAChemRegistryName	NEI HAP Category	NEI Pollutant Name	CASRN
1,2,4-Trichlorobenzene	1,2,4-Trichlorobenzene	1,2,4-Trichlorobenzene	120-82-1
1,2-Butylene oxide	1,2-Epoxybutane	1,2-Epoxybutane	106-88-7
1,2-Dibromo-3-chloropropane	1,2-Dibromo-3-Chloropropane	1,2-Dibromo-3-Chloropropane	96-12-8
1,2-Dichloroethane	Ethylene Dichloride (1,2-Dichloroethane)	Ethylene Dichloride	107-06-2
1,2-Dichloropropane	Propylene Dichloride (1,2-Dichloropropane)	Propylene Dichloride	78-87-5
1,2-Diphenylhydrazine	1,2-Diphenylhydrazine	1,2-Diphenylhydrazine	122-66-7
1,3-Butadiene	1,3-Butadiene	1,3-Butadiene	106-99-0
1,3-Dichloropropene	1,3-Dichloropropene	1,3-Dichloropropene	542-75-6
1,3-Propane sultone	1,3-Propane Sultone	1,3-Propanesultone	1120-71-4
1,4-Dichlorobenzene	1,4-Dichlorobenzene	1,4-Dichlorobenzene	106-46-7
1,4-Dioxane	p-Dioxane	p-Dioxane	123-91-1
1,6-Dinitropyrene	Polycyclic Organic Matter	1,6-Dinitropyrene	42397-64-8
1,8-Dinitropyrene	Polycyclic Organic Matter	1,8-Dinitropyrene	42397-65-9
12-Methylbenz[a]anthrancene	Polycyclic Organic Matter	12-Methylbenz(a)Anthracene	2422-79-9
1-Methylnaphthalene	Polycyclic Organic Matter	1-Methylnaphthalene	90-12-0
1-Methylphenanthrene	Polycyclic Organic Matter	1-Methylphenanthrene	832-69-9
1-Methylpyrene	Polycyclic Organic Matter	1-Methylpyrene	2381-21-7
1-Nitropyrene	Polycyclic Organic Matter	1-Nitropyrene	5522-43-0
2,2,4-Trimethylpentane	2,2,4-Trimethylpentane	2,2,4-Trimethylpentane	540-84-1
2,3,4,6,7,8-Hexachlorodibenzofuran	Dioxins/Furans as 2,3,7,8-TCDD TEQs	2,3,4,6,7,8-Hexachlorodibenzofuran	60851-34-5
2,3,4,7,8-Pentachlorodibenzofuran	Dioxins/Furans as 2,3,7,8-TCDD TEQs	2,3,4,7,8-Pentachlorodibenzofuran	57117-31-4
2,3,7,8-Tetrachlorodibenzofuran	Dioxins/Furans as 2,3,7,8-TCDD TEQs	2,3,7,8-Tetrachlorodibenzofuran	51207-31-9
2,3,7,8-Tetrachlorodibenzo-p-dioxin	Dioxins/Furans as 2,3,7,8-TCDD TEQs	2,3,7,8-Tetrachlorodibenzo-p-Dioxin	1746-01-6
2,3,7,8-Tetrachlorodibenzo-p-dioxin, TEQ	Dioxins/Furans as 2,3,7,8-TCDD TEQs	2,3,7,8-TCDD TEQ	No CAS Number
2,4,5-Trichlorophenol	2,4,5-Trichlorophenol	2,4,5-Trichlorophenol	95-95-4
2,4,6-Trichlorophenol	2,4,6-Trichlorophenol	2,4,6-Trichlorophenol	88-06-2
2,4-D	2,4-D (2,4-Dichlorophenoxyacetic Acid)(Including Salts And Esters)	2,4-Dichlorophenoxy Acetic Acid	94-75-7
2,4-Dinitrophenol	2,4-Dinitrophenol	2,4-Dinitrophenol	51-28-5

Appendix F. Specific HAPs Included in the NEI

EPAChemRegistryName	NEI HAP Category	NEI Pollutant Name	CASRN
2,4-Dinitrotoluene	2,4-Dinitrotoluene	2,4-Dinitrotoluene	121-14-2
2,4-Toluenediamine	Toluene-2,4-Diamine	Toluene-2,4-Diamine	95-80-7
2-Acetylaminofluorene	2-Acetylaminofluorene	2-Acetylaminofluorene	53-96-3
2-Chloroacetophenone	2-Chloroacetophenone	2-Chloroacetophenone	532-27-4
2-Chloronaphthalene	Polycyclic Organic Matter	2-Chloronaphthalene	91-58-7
2-Ethoxyethanol	Glycol Ethers	Cellosolve Solvent	110-80-5
2-Methoxyethanol	Glycol Ethers	Ethylene Glycol Methyl Ether	109-86-4
2-Methoxyethyl oleate	Glycol Ethers	Methoxyethyl Oleate	111-10-4
2-Methylnaphthalene	Polycyclic Organic Matter	2-Methylnaphthalene	91-57-6
2-Nitrofluorene	Polycyclic Organic Matter	2-Nitrofluorene	607-57-8
2-Nitropropane	2-Nitropropane	2-Nitropropane	79-46-9
2-Propoxyethanol acetate	Glycol Ethers	2-Propoxyethyl Acetate	20706-25-6
3,3'-Dichlorobenzidine	3,3'-Dichlorobenzidene	3,3'-Dichlorobenzidene	91-94-1
3,3'-Dimethoxybenzidine	3,3'-Dimethoxybenzidine	3,3'-Dimethoxybenzidine	119-90-4
3,3'-Dimethylbenzidine	3,3'-Dimethylbenzidine	3,3'-Dimethylbenzidine	119-93-7
3-Butoxy-1-propanol	Glycol Ethers	3-Butoxy-1-Propanol	10215-33-5
3-Methylcholanthrene	Polycyclic Organic Matter	3-Methylcholanthrene	56-49-5
4,4'-Methylenebis(2-chloroaniline)	4,4'-Methylenebis(2-Chloroaniline)	4,4'-Methylenebis(2-Chloraniline)	101-14-4
4,4'-Methylenedi(phenyl isocyanate)	4,4'-Methylenediphenyl Diisocyanate (MDI)	4,4'-Methylenediphenyl Diisocyanate	101-68-8
4,4'-Methylenedianiline	4,4'-Methylenedianiline	4,4'-Methylenedianiline	101-77-9
4,6-Dinitro-o-cresol	4,6-Dinitro-o-Cresol (Including Salts)	4,6-Dinitro-o-Cresol	534-52-1
4-Aminobiphenyl	4-Aminobiphenyl	4-Aminobiphenyl	92-67-1
4-Dimethylaminoazobenzene	4-Dimethylaminoazobenzene	4-Dimethylaminoazobenzene	60-11-7
4-Nitrobiphenyl	4-Nitrobiphenyl	4-Nitrobiphenyl	92-93-3
4-Nitrophenol	4-Nitrophenol	4-Nitrophenol	100-02-7
5-Methylchrysene	Polycyclic Organic Matter	5-Methylchrysene	3697-24-3
6-Nitrochrysene	Polycyclic Organic Matter	6-Nitrochrysene	7496-02-8
7,12-Dimethylbenz[a]anthracene	Polycyclic Organic Matter	7,12-Dimethylbenz[a]Anthracene	57-97-6
9-Methylanthracene	Polycyclic Organic Matter	9-Methylbenz(a)Anthracene	779-02-2
Acenaphthene	Polycyclic Organic Matter as 15-PAH	Acenaphthene	83-32-9
Acenaphthylene	Polycyclic Organic Matter as 15-PAH	Acenaphthylene	208-96-8
Acetaldehyde	Acetaldehyde	Acetaldehyde	75-07-0
Acetamide	Acetamide	Acetamide	60-35-5
Acetonitrile	Acetonitrile	Acetonitrile	75-05-8
Acetophenone	Acetophenone	Acetophenone	98-86-2

Appendix F. Specific HAPs Included in the NEI

EPAChemRegistryName	NEI HAP Category	NEI Pollutant Name	CASRN
Acrolein	Acrolein	Acrolein	107-02-8
Acrylamide	Acrylamide	Acrylamide	79-06-1
Acrylic acid	Acrylic Acid	Acrylic Acid	79-10-7
Acrylonitrile	Acrylonitrile	Acrylonitrile	107-13-1
Alkylated lead	Lead Compounds	Alkylated Lead	No CAS Number
Allyl chloride	Allyl Chloride	Allyl Chloride	107-05-1
Ammonium dichromate (VI)	Chromium Compounds	Ammonium Dichromate	7789-09-5
Aniline	Aniline	Aniline	62-53-3
Anthracene	Polycyclic Organic Matter as 15-PAH	Anthracene	120-12-7
Antimonate(1-), hexafluoro-, sodium, (OC-6-11)-	Antimony Compounds	Sodium hexafluoroantimenate	16925-25-0
Antimony	Antimony Compounds	Antimony	7440-36-0
Antimony and compounds	Antimony Compounds	Antimony & Compounds	No CAS Number
Antimony oxide (unspecified)	Antimony Compounds	Antimony Oxide	1327-33-9
Antimony pentafluoride	Antimony Compounds	Antimony Pentafluoride	7783-70-2
Antimony trichloride	Antimony Compounds	Antimony Trichloride	10025-91-9
Antimony trioxide	Antimony Compounds	Antimony Trioxide	1309-64-4
Antimony trisulfide	Antimony Compounds	Antimony Trisulfide	1345-04-6
Arsenic	Arsenic Compounds(Inorganic Including Arsine)	Arsenic	7440-38-2
Arsenic acid	Arsenic Compounds(Inorganic Including Arsine)	Arsenic Acid	7778-39-4
Arsenic acid (H3AsO4), lead(2+) salt (1:1)	Lead Compounds	Lead Arsenate	7784-40-9
Arsenic compounds (inorganic including arsine)	Arsenic Compounds(Inorganic Including Arsine)	Arsenic & Compounds (Inorganic Including Arsine)	No CAS Number
Arsenic(III) trioxide	Arsenic Compounds(Inorganic Including Arsine)	Arsenic Trioxide	1327-53-3
Arsenic(V) pentoxide	Arsenic Compounds(Inorganic Including Arsine)	Arsenic Pentoxide	1303-28-2
Arsenous acid, triethyl ester	Arsenic Compounds(Inorganic Including Arsine)	Arsenous Acid	3141-12-6
Arsine	Arsenic Compounds(Inorganic Including Arsine)	Arsine	7784-42-1
Asbestos	Asbestos	Asbestos	1332-21-4
Aurate(1-), bis(cyano-.kappa.C)-, potassium	Cyanide Compounds	Gold (I) Potassium Cyanide	13967-50-5
Aurate(1-), bis(cyano-.kappa.C)-, potassium	Cyanide Compounds	Gold Potassium Cyanide	13967-50-5

Appendix F. Specific HAPs Included in the NEI

EPAChemRegistryName	NEI HAP Category	NEI Pollutant Name	CASRN
Aziridine	Ethyleneimine (Aziridine)	Ethyleneimine	151-56-4
Benz[a]anthracene	Polycyclic Organic Matter as 7-PAH	Benz[a]Anthracene	56-55-3
Benz[a]anthracene mixt. with chrysene	Polycyclic Organic Matter as 7-PAH	Benz(a)Anthracene/Chrysene	No CAS Number
Benzene	Benzene (Including Benzene From Gasoline)	Benzene	71-43-2
Benzene soluble organics	Coke Oven Emissions	Benzene Soluble Organics (BSO)	No CAS Number
Benzeneacetonitrile	Cyanide Compounds	Benzyl Cyanide	140-29-4
Benzidine	Benzidine	Benzidine	92-87-5
Benzo(b)fluoranthene	Polycyclic Organic Matter as 7-PAH	Benzo[b]Fluoranthene	205-99-2
Benzo[a]fluoranthene	Polycyclic Organic Matter	Benzo(a)fluoranthene	203-33-8
Benzo[a]pyrene	Polycyclic Organic Matter as 7-PAH	Benzo[a]Pyrene	50-32-8
Benzo[b]fluoranthene mixt. with benzo[k]fluoranthene with benzo[k]fluoranthene	Polycyclic Organic Matter as 7-PAH	Benzo[b+k]Fluoranthene	No CAS Number
Benzo[c]phenanthrene	Polycyclic Organic Matter	Benzo(c)phenanthrene	195-19-7
Benzo[e]pyrene	Polycyclic Organic Matter	Benzo[e]Pyrene	192-97-2
Benzo[ghi]fluoranthene	Polycyclic Organic Matter	Benzo(g,h,i)Fluoranthene	203-12-3
Benzo[ghi]perylene	Polycyclic Organic Matter as 15-PAH	Benzo[g,h,i,]Perylene	191-24-2
Benzo[j]fluoranthene	Polycyclic Organic Matter	B[j]Fluoranthen	205-82-3
Benzo[k]fluoranthene	Polycyclic Organic Matter as 7-PAH	Benzo[k]Fluoranthene	207-08-9
Benzofluoranthene	Polycyclic Organic Matter as 7-PAH	Benzofluoranthenes	56832-73-6
Benzotrichloride	Benzotrichloride	Benzotrichloride	98-07-7
Benzyl chloride	Benzyl Chloride	Benzyl Chloride	100-44-7
Beryllium	Beryllium Compounds	Beryllium	7440-41-7
Beryllium and compounds	Beryllium Compounds	Beryllium & Compounds	No CAS Number
Beryllium difluoride	Beryllium Compounds	Beryllium Fluoride	7787-49-7
Beryllium oxide	Beryllium Compounds	Beryllium Oxide	1304-56-9
beta-Propiolactone	Beta-Propiolactone	Beta-Propiolactone	57-57-8
Biphenyl	Biphenyl	Biphenyl	92-52-4
Bis(2-(2-butoxyethoxy)ethyl) phthalate	Glycol Ethers	Di(Ethylene Glycol Monobutyl Ether) Phthalate	16672-39-2
Bis(2-chloroethyl) ether	Dichloroethyl Ether (Bis[2-Chloroethyl]Ether)	Dichloroethyl Ether	111-44-4
Bis(chloromethyl) ether	Bis(Chloromethyl) Ether	Bis(Chloromethyl)Ether	542-88-1
Borate(1-), tetrafluoro-, lead(2+) (2:1)	Lead Compounds	Lead Fluoroborate	13814-96-5

Appendix F. Specific HAPs Included in the NEI

EPAChemRegistryName	NEI HAP Category	NEI Pollutant Name	CASRN
C.I. Pigment Blue 28	Cobalt Compounds	Cobalt Aluminate	1345-16-0
Cadmium	Cadmium Compounds	Cadmium	7440-43-9
Cadmium and compounds	Cadmium Compounds	Cadmium & Compounds	No CAS Number
Cadmium dichloride	Cadmium Compounds	Cadmium Chloride	10108-64-2
Cadmium iodide	Cadmium Compounds	Cadmium Iodide	7790-80-9
Cadmium nitrate	Cadmium Compounds	Cadmium Nitrate	10325-94-7
Cadmium oxide	Cadmium Compounds	Cadmium Oxide	1306-19-0
Cadmium sulfide	Cadmium Compounds	Cadmium Sulfide	1306-23-6
Captan	Captan	Captan	133-06-2
Carbaryl	Carbaryl	Carbaryl	63-25-2
Carbon disulfide	Carbon Disulfide	Carbon Disulfide	75-15-0
Carbon tetrachloride	Carbon Tetrachloride	Carbon Tetrachloride	56-23-5
Carbonic acid, lead(2+) salt (1:1)	Lead Compounds	Lead Carbonate	598-63-0
Carbonic acid, nickel(2+) salt (1:1)	Nickel Compounds	Nickel Carbonate	3333-67-3
Carbonyl sulfide	Carbonyl Sulfide	Carbonyl Sulfide	463-58-1
Catechol	Catechol	Catechol	120-80-9
Ceramic fibers, man-made	Fine Mineral Fibers	Ceramic Fibers (Man-Made)	No CAS Number
Chloramben	Chloramben	Chloramben	133-90-4
Chlordane	Chlordane	Chlordane	57-74-9
Chlorinated dibenzo-p-dioxins	Dioxins/Furans (total, non TEQ)	Dioxins, Total, w/o Individ. Isomers Reported {PCDDs}	136677-09-3
Chlorinated dibenzo-p-dioxins	Dioxins/Furans (total, non TEQ)	Polychlorinated Dibenzo-p-Dioxins, Total	136677-09-3
Chlorine	Chlorine	Chlorine	7782-50-5
Chloroacetic acid	Chloroacetic Acid	Chloroacetic Acid	79-11-8
Chlorobenzene	Chlorobenzene	Chlorobenzene	108-90-7
Chlorobenzilate	Chlorobenzilate	Chlorobenzilate	510-15-6
Chlorodibenzofurans	Dioxins/Furans (total, non TEQ)	Dibenzofurans (Chlorinated) {PCDFs}	136677-10-6
Chlorodibenzofurans	Dioxins/Furans (total, non TEQ)	Polychlorinated Dibenzofurans, Total	136677-10-6
Chloroethane	Ethyl Chloride	Ethyl Chloride	75-00-3
Chloroform	Chloroform	Chloroform	67-66-3
Chloromethane	Methyl Chloride (Chloromethane)	Methyl Chloride	74-87-3
Chloromethyl methyl ether	Chloromethyl Methyl Ether	Chloromethyl Methyl Ether	107-30-2
Chloroprene	Chloroprene	Chloroprene	126-99-8

Appendix F. Specific HAPs Included in the NEI

EPAChemRegistryName	NEI HAP Category	NEI Pollutant Name	CASRN
Chlorpyrifos	Phosphorus Compounds	Phosphorothioic Acid	2921-88-2
Chromic acid (H2CrO4), barium salt (1:1)	Chromium Compounds	Barium Chromate	10294-40-3
Chromic acid (H2CrO4), calcium salt (1:1)	Chromium Compounds	Calcium Chromate	13765-19-0
Chromic acid (H2CrO4), lead(2+) salt (1:1)	Lead Compounds	Lead Chromate	7758-97-6
Chromic acid (H2CrO4), strontium salt (1:1)	Chromium Compounds	Strontium Chromate	7789-06-2
Chromic acid, mixt. with sulfuric acid	Chromium Compounds	Chromic Sulfuric Acid	No CAS Number
Chromic(VI) acid	Chromium Compounds	Chromic Acid	7738-94-5
Chromic(VI) acid	Chromium Compounds	Chromic Acid (VI)	7738-94-5
Chromium	Chromium Compounds	Chromium	7440-47-3
Chromium and compounds	Chromium Compounds	Chromium & Compounds	No CAS Number
Chromium chloride, hexahydrate	Chromium Compounds	Chromium Chloride	10060-12-5
Chromium difluoride dioxide	Chromium Compounds	Chromyl Fluoride	7788-96-7
Chromium oxide (CrO2)	Chromium Compounds	Chromium Dioxide	12018-01-8
Chromium zinc oxide (Cr2ZnO4)	Chromium Compounds	Chromium Zinc Oxide	12018-19-8
Chromium zinc oxide (unspecified)	Chromium Compounds	Zinc Chromite	50922-29-7
Chromium(III)	Chromium Compounds	Chromium III	16065-83-1
Chromium(III) acetylacetonate	Chromium Compounds	Chromium (III)-AA	21679-31-2
Chromium(III) hydroxide	Chromium Compounds	Chromium Hydroxide	1308-14-1
Chromium(III) oxide	Chromium Compounds	Chromic Oxide	1308-38-9
Chromium(VI)	Chromium Compounds	Chromium (VI)	18540-29-9
Chromium(VI) dioxychloride	Chromium Compounds	Chromyl Chloride	14977-61-8
Chromium(VI) trioxide	Chromium Compounds	Chromium Trioxide	1333-82-0
Chrysene	Polycyclic Organic Matter as 7-PAH	Chrysene	218-01-9
Coal tar	Coke Oven Emissions	Coal Tar	8007-45-2
Cobalt	Cobalt Compounds	Cobalt	7440-48-4
Cobalt and compounds	Cobalt Compounds	Cobalt & Compounds	No CAS Number
Cobalt hydrocarbonyl	Cobalt Compounds	Cobalt Hydrocarbonyl	16842-03-8
Cobalt naphthenate	Cobalt Compounds	Cobalt Naphtha	61789-51-3
Cobalt tetraoxide	Cobalt Compounds	Cobalt Oxide (II,III)	1308-06-1
Cobalt(II) oxide	Cobalt Compounds	Cobalt Oxide	1307-96-6
Cobalt(II) sulfide	Cobalt Compounds	Cobalt Sulfide	1317-42-6
Cobalt, tetracarbonylhydro-	Cobalt Compounds	Cobalt Carbonate	16842-03-8
Coke oven emissions	Coke Oven Emissions	Coke Oven Emissions	No CAS Number
Copper(I) cyanide	Cyanide Compounds	Copper Cyanide	544-92-3
Cresol	Cresol/Cresylic Acid (Mixed Isomers)	Cresol	1319-77-3

Appendix F. Specific HAPs Included in the NEI

EPAChemRegistryName	NEI HAP Category	NEI Pollutant Name	CASRN
Cresol	Cresol/Cresylic Acid (Mixed Isomers)	Cresols (Includes o, m, & p)/Cresylic Acids	1319-77-3
Cumene	Cumene	Cumene	98-82-8
Cyanamide, calcium salt (1:1)	Calcium Cyanamide	Calcium Cyanamide	156-62-7
Cyanide	Cyanide Compounds	Cyanide	57-12-5
Cyanide and compounds	Cyanide Compounds	Cyanide & Compounds	No CAS Number
Cyclonaphthenes	Polycyclic Organic Matter	Naphthenes (Cyclo)	No CAS Number
Di(2-ethylhexyl) phthalate	Bis(2-Ethylhexyl)Phthalate (Dehp)	Bis(2-Ethylhexyl)Phthalate	117-81-7
Diazomethane	Diazomethane	Diazomethane	334-88-3
Dibenz[a,h]anthracene	Polycyclic Organic Matter as 7-PAH	Dibenzo[a,h]Anthracene	53-70-3
Dibenz[a,j]acridine	Polycyclic Organic Matter	Dibenzo[a,j]Acridine	224-42-0
Dibenzo[a,e]pyrene	Polycyclic Organic Matter	Dibenzo[a,e]Pyrene	192-65-4
Dibenzo[a,h]pyrene	Polycyclic Organic Matter	Dibenzo[a,h]Pyrene	189-64-0
Dibenzo[a,i]pyrene	Polycyclic Organic Matter	Dibenzo[a,i]Pyrene	189-55-9
Dibenzo[a,l]pyrene	Polycyclic Organic Matter	Dibenzo[a,l]Pyrene	191-30-0
Dibenzofuran	Dibenzofuran	Dibenzofuran	132-64-9
Dibenzo-p-dioxin	Dioxins/Furans (total, non TEQ)	Dibenzo-p-Dioxin	262-12-4
Dibutyl phthalate	Dibutyl Phthalate	Dibutyl Phthalate	84-74-2
Dichlorvos	Dichlorvos	Dichlorvos	62-73-7
Diethanolamine	Diethanolamine	Diethanolamine	111-42-2
Diethyl sulfate	Diethyl Sulfate	Diethyl Sulfate	64-67-5
Diethylene glycol dibenzoate	Glycol Ethers	Diethylene Glycol Dibenzoate	120-55-8
Diethylene glycol diethyl ether	Glycol Ethers	Diethylene glycol diethyl ether	112-36-7
Diethylene glycol diglycidyl ether	Glycol Ethers	Diethylene Glycol Diglycidyl Ether	4206-61-5
Diethylene glycol dimethyl ether	Glycol Ethers	Diethylene Glycol Dimethyl Ether	111-96-6
Diethylene glycol dinitrate	Glycol Ethers	Diethylene Glycol Dinitrate	693-21-0
Diethylene glycol ethyl methyl ether	Glycol Ethers	Diethylene Glycol Ethyl Methyl Ether	1002-67-1
Diethylene glycol mono-2-cyanoethyl ether	Glycol Ethers	Diethylene Glycol Mono-2-Cyanoethyl Ether	10143-54-1
Diethylene glycol mono-2-methylpentyl ether	Glycol Ethers	Diethyleneglycol-Mono-2-Methyl-Pentyl Ether	10143-56-3
Diethylene glycol monobutyl ether	Glycol Ethers	Diethylene Glycol Monobutyl Ether	112-34-5
Diethylene glycol monobutyl ether acetate	Glycol Ethers	Butyl Carbitol Acetate	124-17-4

Appendix F. Specific HAPs Included in the NEI

EPAChemRegistryName	NEI HAP Category	NEI Pollutant Name	CASRN
Diethylene glycol monoethyl ether	Glycol Ethers	Diethylene Glycol Monoethyl Ether	111-90-0
Diethylene glycol monoethyl ether acetate	Glycol Ethers	Carbitol Acetate	112-15-2
Diethylene glycol monohexyl ether	Glycol Ethers	N-Hexyl Carbitol	112-59-4
Diethylene glycol monoisobutyl ether	Glycol Ethers	Diethylene Glycol Monoisobutyl Ether	18912-80-6
Diethylene glycol monomethyl ether	Glycol Ethers	Diethylene Glycol Monomethyl Ether	111-77-3
Diethylene glycol monovinyl ether	Glycol Ethers	Diethylene Glycol Monovinyl Ether	929-37-3
Dimethyl mercury	Mercury Compounds	Methyl Mercury	593-74-8
Dimethyl phthalate	Dimethyl Phthalate	Dimethyl Phthalate	131-11-3
Dimethyl sulfate	Dimethyl Sulfate	Dimethyl Sulfate	77-78-1
Dimethylcarbamoyl chloride	Dimethylcarbamoyl Chloride	Dimethylcarbamoyl Chloride	79-44-7
Dioxins	Dioxins/Furans (total, non TEQ)	Dioxins	No CAS Number
Epichlorohydrin	Epichlorohydrin (1-Chloro-2,3-Epoxypropane)	1-Chloro-2,3-Epoxypropane	106-89-8
Ethanol, 2-(phenylmethoxy)-	Glycol Ethers	Ethylene Glycol Monobenzyl Ether	622-08-2
Ethene, [2-(2-ethoxyethoxy)ethoxy]-	Glycol Ethers	Diethylene Glycol Ethylvinyl Ether	10143-53-0
Ethene, 1,1'-[oxybis(2,1-ethanediyloxy)]bis-	Glycol Ethers	Diethylene Glycol Divinyl Ether	764-99-8
Ethyl acrylate	Ethyl Acrylate	Ethyl Acrylate	140-88-5
Ethylbenzene	Ethylbenzene	Ethyl Benzene	100-41-4
Ethylene dibromide	Ethylene Dibromide (Dibromoethane)	Ethylene Dibromide	106-93-4
Ethylene glycol	Ethylene Glycol	Ethylene Glycol	107-21-1
Ethylene glycol bis(2,3-epoxy-2-methylpropyl) ether	Glycol Ethers	Ethylene Glycol Bis(2,3-Epoxy-2-Methylpropyl) Ether	3775-85-7
Ethylene glycol diallyl ether	Glycol Ethers	Ethylene Glycol Diallyl Ether	7529-27-3
Ethylene glycol diethyl ether	Glycol Ethers	Ethylene Glycol Diethyl Ether	629-14-1
Ethylene glycol dimethyl ether	Glycol Ethers	1,2-Dimethoxyethane	110-71-4
Ethylene glycol mono-2,6,8-trimethyl-4-nonyl ether	Glycol Ethers	Ethyleneglycolmono-2,6,8-Trimethyl-4-Nonyl Ether	10137-98-1
Ethylene glycol mono-2-methylpentyl ether	Glycol Ethers	Ethyleneglycol Mono-2-Methylpentyl Ether	10137-96-9
Ethylene glycol monobutyl ether	Glycol Ethers	Butyl Cellosolve	111-76-2

Appendix F. Specific HAPs Included in the NEI

EPAChemRegistryName	NEI HAP Category	NEI Pollutant Name	CASRN
Ethylene glycol monobutyl ether acetate	Glycol Ethers	2-Butoxyethyl Acetate	112-07-2
Ethylene glycol monoethyl ether acetate	Glycol Ethers	Cellosolve Acetate	111-15-9
Ethylene glycol monohexyl ether	Glycol Ethers	2-(Hexyloxy)Ethanol	112-25-4
Ethylene glycol monoisobutyl ether	Glycol Ethers	Isobutyl Cellosolve	4439-24-1
Ethylene glycol monomethyl ether acetate	Glycol Ethers	Ethylene Glycol Monomethyl Ether Acetate	110-49-6
Ethylene glycol monomethyl ether acrylate	Glycol Ethers	Methyl Cellosolve Acrylate	3121-61-7
Ethylene glycol monophenyl ether	Glycol Ethers	Phenyl Cellosolve	122-99-6
Ethylene glycol monophenyl ether propionate	Glycol Ethers	Ethyleneglycol Monophenyl Ether Propionate	23495-12-7
Ethylene glycol monopropyl ether	Glycol Ethers	Propyl Cellosolve	2807-30-9
Ethylene glycol mono-sec-butyl ether	Glycol Ethers	Ethylene Glycol Mono-Sec-Butyl Ether	7795-91-7
Ethylene glycol monovinyl ether	Glycol Ethers	Ethylene Glycol Monovinyl Ether	764-48-7
Ethylene oxide	Ethylene Oxide	Ethylene Oxide	75-21-8
Ethylene thiourea	Ethylene Thiourea	Ethylene Thiourea	96-45-7
Ethylenebis(oxyethylenenitrilo)tetraacetic acid	Glycol Ethers	(Ethylenebis(Oxyethylenenitrilo)) Tetraacetic Acid	67-42-5
Extractable organic matter (EOM)	Polycyclic Organic Matter	Extractable Organic Matter (EOM)	No CAS Number
Fine mineral fibers	Fine Mineral Fibers	Fine Mineral Fibers	No CAS Number
Fine mineral fibers	Fine Mineral Fibers	Glasswool (Man-Made Fibers)	No CAS Number
Fine mineral fibers	Fine Mineral Fibers	Slagwool (Man-Made Fibers)	No CAS Number
Fine mineral fibers	Fine Mineral Fibers	Rockwool (Man-Made Fibers)	No CAS Number
Fluoranthene	Polycyclic Organic Matter as 15-PAH	Fluoranthene	206-44-0
Fluorene	Polycyclic Organic Matter as 15-PAH	Fluorene	86-73-7
Formaldehyde	Formaldehyde	Formaldehyde	50-00-0
Glycol ethers -- CAA 112B	Glycol Ethers	Glycol Ethers	No CAS Number
Gold cyanide	Cyanide Compounds	Gold Cyanide	37187-64-7
Heptachlor	Heptachlor	Heptachlor	76-44-8
Heptachlorodibenzofuran	Dioxins/Furans (total, non TEQ)	Total Heptachlorodibenzofuran	38998-75-3
Heptachlorodibenzo-p-dioxin	Dioxins/Furans (total, non TEQ)	Total Heptachlorodibenzo-p-Dioxin	37871-00-4
Hexachlorobenzene	Hexachlorobenzene	Hexachlorobenzene	118-74-1
Hexachlorobutadiene	Hexachlorobutadiene	Hexachlorobutadiene	87-68-3
Hexachlorocyclopentadiene	Hexachlorocyclopentadiene	Hexachlorocyclopentadiene	77-47-4

Appendix F. Specific HAPs Included in the NEI

EPAChemRegistryName	NEI HAP Category	NEI Pollutant Name	CASRN
Hexachlorodibenzofuran	Dioxins/Furans (total, non TEQ)	Total Hexachlorodibenzofuran	55684-94-1
Hexachlorodibenzo-p-dioxin	Dioxins/Furans (total, non TEQ)	Hexachlorodibenzo-p-Dioxin	34465-46-8
Hexachlorodibenzo-p-dioxin	Dioxins/Furans (total, non TEQ)	Hexachlorodibenzo-p-Dioxins, Total	34465-46-8
Hexachloroethane	Hexachloroethane	Hexachloroethane	67-72-1
Hexamethylene-1,6-diisocyanate	Hexamethylene Diisocyanate	Hexamethylene Diisocyanate	822-06-0
Hexamethylphosphoramide	Hexamethylphosphoramide	Hexamethylphosphoramide	680-31-9
Hexane	Hexane	Hexane	110-54-3
Hexanoic acid, 2-ethyl-, cobalt(2+) salt	Cobalt Compounds	Cobalt 2-ethylhexanoate	136-52-7
Hydrazine	Hydrazine	Hydrazine	302-01-2
Hydrochloric acid	Hydrochloric Acid (Hydrogen Chloride [Gas Only])	Hydrochloric Acid	7647-01-0
Hydrofluoric acid	Hydrogen Fluoride (Hydrofluoric Acid)	Hydrogen Fluoride	7664-39-3
Hydrogen cyanide	Cyanide Compounds	Hydrogen Cyanide	74-90-8
Hydroquinone	Hydroquinone	Hydroquinone	123-31-9
Indeno[1,2,3-cd]pyrene	Polycyclic Organic Matter as 7-PAH	Indeno[1,2,3-c,d]Pyrene	193-39-5
Iodine-131	Radionuclides (Including Radon)	Iodine-131	10043-66-0
Isobutyronitrile	Cyanide Compounds	2-Methyl-Propanenitrile	78-82-0
Isophorone	Isophorone	Isophorone	78-59-1
Lead	Lead Compounds	Lead	7439-92-1
Lead acetate	Lead Compounds	Lead Subacetate	1335-32-6
Lead and compounds	Lead Compounds	Lead & Compounds	No CAS Number
Lead and compounds (other than inorganic)	Lead Compounds	Lead Compounds (Other Than Inorganic)	No CAS Number
Lead and compounds, inorganic	Lead Compounds	Lead Compounds (Inorganic)	No CAS Number
Lead arsenite (Pb(AsO2)2)	Lead Compounds	Lead Arsenite	10031-13-7
Lead chromate(VI) oxide	Lead Compounds	Lead Chromate Oxide	18454-12-1
Lead dioxide	Lead Compounds	Lead Dioxide	1309-60-0
Lead dioxide	Lead Compounds	Lead Dioxide, Unknown CAS #	1309-60-0
Lead monoxide	Lead Compounds	Lead (II) Oxide	1317-36-8
Lead naphthenate	Lead Compounds	Lead Naphthenate	61790-14-5
Lead nitrate (Pb(NO3)2)	Lead Compounds	Lead Nitrate	10099-74-8
Lead oxide	Lead Compounds	Lead Oxide	1335-25-7
Lead stearate	Lead Compounds	Lead Stearate	7428-48-0
Lead tetraoxide	Lead Compounds	Lead (II, IV) Oxide	1314-41-6
Lead titanium oxide (PbTiO3)	Lead Compounds	Lead Titanate	12060-00-3

Appendix F. Specific HAPs Included in the NEI

EPAChemRegistryName	NEI HAP Category	NEI Pollutant Name	CASRN
Lead titanium zirconium oxide (Pb(Ti,Zr)O3)	Lead Compounds	Lead Titanate Zircon	12626-81-2
Lead(II) acetate	Lead Compounds	Lead Acetate	301-04-2
Lindane	1,2,3,4,5,6-Hexachlorocyclyhexane (All Stereo Isomers, Including Lindane)	1,2,3,4,5,6-Hexachlorocyclyhexane	58-89-9
Lithium chromate	Chromium Compounds	Lithium Chromate	14307-35-8
Maleic anhydride	Maleic Anhydride	Maleic Anhydride	108-31-6
Manganese	Manganese Compounds	Manganese	7439-96-5
Manganese and compounds	Manganese Compounds	Manganese & Compounds	No CAS Number
Manganese dioxide	Manganese Compounds	Manganese Dioxide	1313-13-9
Manganese naphthenate	Manganese Compounds	Manganese Napthenate	1336-93-2
Manganese tallate	Manganese Compounds	Manganese Tallate	8030-70-4
Manganese tetraoxide	Manganese Compounds	Manganese Tetroxide	1317-35-7
Manganese(II) hypophosphite monohydrate	Manganese Compounds	Manganesehypophosphi	7783-16-6
Manganese(III) oxide	Manganese Compounds	Manganese Trioxide	1317-34-6
m-Cresol	Cresol/Cresylic Acid (Mixed Isomers)	m-Cresol	108-39-4
Mercuric chloride	Mercury Compounds	Mercuric Chloride	7487-94-7
Mercury	Mercury Compounds	Elemental Gaseous Mercury	7439-97-6
Mercury	Mercury Compounds	Mercury	7439-97-6
Mercury and compounds	Mercury Compounds	Mercury & Compounds	No CAS Number
Mercury, divalent	Mercury Compounds	Gaseous Divalent Mercury	14302-87-5
Mercury, divalent	Mercury Compounds	Particulate Divalent Mercury	14302-87-5
Methanol	Methanol	Methanol	67-56-1
Methoxychlor	Methoxychlor	Methoxychlor	72-43-5
Methyl bromide	Methyl Bromide (Bromomethane)	Methyl Bromide	74-83-9
Methyl cellosolve acetyl ricinoleate	Glycol Ethers	Methyl Cellosolve Acetylricinoleate	140-05-6
Methyl ethyl ketone	Methyl Ethyl Ketone (2-Butanone)	Methyl Ethyl Ketone	78-93-3
Methyl hydrazine	Methylhydrazine	Methylhydrazine	60-34-4
Methyl iodide	Methyl Iodide (Iodomethane)	Methyl Iodide	74-88-4
Methyl isobutyl ketone	Methyl Isobutyl Ketone (Hexone)	Methyl Isobutyl Ketone	108-10-1
Methyl isocyanate	Methyl Isocyanate	Methyl Isocyanate	624-83-9
Methyl methacrylate	Methyl Methacrylate	Methyl Methacrylate	80-62-6
Methyl tert-butyl ether	Methyl Tert-Butyl Ether	Methyl Tert-Butyl Ether	1634-04-4
Methylanthracene	Polycyclic Organic Matter	Methylanthracene	26914-18-1
Methylbenzopyrene	Polycyclic Organic Matter	Methylbenzopyrenes	65357-69-9

Appendix F. Specific HAPs Included in the NEI

EPAChemRegistryName	NEI HAP Category	NEI Pollutant Name	CASRN
Methylchrysene	Polycyclic Organic Matter	Methylchrysene	41637-90-5
Methylene chloride	Methylene Chloride (Dichloromethane)	Methylene Chloride	75-09-2
Methylene chloride soluble organics	Coke Oven Emissions	Methylene Chloride Soluble Organics (MCSO)	No CAS Number
Methylmercury	Mercury Compounds	Mercury (Organic)	22967-92-6
m-Xylene	Xylenes (Mixed Isomers)	m-Xylene	108-38-3
N,N-Dimethylaniline	N,N-Dimethylaniline	N,N-Dimethylaniline	121-69-7
N,N-Dimethylformamide	N,N-Dimethylformamide	N,N-Dimethylformamide	68-12-2
Naphthalene	Naphthalene	Naphthalene	91-20-3
Neodecanoic acid, lead salt	Lead Compounds	Lead Neodecanoate	27253-28-7
Nickel	Nickel Compounds	Nickel	7440-02-0
Nickel and compounds	Nickel Compounds	Nickel & Compounds	No CAS Number
Nickel carbide	Nickel Compounds	Nickel Carbide	12710-36-0
Nickel carbonyl	Nickel Compounds	Nickel Carbonyl	13463-39-3
Nickel diacetate tetrahydrate	Nickel Compounds	Nickel Diacetate TET	6018-89-9
Nickel hydroxide (Ni(OH)2)	Nickel Compounds	Nickel Hydroxide	12054-48-7
Nickel refinery dust	Nickel Compounds	Nickel Refinery Dust	No CAS Number
Nickel subsulfide	Nickel Compounds	Nickel Subsulfide	12035-72-2
Nickel(II) acetate	Nickel Compounds	Nickel Acetate	373-02-4
Nickel(II) bromide	Nickel Compounds	Nickel Bromide	13462-88-9
Nickel(II) chloride	Nickel Compounds	Nickel Chloride	7718-54-9
Nickel(II) nitrate	Nickel Compounds	Nickel Nitrate	13138-45-9
Nickel(II) oxide	Nickel Compounds	Nickel Oxide	1313-99-1
Nickel(III) oxide	Nickel Compounds	Nickel Peroxide	1314-06-3
Nickel-59	Nickel Compounds	Nickel (NI 059)	14336-70-0
Nickelate(2-), tetrakis(cyano-.kappa.C)-, dipotassium, (SP-4-1)-	Cyanide Compounds	Potass Nickel Cyanid	14220-17-8
Nickelocene	Nickel Compounds	Nickelocene	1271-28-9
Nitric acid, manganese(2+) salt	Manganese Compounds	Manganese Nitrate	10377-66-9
Nitrobenzene	Nitrobenzene	Nitrobenzene	98-95-3
N-Nitrosodimethylamine	N-Nitrosodimethylamine	N-Nitrosodimethylamine	62-75-9
N-Nitrosomorpholine	N-Nitrosomorpholine	N-Nitrosomorpholine	59-89-2
N-Nitroso-N-methylurea	N-Nitroso-N-Methylurea	N-Nitroso-N-Methylurea	684-93-5
o-Anisidine	o-Anisidine	o-Anisidine	90-04-0
o-Cresol	Cresol/Cresylic Acid (Mixed Isomers)	o-Cresol	95-48-7

Appendix F. Specific HAPs Included in the NEI

EPAChemRegistryName	NEI HAP Category	NEI Pollutant Name	CASRN
o-Toluidine	o-Toluidine	o-Toluidine	95-53-4
o-Xylene	Xylenes (Mixed Isomers)	o-Xylene	95-47-6
p,p'-DDE	Dde (1,1-Dichloro-2,2-Bis(p-Chlorophenyl) Ethylene)	Dde (1,1-Dichloro-2,2-Bis(p-Chlorophenyl) Ethylene)	72-55-9
Parathion	Parathion	Parathion	56-38-2
p-Cresol	Cresol/Cresylic Acid (Mixed Isomers)	p-Cresol	106-44-5
Pentachlorodibenzofuran	Dioxins/Furans (total, non TEQ)	Total Pentachlorodibenzofuran	30402-15-4
Pentachlorodibenzo-p-dioxin	Dioxins/Furans (total, non TEQ)	Total Pentachlorodibenzo-p-Dioxin	36088-22-9
Pentachloronitrobenzene	Pentachloronitrobenzene (Quintobenzene)	Pentachloronitrobenzene	82-68-8
Pentachlorophenol	Pentachlorophenol	Pentachlorophenol	87-86-5
Perylene	Polycyclic Organic Matter	Perylene	198-55-0
Phenanthrene	Polycyclic Organic Matter as 15-PAH	Phenanthrene	85-01-8
Phenol	Phenol	Phenol	108-95-2
Phenylmercury acetate	Mercury Compounds	Mercury Acetato Phen	62-38-4
Phosgene	Phosgene	Phosgene	75-44-5
Phosphine	Phosphine	Phosphine	7803-51-2
Phosphoric acid	Phosphorus Compounds	Phosphoric Acid	7664-38-2
Phosphoric acid, lead(2+) salt (2:3)	Lead Compounds	Lead Phosphate	7446-27-7
Phosphoric acid, monoammonium monosodium salt	Phosphorus Compounds	Phosphorous Salt	13011-54-6
Phosphoric acid, reaction products with aluminum hydroxide and chromium oxide (CrO3)	Phosphorus Compounds	Phosphoric Acid,Rx P	92203-02-6
Phosphoric acid, zinc salt (2:3)	Phosphorus Compounds	Zinc Phosphate	7779-90-0
Phosphorous acid	Phosphorus Compounds	Phosphorous Acid	10294-56-1
Phosphorus	Phosphorus Compounds	Phosphorus	7723-14-0
Phosphorus and compounds	Phosphorus Compounds	Phosphorus & Compounds	No CAS Number
Phosphorus nitride (P3N5)	Phosphorus Compounds	Phosphorous Nitride	12136-91-3
Phosphorus oxychloride	Phosphorus Compounds	Phosphorus Oxychloride	10025-87-3
Phosphorus pentasulfide	Phosphorus Compounds	Phosphorus Pentasulfide	1314-80-3
Phosphorus pentoxide	Phosphorus Compounds	Phosphorus Pentoxide	1314-56-3
Phosphorus trichloride	Phosphorus Compounds	Phosphorus Trichloride	7719-12-2
Phosphorus trioxide	Phosphorus Compounds	Phosphorus Trioxide	1314-24-5
Phthalic anhydride	Phthalic Anhydride	Phthalic Anhydride	85-44-9

Appendix F. Specific HAPs Included in the NEI

EPAChemRegistryName	NEI HAP Category	NEI Pollutant Name	CASRN
Polycyclic aromatic hydrocarbons	Polycyclic Organic Matter as 7-PAH	PAH, Total	130498-29-2
Polycyclic aromatic hydrocarbons - 16-PAH	Polycyclic Organic Matter	16-PAH	No CAS Number
Polycyclic aromatic hydrocarbons - 7-PAH	Polycyclic Organic Matter as 7-PAH	7-PAH	No CAS Number
Polycyclic organic matter - including 15-PAH	Polycyclic Organic Matter as 7-PAH	Polycyclic Organic Matter	No CAS Number
Potassium chromate (VI)	Chromium Compounds	Potassium Chromate	7789-00-6
Potassium cyanide	Cyanide Compounds	Potassium Cyanide	151-50-8
Potassium dichromate	Chromium Compounds	Potassium Dichromate	7778-50-9
Potassium ferrocyanide	Cyanide Compounds	Potassium Ferrocyani	13943-58-3
Potassium permanganate	Manganese Compounds	Potassium permanganate	7722-64-7
Potassium zinc chromate hydroxide (KZn2(CrO4)2(OH))	Chromium Compounds	Zinc Potassium Chromate	11103-86-9
p-Phenylenediamine	p-Phenylenediamine	p-Phenylenediamine	106-50-3
Propionaldehyde	Propionaldehyde	Propionaldehyde	123-38-6
Propoxur	Propoxur (Baygon)	Propoxur	114-26-1
Propylene glycol monoisobutyl ether	Glycol Ethers	1-Isobutoxy-2-Propanol	23436-19-3
Propylene oxide	Propylene Oxide	Propylene Oxide	75-56-9
Propyleneimine	1,2-Propylenimine (2-Methylaziridine)	1,2-Propylenimine	75-55-8
p-Xylene	Xylenes (Mixed Isomers)	p-Xylene	106-42-3
Pyrene	Polycyclic Organic Matter as 15-PAH	Pyrene	129-00-0
Quinoline	Quinoline	Quinoline	91-22-5
Quinone	Quinone (p-Benzoquinone)	Quinone	106-51-4
Radionuclides (including radon)	Radionuclides (Including Radon)	Radionuclides (Including Radon)	No CAS Number
Radionuclides (including radon)	Radionuclides (Including Radon)	Radionuclides	No CAS Number
Radon and its decay products	Radionuclides (Including Radon)	Radon And Its Decay Products	No CAS Number
Selenious acid (H2SeO3)	Selenium Compounds	Selenous Acid	7783-00-8
Selenium	Selenium Compounds	Selenium	7782-49-2
Selenium and compounds	Selenium Compounds	Selenium & Compounds	No CAS Number
Selenium dioxide	Selenium Compounds	Selenium Dioxide	7446-08-4
Selenium disulfide	Selenium Compounds	Selenium Disulfide	7488-56-4
Selenium hexafluoride	Selenium Compounds	Selenium Hexafluoride	7783-79-1
Selenium monosulfide	Selenium Compounds	Selenium Monosulfide	7446-34-6
Selenium oxide	Selenium Compounds	Selenium Oxide	12640-89-0
Silver cyanide	Cyanide Compounds	Silver Cyanide	506-64-9
Sodium chromate (VI)	Chromium Compounds	Sodium Chromate	7775-11-3
Sodium chromate(VI), tetrahydrate	Chromium Compounds	Sodium Chromate(VI)	10034-82-9
Sodium cyanide	Cyanide Compounds	Sodium Cyanide	143-33-9

Appendix F. Specific HAPs Included in the NEI

EPAChemRegistryName	NEI HAP Category	NEI Pollutant Name	CASRN
Sodium dichromate	Chromium Compounds	Sodium Dichromate	10588-01-9
Sodium permanganate	Manganese Compounds	Permanganic acid	10101-50-5
Styrene	Styrene	Styrene	100-42-5
Styrene oxide	Styrene Oxide	Styrene Oxide	96-09-3
Sulfamic acid, nickel(2+) salt (2:1)	Nickel Compounds	Nickel Sulfamate	13770-89-3
Sulfuric acid, beryllium salt (1:1)	Beryllium Compounds	Beryllium Sulfate	13510-49-1
Sulfuric acid, cadmium salt (1:1)	Cadmium Compounds	Cadmium Sulfate	10124-36-4
Sulfuric acid, chromium(3+) salt (3:2)	Chromium Compounds	Chromic Sulfate	10101-53-8
Sulfuric acid, cobalt(2+) salt (1:1)	Cobalt Compounds	Cobalt Sulfate	10124-43-3
Sulfuric acid, lead(2+) salt (1:1)	Lead Compounds	Lead Sulfate	7446-14-2
Sulfuric acid, manganese(2+) salt (1:1)	Manganese Compounds	Manganese Sulfate	7785-87-7
Sulfuric acid, nickel(2+) salt (1:1)	Nickel Compounds	Nickel Sulfate	7786-81-4
Sulfuric acid, nickel(2+) salt (1:1), hexahydrate	Nickel Compounds	Nickel (II) Sulfate Hexahydrate	10101-97-0
Tetrachlorodibenzofuran	Dioxins/Furans (total, non TEQ)	Total Tetrachlorodibenzofuran	30402-14-3
Tetrachlorodibenzo-p-dioxin	Dioxins/Furans (total, non TEQ)	Total Tetrachlorodibenzo-p-Dioxin	41903-57-5
Tetrachloroethylene	Tetrachloroethylene (Perchloroethylene)	Tetrachloroethylene	127-18-4
Tetraethyl lead	Lead Compounds	Tetraethyl Lead	78-00-2
Titanium tetrachloride	Titanium Tetrachloride	Titanium Tetrachloride	7550-45-0
Toluene	Toluene	Toluene	108-88-3
Toluene-2,4-diisocyanate	2,4-Toluene Diisocyanate	2,4-Toluene Diisocyanate	584-84-9
Toxaphene	Toxaphene (Chlorinated Camphene)	Toxaphene	8001-35-2
Tribromomethane	Bromoform	Bromoform	75-25-2
Trichloroethylene	Trichloroethylene	Trichloroethylene	79-01-6
Triethylamine	Triethylamine	Triethylamine	121-44-8
Triethylene glycol	Glycol Ethers	Triethylene glycol	112-27-6
Triethylene glycol dimethyl ether	Glycol Ethers	Triethylene Glycol Dimethyl Ether	112-49-2
Triethylene glycol monobutyl ether	Glycol Ethers	Triglycol Monobutyl Ether	143-22-6
Triethylene glycol monoethyl ether	Glycol Ethers	Ethoxytriglycol	112-50-5
Triethylene glycol monomethyl ether	Glycol Ethers	Methoxytriglycol	112-35-6
Trifluralin	Trifluralin	Trifluralin	1582-09-8
Trimethylene glycol monomethyl ether	Glycol Ethers	3-Methoxy-1-Propanol	1589-49-7
Tri-o-cresyl phosphate	Phosphorus Compounds	Triorthocresyl Phosphate	78-30-8
Triphenyl phosphate	Phosphorus Compounds	Triphenyl Phosphate	115-86-6

Appendix F. Specific HAPs Included in the NEI

EPAChemRegistryName	NEI HAP Category	NEI Pollutant Name	CASRN
Triphenyl phosphite	Phosphorus Compounds	Triphenyl Phosphite	101-02-0
Uranium-238	Radionuclides (Including Radon)	Uranium	7440-61-1
Urethane	Ethyl Carbamate (Urethane) Chloride (Chloroethane)	Ethyl Carbamate Chloride	51-79-6
Vinyl acetate	Vinyl Acetate	Vinyl Acetate	108-05-4
Vinyl bromide	Vinyl Bromide	Vinyl Bromide	593-60-2
Vinyl chloride	Vinyl Chloride	Vinyl Chloride	75-01-4
Xylene	Xylenes (Mixed Isomers)	Xylenes (Mixture of o, m, and p Isomers)	1330-20-7
Zinc chromate	Chromium Compounds	Zinc Chromate	13530-65-9
Zinc chromate	Chromium Compounds	Zinc Chromate	13530-65-9
Zinc cyanide	Cyanide Compounds	Zinc Cyanide	557-21-1

Field Definitions:

- "**EPAChemRegistryName**" (EPA Chemical Registry Name) - the name EPA has selected as the name to be commonly used by EPA in referring to a chemical substance
- "**NEI HAP Category**" - Grouping of related NEI pollutants
- "**NEI Pollutant Name**" - HAP name for NEI pollutant
- "**CASRN**" (Chemical Abstracts Service Registry Number)- the unique number assigned by Chemical Abstracts Service (CAS) to a chemical substance

Table from:
EPA. 2003. *1999 NEI Final Version 3 for Hazardous Air Pollutants Point, non point, and mobile sources. Documentation for the 1999 NEI Final Version 3 for Hazardous Air Pollutants. HAPs list with chemical ID standard fields - August 2003* OAQPS. Available at **http://www.epa.gov/ttn/chief/net/1999inventory.html#final3haps**

Field Definitions from:
1999 NEI Final Version 3 for Hazardous Air Pollutants Point, non point, and mobile sources (September 2003). Documentation for the 1999 NEI Final Version 3 for Hazardous Air Pollutants.Readme file for HAPs list with chemical ID standard fields - August 2003. Available at **http://www.epa.gov/ttn/chief/net/1999inventory.html#final3haps**

Appendix G Atmospheric and Meteorological Concepts Relevant to Dispersal, Transport, and Fate of Air Toxics

Table of Contents

1.0 Structure and Composition of the Atmosphere 1

2.0 Atmospheric Energy 1
- 2.1 Solar Radiation and Differential Heating 1
- 2.2 Effects of Topography 3

3.0 Atmospheric Motions 4
- 3.1 Horizontal Air Motions 4
- 3.2 Vertical Air Motions 5

4.0 Meteorological Data 8
- 4.1 Wind Speed and Direction 8
- 4.2 Other Important Meteorological Data 9
- 4.3 Sources of Meteorological Data 11

References 12

This Appendix defines and discusses atmospheric and meteorological concepts relevant to modeling dispersion, transport, and fate of air toxics. In addition, this appendix provides information on sources of meteorological data that can be used for air toxics modeling. Much of this information was obtained from EPA's primer on air pollution meteorology (see http://www.epa.gov/oar/oaqps/eog/catalog/si409.html). Basic textbooks on meteorology provide more detailed discussions of the material summarized in this Appendix.

1.0 Structure and Composition of the Atmosphere

The atmosphere consists of mixture of about 78 percent nitrogen, 21 percent oxygen and one percent argon up to about 90 km. Within this region trace gases include carbon dioxide, neon, helium, and water vapor. Although the water vapor content of the air is fairly small it is highly variable. Water vapor absorbs six times more radiation energy than any other atmospheric constituent and is therefore a very important component of the atmosphere. Similarly, carbon dioxide is highly variable and is important gas because it absorbs and re-radiates back some of the infrared radiation emitted by the earth.

The atmosphere has been divided into four regions (Exhibit 1) based on temperature changes with height: the troposphere, stratosphere, mesosphere, and ionosphere. The troposphere accounts for about three quarters of the mass of the atmosphere and contains nearly all of the water in the atmosphere (in the forms of vapor, clouds, and precipitation). The depth of the troposphere is on average about 16.5 km (54,000 ft) over the equator and about 8.5 km (28,000 ft) over the poles. The troposphere also tends to be thicker in summer (when the air is warmer) than in the winter. The depth of the troposphere changes constantly due to changes in atmospheric temperature. The troposphere is the most important layer of the atmosphere with respect to air toxics, because this is the region in which most of the air toxics are released. Of the other regions of the atmosphere only the stratosphere has a direct role for some air toxics. Some air toxic emissions can be circulated into the lower stratosphere via weather system or directly emitted from aircraft or volcanic eruption. Once air toxics reach the stratosphere they maybe transported very long distances.

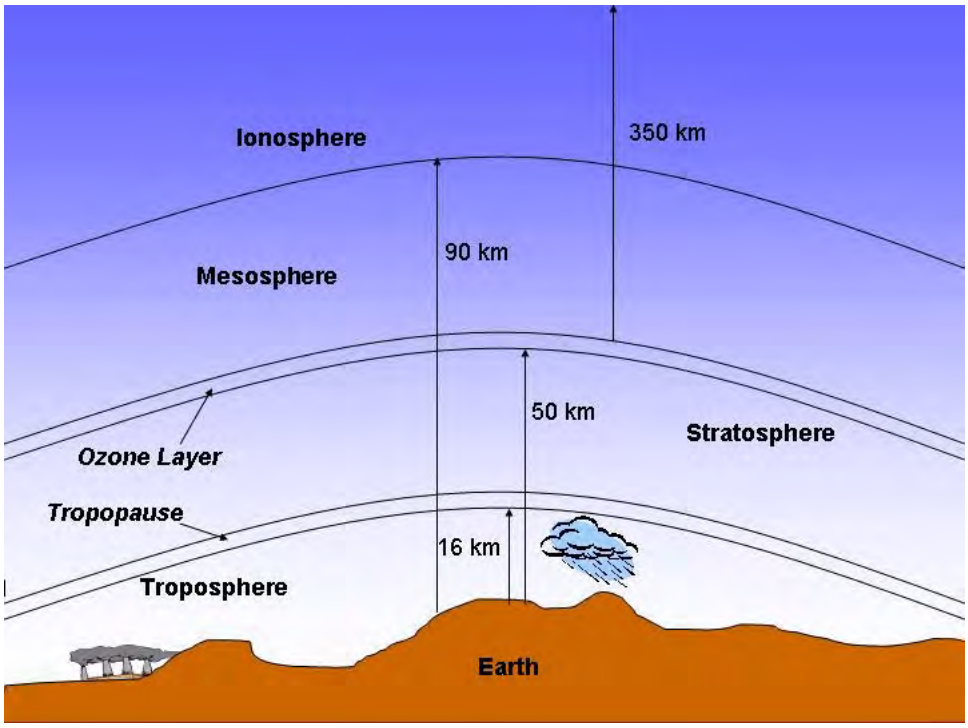
2.0 Atmospheric Energy

The troposphere is the most variable layer of the atmosphere and is the layer where weather occurs. It is where air masses, weather fronts, and storms reside. Weather conditions are governed by a number of factors, including solar radiation, atmospheric circulation, water vapor and topography. However, the underlying driving force in all cases is the radiant energy from the sun.

2.1 Solar Radiation and Differential Heating

The amount of incident sunlight influences the heating of the surface of the earth and the overlying atmosphere. The radiation received directly from the sun is called **solar radiation**. The amount of incoming solar radiation received at a particular time and location (insolation) on the earth is governed by:

Exhibit 1. Structure of the Atmosphere



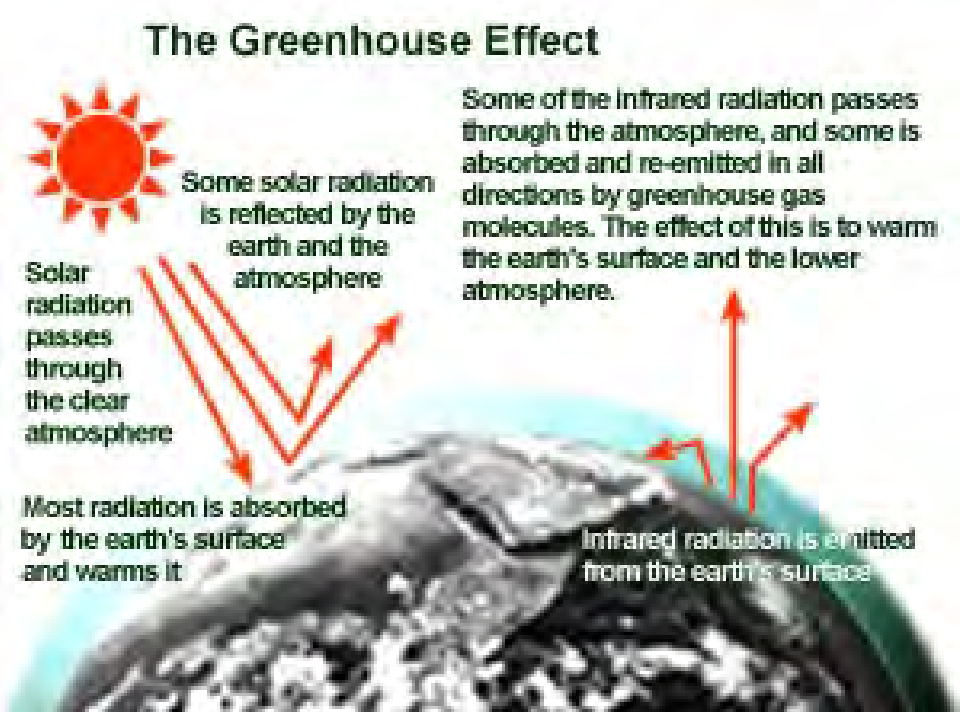
- The transparency of the atmosphere (for example, clouds reflect solar radiation);
- Hours of daylight; and
- The angle at which the sun's rays strike the earth.

The earth's surface absorbs short-wave solar radiation and emits longer wavelength **terrestrial radiation**. In the atmosphere, clouds, water vapor, and to a lesser extent carbon dioxide absorb terrestrial radiation, which causes the atmosphere to warm. The atmosphere absorbs much more terrestrial radiation than solar radiation. The atmosphere also radiates energy to outer space and back to the earth's surface. The earth-atmosphere system emits terrestrial radiation continuously. The atmospheric absorption of terrestrial radiation benefits the earth by retaining energy that would otherwise be radiated to space. This phenomenon explains how air temperatures are generally warmer on nights when cloud cover is present. The **greenhouse effect** is the descriptive name given to the result of the energy exchange process that causes the earth's surface to be warmer than it would be if the atmosphere did not radiate energy back to earth. Gases such as carbon dioxide and methane (and other similarly behaving gases often called **greenhouse gases**) also increase the ability of the atmosphere to absorb radiation (Exhibit 2).

The amount of solar radiation reaching the earth's surface varies from place to place. In addition, different types of earth surfaces (and man-made structures) vary in their ability to absorb and store heat energy. For example, land masses absorb and store heat differently than water masses. The color, shape, surface texture, vegetation and presence of buildings can all influence the heating and cooling of the ground. Generally, dry surfaces heat and cool faster than moist

surfaces. Plowed fields, sandy beaches, and paved roads become hotter than surrounding meadows and wooded areas. During the day, the air over a plowed field is warmer than over a forest or swamp; during the night, the situation is reversed. The property of different surfaces which causes them to heat and cool at different rates is referred to as **differential heating**.

Exhibit 2. The Greenhouse Effect



Source: EPA's "Global Warming Kids Site."[1]

Heat is transferred within the atmosphere by conduction, convection, and advection. These processes affect the temperature of the atmosphere near the surface of the earth. **Conduction** is the process by which heat is transferred through matter without movement of the matter itself. For example, the handle of an iron skillet becomes hot due to the conduction of heat from the stove burner. Conduction occurs from a warmer to a cooler object. Heat transfer also occurs due to the movement of atmospheric gases. Meteorologists use the term **convection** to denote the transfer of heat that occurs mainly by vertical motion. Air that is warmed by a heated land surface will rise because it is lighter than the surrounding air. Likewise, cooler air aloft will sink because it is heavier than the surrounding air. Meteorologists use the term **advection** to denote heat transfer that occurs mainly by horizontal motion. All of these energy exchange processes, particularly between the earth surface and the atmosphere, produce the complex atmospheric motions of weather. As a result of these process air toxics maybe widely distributed far from their location of origin.

2.2 Effects of Topography

The physical characteristics of the earth's surface are referred to as **terrain features** or **topography**. Topography can be grouped into four general categories: flat, mountain/valley,

land/water, and urban. Topography also causes two types of turbulence in the atmosphere. As noted above, topography causes **thermal turbulence** through differential heating. Topography causes **mechanical turbulence** as the result of the wind flowing over different sizes and shapes of objects. Physical features induce a frictional effect on wind speed and direction. For example, urban settings with dense construction and tall buildings exert a strong frictional force on the wind causing it to slow down, change direction, and become more turbulent.

Urban areas have a special effect on the atmosphere due to the high density of man-made features. Building materials such as brick and concrete absorb and store heat more efficiently than soil and vegetation found in rural areas. After sunset, the urban areas continue to radiate the stored heat from buildings and paved surfaces. Air is warmed by this urban complex and rises to create a dome (**heat island**) over an urban area. Large cities continue to emit heat throughout the night and generally never completely cool down to the more stable surrounding conditions before the sun rises and begins to heat the urban complex again. The overall effect of the urban landscape is to increase the dispersion of air toxics through increased mixing.

3.0 Atmospheric Motions

The differential heating of the earth's surface causes imbalances in **air pressure**. The atmospheric pressure at any point is due to the weight of the air pressing down from above due to gravity. In any gas such as air, molecules are moving around in all directions at very high speeds. The speed actually depends on the temperature of the gas. Air pressure is caused by the molecules of atmospheric gases bumping into each other and other surfaces and bouncing off. Air pressure is a function of the number of air molecules in a given volume and the speed at which they are moving. When air is warmed, the molecules speed up, and air pressure increases. As air cools, the molecules slow down, and air pressure decreases.

3.1 Horizontal Air Motions

Air moves in an attempt to equalize response to imbalances in pressure. The movement of air (**wind**) tends to move from areas of high to low pressure. Wind is the basic element in the general circulation of the atmosphere. Wind movements from small gusts to large air masses all contribute to transport of heat, moisture and as well as air toxics around the earth. Winds are always named by the direction from which they blow. Thus a "north wind" is a wind blowing from the north to the south and a "westerly wind" blows from west to east. When wind blows more frequently from one direction than from any other, the direction is termed the **prevailing wind**. Section 4.1 provides further information on how meteorologists measure and describe wind speed and direction.

Wind speed is heavily influenced by the presence or absence of friction ("drag") and increases rapidly with height about the ground level. Wind is commonly not a steady current but is made up of a succession of gusts, slightly variable in direction, separated by lulls. Close to the earth, wind gustiness is caused by irregularities of the surface, which create **eddies**, which are variations from the main current of wind flow. Larger irregularities are caused by convection (vertical transport of heat). These and other forms of turbulence contribute to the movement of heat, moisture, dust, and pollutants into the air. See Section 2.2 for additional information on how topography affects air motions.

Air masses cover hundreds of thousands of square miles and extend upward for several miles. They are relatively homogeneous volumes of air with regard to temperature and moisture, and they acquire the characteristics of the region over which they form and travel. Pollutants released into an air mass tend to travel and disperse within the air mass. Air masses develop more commonly in some regions than in others. Air masses are classified as maritime or continental according to their origin over ocean or land, and as arctic, polar, or tropical depending principally on the latitude of origin. Continental polar air masses are similar to arctic air masses, but not as cold and dry as arctic air masses. The chief air masses that affect the weather of North America are continental polar, maritime polar, and maritime tropical.

Frontal patterns are formed by the interaction of adjacent air masses. A cold front is a transition zone where a cold air mass is moving into the area previously occupied by a warm air mass. The rise of warm air over an advancing cold front and the subsequent expansive cooling of this air lead to cloud formation, and if sufficient moisture is available precipitation near the leading edge of the front. A warm front is a transition zone where a warm air mass is moving into the area previously occupied by a cold air mass. Precipitation commonly occurs in advance of a warm front, as the warm air slowly rises above the cold air.

3.2 Vertical Air Motions

When air is displaced vertically, atmospheric behavior is a function of atmospheric stability. A stable atmosphere resists vertical motion, and air that is displaced vertically in a stable atmosphere tends to return to its original position. This atmospheric characteristic determines the ability of the atmosphere to disperse pollutants. To understand atmospheric stability and the role it plays in pollution dispersion, it is important to understand the mechanics of the atmosphere as they relate to vertical atmospheric motion.

The degree of stability of the atmosphere is determined by the temperature difference between an air parcel and the surrounding air. This difference can cause the parcel to rise or fall. There are three general categories of atmospheric stability.

- In **stable** conditions, vertical movement tends not to occur. Stable conditions occur at night when there is little or no wind. Air that is lifted vertically will remain cooler, and therefore denser than the surrounding air. Once the lifting force is removed, the air that has been lifted will return to its original position.

- **Neutral** conditions (“well mixed”) neither encourage nor discourage air movement. Neutral stability occurs on windy days or when there is cloud cover such that there is neither strong heating nor cooling of the earth’s surface. Air lifted vertically will generally remain at the lifted height.

- In **unstable** conditions, the air parcel tends to move upward or downward and to continue that movement. Unstable conditions most commonly develop on sunny days with low wind speeds where strong solar radiation is present. The earth rapidly absorbs heat and transfers some of it to the surface air layer. As warm air rises, cooler air moves underneath. The cooler air, in turn, may be heated by the earth’s surface and begin to rise. Under such conditions, vertical motion in both directions is enhanced, and considerable vertical mixing occurs.

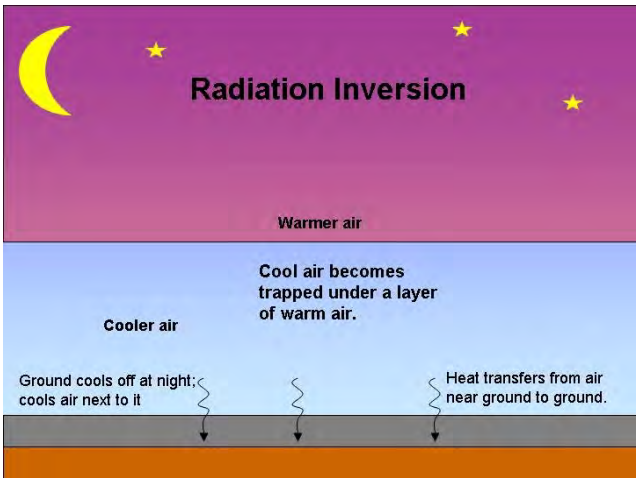
Inversions occur whenever warm over-runs cold air and "traps" the cold air beneath. Within these inversions there is little air motion, and the air becomes relatively stagnant. High air toxic concentrations can occur within inversions due to the limited amount of mixing between the "trapped" air and the surrounding atmosphere. Inversions can limit the volume of air into which emissions are dispersed, even from tall stacks. Exhibit 3 illustrates the three major types of inversions that are caused by different atmospheric interactions and can persist for different amounts of time.

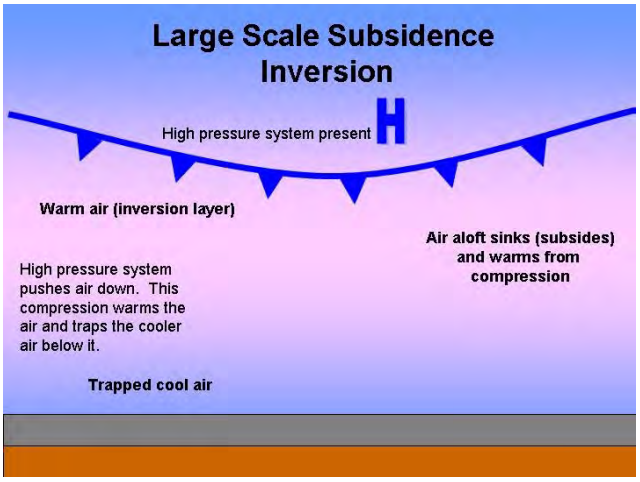
Most common is the **radiation inversion**, which occurs when the earth's surface cools rapidly. As the earth cools, it also cools the layer of air close to the surface, which becomes trapped under the layer of warmer air above. Radiation inversions usually occur in the late evening through the early morning under clear skies with calm winds, when the cooling effect is greatest. In many cases, solar radiation following sunrise results in vigorous vertical mixing, which breaks down the inversion and disperses any trapped air pollutants. Under some conditions (e.g., thick fog), the daily warming may not be strong enough to break down the inversion layer. Inversions persisting for several days may lead to increased pollutant concentrations. This situation is most likely to occur in an enclosed valley, where nocturnal, cool, downslope air movement can reinforce a radiation inversion and encourage fog formation.

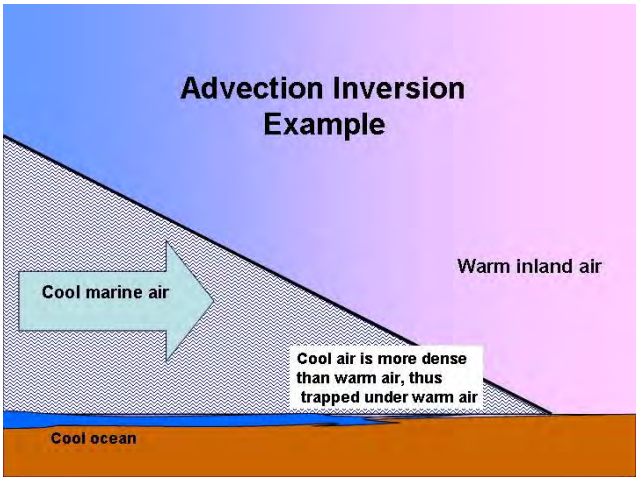
The **subsidence inversion** is almost always associated with high pressure systems. Air in a high pressure system descends and flows outward in a clockwise rotation in the Northern Hemisphere. As the air descends, the higher pressure present at lower altitudes enhances compression and warming. The inversion layer thus formed is often elevated several hundred meters above the ground surface during the day. At night, when the surface air cools, the base of the subsidence inversion often descends, even to the ground. The clear, cloudless days characteristic of high pressure systems encourage radiation inversions, so that there may be a surface inversion at night and an elevated inversion during the day. Although the layer below the inversion may vary diurnally, it will never become very deep. Subsidence inversions, unlike radiation inversions, last a relatively long time. They are associated with both the semi permanent high pressure systems centered on each ocean and the slow-moving high pressure systems that move generally from west to east across the United States. When a high pressure system stagnates, pollutant concentrations may become unusually high. The most severe air pollution episodes in the United States have occurred either under a stagnant high pressure system (for example, New York in November, 1966 and Pennsylvania in October, 1948) or under the eastern edge of the semi permanent high pressure system associated with the Pacific Ocean (Los Angeles).

Advection inversions are associated with air masses moving across surfaces of different temperatures than themselves. When warm air moves over a cold surface, the principles of conduction and convection cool the air nearer to the surface, causing a surface-based inversion. This inversion is most likely to occur in winter when warm air passes over snow cover or extremely cold land. The same type of inversion can occur when air cooled by a cold surface, such as the ocean, flows towards a warmer air mass, such as inland air in the summer.

Table 3. Types of Inversions







4.0 Meteorological Data

Measuring and recording meteorological variables provides the necessary information to manage the release of air contaminants into the atmosphere and to understand the transport and dispersion of emitted air pollutants. The most useful data in air pollution studies are wind speed and direction, ambient temperature and vertical temperature difference, solar radiation and mixing height. For indirect exposure, precipitation data are needed as well. These same variables can be used to make qualitative and quantitative predictions of ambient air toxic concentrations resulting from the release of air toxics, and to conduct quantitative risk assessments.

4.1 Wind Speed and Direction

It is common to consider wind speed and wind direction as separate variables. Wind speed determines the amount of initial dilution experienced by air toxics released into the atmosphere. Wind speed also influences the height to which the toxics will rise after being released from an elevated source - as wind increases, the air toxics are kept lower to the ground, allowing them to impact the ground at shorter distances downwind.

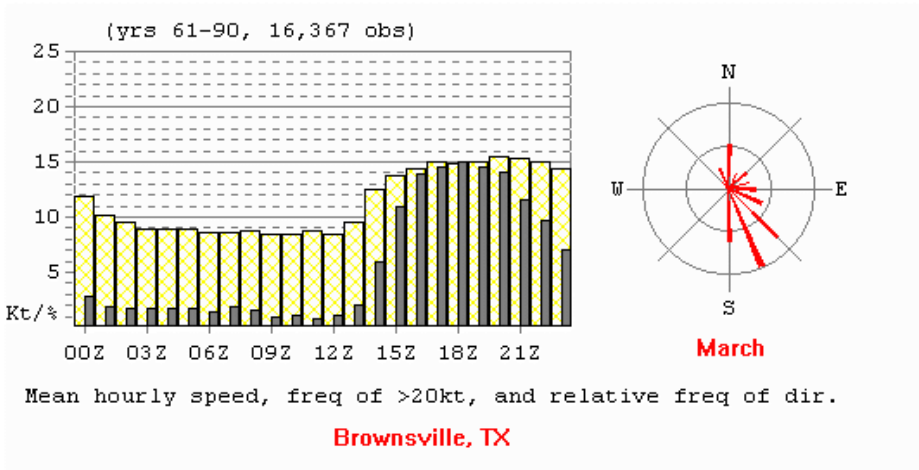
Wind direction for meteorological purposes is defined as the direction from which the wind is blowing. However, wind direction has both horizontal and vertical components. The horizontal and vertical components of the wind direction can be measured with a bi-directional wind vane or an anemometer.

Wind roses are often used to graphically depict the prevailing wind direction of an area. The wind rose depicts the relative frequency of wind direction, typically on a 16-point compass, with north, east, south, and west directions going clockwise. Each ring on the wind rose represents a frequency of the total. The WINDROSE program, which calculates and prints a frequency distribution for wind speed and wind direction for 36 (10 degree) sectors, can be obtained from EPA.[(2)]

Exhibit 4 presents an example wind rose for Brownsville, Texas. The right hand shows that the winds are predominantly from the south-southeasterly direction. The left hand side shows that the strongest winds occur between 14 and 21 UTC (8 A.M. to 3 P.M. CST). On average, 2 P.M. is the windiest time of day, averaging just over 15 knots (18 UTC). The shaded portion of the bar shows the frequency of winds over 20 knots. At noon CST, winds are over 20 knots approximately 15 percent of the time.

The distribution of pollutants is determined by the wind directions. A wind rose can provide information regarding the percentage of time that the direction(s) and speed(s) associated with a certain air quality can be expected over a time period. However, due to the influences of local terrain, possible coastal effects, exposure of the instruments, and temporal variability of the wind, the wind rose statistics from a nearby weather station may not always be representative of true wind speed and direction for the area of concern.

Exhibit 4. Example of a Wind Rose



Source: Brownsville CLIMATE page at:
http://www.srh.noaa.gov/bro/roshelp.htm

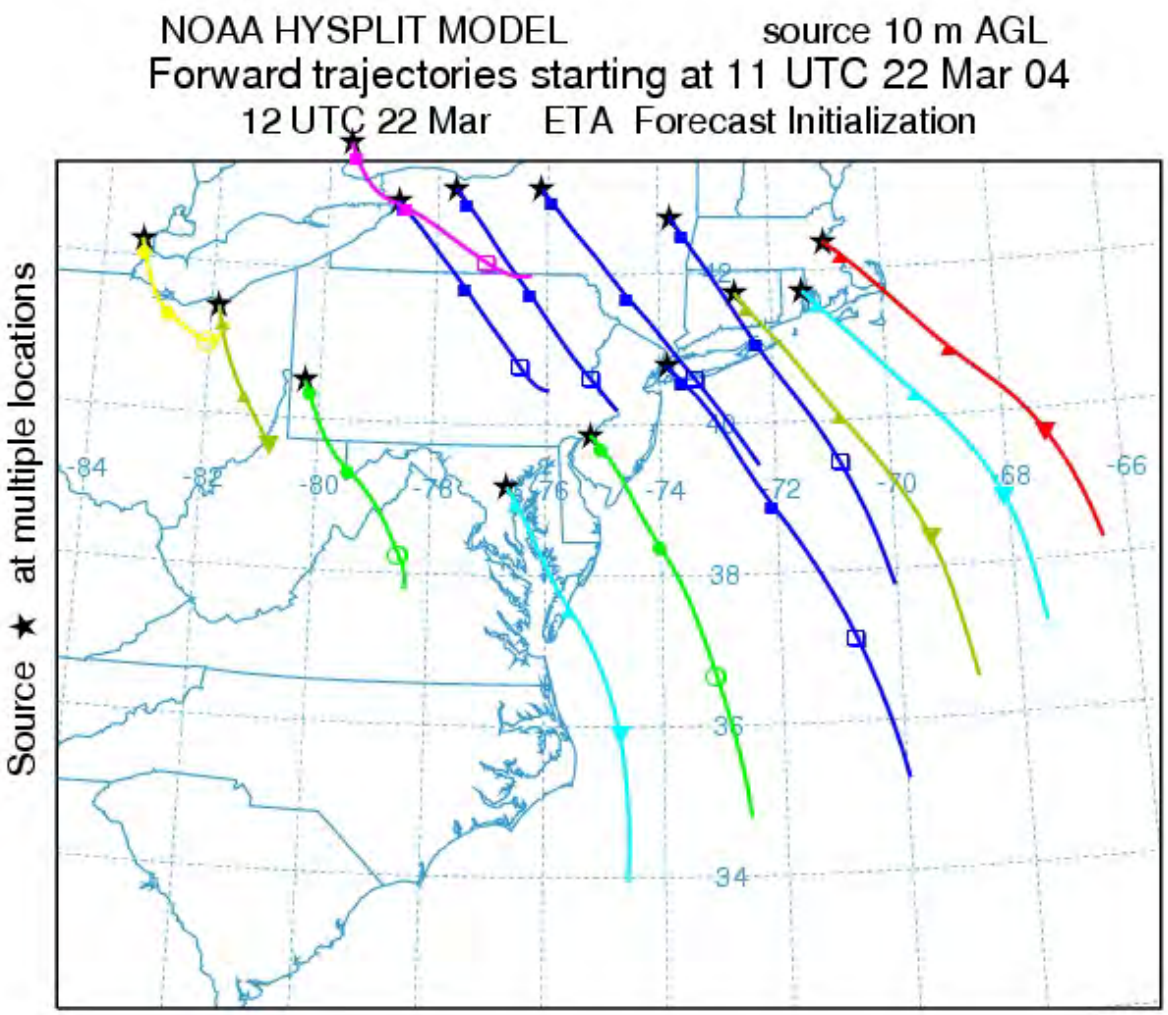
Another tool useful for understanding the distribution of pollutants is wind **trajectories**, which are aerial maps showing the path taken by a parcel of air over a period of time. Trajectories are important for understanding the transport of air toxics and/or the potential geographic regions from which sources of air toxics may emanate. Trajectories illustrate estimates of the general path that air has traveled over a recent time period in order to arrive at a particular location, and where it is likely to be going immediately afterward. The meteorological dynamics that cause air to rise or fall, and that determine its path, can affect air quality by carrying air toxics many miles from their sources. Exhibit 5 presents an example of a trajectory map for the Northeastern United States.

4.2 Other Important Meteorological Data

Both **ambient air temperatures** at a single level (typically 1.5 to 2 m) and **temperature differences** between two levels (typically 2 m and 10 m) are useful in air pollution studies. These temperature measurements are used in calculations of plume rise and can be used in determining atmospheric stability.

Solar radiation is related to the stability of the atmosphere. Cloud cover and ceiling height (height of the base of the cloud deck that obscures at least half the sky) data, taken routinely at National Weather Service (NWS) stations, provide an indirect estimation of radiation effects, and are used in conjunction with wind speed to derive an atmospheric stability category. If representative information is not available from routine NWS observations, it may be appropriate to measure solar radiation for use in determining atmospheric stability. For information on the use of cloud cover and ceiling height data in air toxics modeling, refer to EPA's Guideline on Air Quality Models.[3]

Exhibit 5. Example of a Trajectory Map



Source: National Oceanic and Atmospheric Administration (NOAA) HYSPLIT Model. [4]

The vertical depth of the atmosphere through which vertical mixing takes place is called the **mixing layer**. The top of the mixing layer is referred to as the **mixing height**. The mixing height is an important variable in air toxic studies, as it limits the vertical mixing of air toxics. Daytime mixing heights may reach as high as several kilometers during the day. Although mixing heights are not typically measured directly, they can be approximated from routine upper-air and surface meteorological measurements. In the daytime the mixing height is determined by the depth of the layer thorough which the sun's heating has established a well mixed conditions. On clear nights, radiational cooling might be expected to establish an inversions and reduce the mixing height to near zero. However, it has been found that in metropolitan areas, the urban heat island effect keeps the mixing height between 100 and 200 meters. The mixing heights are used in air quality models as an upper boundary to which air

toxics can be mixed. The level of the mixing height is most important for elevated stacks and much less so for ground level sources.

4.3 Sources of Meteorological Data

The principal federal sources for meteorological data include:

- The National Climatic Data Center (NCDC) located in Asheville, NC.
- The National Weather Service (NWS) Forecast Centers
- The EPA Support Center for Regulatory Models (SCRAM) at Research Triangle Park, NC.

State climatological offices are excellent sources of meteorological data. Data can often be obtained in a text format, and can be used in conjunction with applications that are available as downloads from federal and state data Internet sites. Commercial and university Internet sites are also sources of current weather conditions.

The NCDC is the most extensive source of historical meteorological and climatological data. EPA's SCRAM site has surface and mixing height data that can be used to create wind roses and/or used in air dispersion models. These data are for the major NWS stations throughout the United States. The data are mostly for the years 1984 through 1992 (for surface data) or 1991 (for upper air data used for mixing heights). Exhibit 6 presents a list of Internet sites where meteorological data are available.

Exhibit 6. Internet Sites with Meteorological Data
National Climatic Data Center (http:// www.ncdc.noaa.gov/oa/ncdc.html)
EPA SCRAM Site (http://www.epa.gov/scram001/)
Weather Underground (http://www.wunderground.com/)
UNSYSIS (http://weather.unisys.com/)
NWS Pleasant Hill, MO (http://www.crh.noaa.gov/eax/)
Western Regional Climate Center (http://www.wrcc.dri.edu/)
Northeast Regional Climate Center (http://met-www.cit.cornell.edu/nrcc_home.html)
Midwest Regional Climate Center (http://mcc.sws.uiuc.edu/)
High Plains Regional Climate Center (http://www.hprcc.unl.edu/)
Southern Regional Climate Center (http://www.srcc.lsu.edu/)
Southeast Regional Climate Center (http://www.sercc.com/)
WebMET.com (http://www.webmet.com/)

References

1. U.S. Environmental Protection Agency. 2004. Global Warming Kids Site. *Greenhouse Effect*. Updated March 1, 2004. Available at: http://www.epa.gov/globalwarming/kids/greenhouse.html. (Last accessed March 2004).

2. U.S. Environmental Protection Agency. 2004. Technology Transfer Network. *Support Center for Regulatory Air Models, Meteorological Data, Related Programs*. Updated March 8, 2004. Available at: http://www.epa.gov/scram001/tt24.htm#relatedprograms (Last accessed March 2004).

3. U.S. Environmental Protection Agency. 2004. Technology Transfer Network. *Support Center for Regulatory Air Models, Guidance/Support, Modeling Guidance*. Updated March 22, 2004. Available at http://www.epa.gov/scram001/tt25.htm#guidance (Last accessed March 2004).

4. Rolph, G.D. 2003. Real-time Environmental Applications and Display sYstem (READY) Website. Available at: http://www.arl.noaa.gov/ready/hysplit4.html. NOAA Air Resources Laboratory, Silver Spring, MD. (Last accessed March 2004)

Appendix H Data Quality Evaluation

Table of Contents

1.0 Introduction 1

2.0 Step 1: Gather All Data Available from the Sampling Investigation and Sort by Medium 2

3.0 Step 2: Evaluate the Analytical Methods Used 4

4.0 Step 3: Evaluate the Quality of Data with Respect to Sample Quantitation Limits 5
- 4.1 Sample Quantitation Limits (SQLs) That Are Greater Than Benchmark Concentrations 5
- 4.2 Unusually High SQLs 7
- 4.3 When Only Some Samples in a Medium Test Positive For a Chemical 8
- 4.4 When SQLs Are Not Available 8
- 4.5 When Air Toxics Are Not Detected in Any Samples in a Medium 8

5.0 Step 4: Evaluate the Quality of Data with Respect to Qualifiers and Codes 8
- 5.1 Types of Qualifiers 9
- 5.2 Using the Appropriate Qualifiers 12

6.0 Step 5: Evaluate the Quality of Data with Respect to Blanks 13

7.0 Step 6: Evaluate Tentatively Identified Compounds 14

8.0 Step 7: Compare Potential Contamination with Background 15

9.0 Step 8: Develop a Set of Data for Use in the Risk Assessment 17

10.0 Step 9: Further Limit the Number of Chemicals to Be Carried Through the Risk Assessment, If Appropriate 17
- 10.1 Conduct Initial Activities 18
- 10.2 Group Chemicals by Class 18
- 10.3 Evaluate Frequency of Detection 18
- 10.4 Use a Toxicity-Weighted or Risk-based Screening Analysis 19

11.0 Summarize and Present Data 19
- 11.1 Summarize Data Collection and Evaluation Results in Text 19
- 11.2 Summarize Data Collection and Evaluation Results in Tables and Graphics 21

References 22

1.0 Introduction

This appendix presents information for assembling the analytical data available after a monitoring investigation has been completed and deciding which of the data are of sufficient quality to be used in the risk assessment. Each sample may have been analyzed for the presence of many different air toxics, and many of those substances may have been detected. The following nine steps describe an approach to organize the data for use in a risk assessment. This stepwise approach is modified from that described in Chapter 5 of EPA's *Risk Assessment Guidance for Superfund*.[1] Note that the application of this stepwise approach requires considerable knowledge related to sampling and analysis methods and risk assessment and therefore should be done in consultation with appropriate experts.

1. Gather all data available from the sampling investigation and sort by medium (Section 2);
2. Evaluate the analytical methods used (Section 3);
3. Evaluate the quality of data with respect to sample quantitation limits (Section 4);
4. Evaluate the quality of data with respect to qualifiers and codes (Section 5);
5. Evaluate the quality of data with respect to blanks (Section 6);
6. Evaluate tentatively identified compounds (Section 7);
7. Compare potential contamination with background (Section 8);
8. Develop a set of data for use in the risk assessment (Section 9); and
9. Further limit the number of chemicals to be carried through the risk assessment, if appropriate (Section 10).
10. Summarize and present data (Section 11).

Acronyms for Appendix H

CLP	=	Contract Laboratory Program
CRDL	=	Contract-Required Detection Limit
CRQL	=	Contract-Required Quantitation Limit
EQL	=	Estimated Quantitation Limits
DL	=	Detection Limit
FIT	=	Field Investigation Team
IDL	=	Instrument Detection Limit
MDL	=	Method Detection Limit
ND	=	Non-detect
PE	=	Performance Evaluation
PQL	=	Practical Quantitation Limit
QA/QC	=	Quality Assurance/Quality Control
QL	=	Quantitation Limit
RfC	=	Inhalation Reference Concentration
RfD	=	Oral Reference Dose
SQL	=	Sample Quantitation Limit
SVOC	=	Semivolatile Organic Chemical
TCL	=	Target Compound List
TIC	=	Tentatively Identified Compound
TOC	=	Total Organic Carbon
TOX	=	Total Organic Halogens
VOC	=	Volatile Organic Chemical

The outcome of this evaluation is (1) the identification of contaminants of potential concern (COPC) that will be carried through the risk assessment and (2) reported concentrations that are of acceptable quality for use in a quantitative risk assessment. If the nine data evaluation steps are followed, the number of air toxics to be considered in the remainder of the risk assessment usually will be less than the number of substances initially identified. A suggested process for averaging acceptable data to develop chemical specific exposure concentrations is provided in Appendix I.

Definitions for Appendix H

Chemicals of Potential Concern. Air toxics that are evaluated in the risk assessment because they have the potential to affect the risk management decision. The corresponding term for ecological risk assessment are chemicals of potential ecological concern (COPEC). The risk assessment often finds that most of the risk is associated with a subset of the COPC. The subset, which drives the risk management decisions, is referred to as chemicals of concern (COC).

Common Laboratory Contaminants. Certain organic chemicals (e.g., acetone, 2-butanone, methylene chloride, toluene, and the phthalate esters) that are commonly used in the laboratory and thus may be introduced into a sample from laboratory cross-contamination.

Contract-required Quantitation Limit (CRQL). Chemical-specific levels that the laboratory must be able to routinely and reliably detect and quantitate in specified sample matrices to meet pre-specified data quality objectives. May or may not be equal to the reported quantitation limit of a given chemical in a given sample. (This term is also used in the Superfund Program under their Contract Laboratory Program.)

Detection Limit (DL). The lowest amount that can be distinguished from the normal "noise" of an analytical instrument or method.

Non-detects (NDs). Chemicals that are not detected in a particular sample above a certain limit, usually the quantitation limit for the chemical in that sample. Non-detects are often indicated by a "U" data qualifier.

Positive Data. Analytical results for which measurable concentrations (i.e., above a quantitation limit) are reported. May have data qualifiers attached (except a U, which indicates a non-detect).

Quantitation Limit (QL). The lowest level at which a chemical can be accurately and reproducibly quantitated. Usually equal to the instrument detection limit multiplied by a factor of three to five, but varies for different chemicals and different samples.

2.0 Step 1: Gather All Data Available from the Sampling Investigation and Sort by Medium

Gather data, which may be from several different sampling periods and based on several different analytical methods, from all available sources. Sort data by medium (i.e., air, water, sediment, soil, and biota, if appropriate). Exhibit 1 illustrates a useful table format for presenting data.

The data should be given to the risk assessor in a data summary report (or reports) that provides information on a number of critical elements that allow the assessor to judge the adequacy of the data to perform the risk analysis. Some of the critical elements include:

- Description of the study area,
- Sampling design and sampling locations,
- Procedures followed to ensure quality data (e.g., SOPs, QAPPs),
- Analytical methods and quantitation limits,

- Chemical-specific results on a per sample basis,

Exhibit 1. Example of Output Format for Validated Data									
Hypothetical Soil Sampling Results from Area X									
Sample medium	Soil			Soil			Soil		
Sample ID	SRB-3-1			SRB-3-1DU			SRB-3-2		
Sample or screen depth	0-1'			0-1'			2-4'		
Date collected	12/14/99			12/14/99			12/14/99		
Air Toxic	SQL[a]	Concen-tration	Quali-fier[b]	SQL[a]	Concen-tration	Quali-fier[b]	SQL[a]	Concen-tration	Quali-fier[b]
toxaphene	80	80	U	80	80	U	80	40	J
2,4,7,8-TCDD	20	10	J	20	8	J	200	200	U/J
lead	160	120	J	160	110	J	400	360	J
mercury	60	30	J	60	44	J	300	300	U/J

Note: All values other than qualifiers must be entered as numbers, not labels.
[a] Sample quantitation limit. Values for illustration only.
[b] Refer to Section 5.1 (Exhibit 3) for an explanation of qualifiers.

- Field conditions, including meteorological conditions,
- Data validation reports (both by the laboratory and any secondary validation), and
- A description of any issues with field collection, transportation/storage, or analysis that impact the veracity of the data.

The data reports provided to the risk assessor must be sufficient to allow the assessor to judge the completeness, comparability, representativeness, precision, and accuracy of the data.

[A more thorough overview of the process for assessing the usability of data for risk assessment purposes, including minimum data and documentation needs, is provided in reference 2. While this document was developed for the Superfund program, it provides relevant information for the evaluation of environmental monitoring data in a risk assessment context and, as such, is referenced here. Assessors are strongly encouraged to review this document prior to planning and scoping a assessment. This will help to ensure that all the information necessary to assess the useability of data for risk assessment purposes will be developed during the sampling and analysis phase of the assessment. (For example, assessing precision of sampling results is usually performed by establishing duplicate monitors at one or more sampling stations. The requirements for duplicate sampling must be written into the analytical plan during the planning and scoping phase of the assessment.) Reference 2 may also be consulted for information on assessing the useability of historical data for risk assessment.][2]

Evaluate data from different time periods to determine if concentrations of air toxics are similar or if changes have occurred between sampling periods (e.g., during different seasons of the year). If the methods used to analyze samples from different time periods are similar in terms of the types of analyses conducted and the QA/QC procedures followed, then the data may be combined for the purposes of quantitative risk assessment. Usually, this means averaging at least one year's worth of data to develop an estimate of long term average concentration (see Appendix I for a suggested methodology for combining results from air monitoring to estimate exposure concentration for the inhalation pathway). If concentrations of air toxics change significantly between sampling periods, it may be useful to also note temporal variation in the risk characterization. If data are available that spans long periods of time (e.g., multiple years) one could use only the most recent data in the quantitative risk assessment and evaluate older data in a qualitative analysis of changes in concentrations over time. When data are eliminated from a data set, justification for such elimination should be fully described in the risk assessment report. (A good understanding of the risk management goals will help in deciding what data to keep and how to combine data.)

3.0 Step 2: Evaluate the Analytical Methods Used

Group data according to the types of analyses conducted (e.g., Toxic Organic method, semivolatiles analyzed by EPA methods for air) to determine which analytical method results are appropriate for use in quantitative risk assessment.

Some types of data usually are ***not*** appropriate for use in quantitative risk assessment, even though they may be available. For example, analytical results that are not specific for a particular compound (e.g., total organic carbon [TOC], total organic halogens [TOX]), or results from insensitive analytical methods (e.g., analyses using portable field instruments such as organic vapor analyzers and other field screening methods) may be useful for identifying potential monitoring locations and/or examining the potential fate and transport of contaminants. These types of analytical results, however, generally are not appropriate for quantitative risk assessment. In addition, the results of analytical methods associated with unknown, few, or no QA/QC procedures are generally eliminated from further quantitative use. (Note that one of the purposes of the data quality objectives (DQO) process described in Chapter 6 and elsewhere in this manual is to avoid the use of sampling and analysis protocols that will not provide data that are useable for the risk assessment). These types of results, however, may be useful for qualitative discussions of risk.

The outcome of this step is a set of study-specific data that has been developed according to a standard set of sensitive, chemical-specific methods (see Chapters 10 and 19 for links to identified, standardized methods).

Note however that even when standardized, verified field and analytical procedures and associated QA/QC have been used during sampling and analysis, there is no guarantee that all analytical results are consistently of sufficient quality and reliability for use in quantitative risk assessment. Instead, it is important to determine – according to the steps discussed below – the limitations and uncertainties associated with the data, so that only data that are appropriate and reliable for use in a quantitative risk assessment are carried through the process.

4.0 Step 3: Evaluate the Quality of Data with Respect to Sample Quantitation Limits

This step involves evaluation of quantitation limits (QLs) and detection limits (DLs) for all of the air toxics assessed. This evaluation may lead to the re-analysis of some samples, the use of "proxy" (or estimated) concentrations, and/or the elimination of certain air toxics from further consideration (because they are believed to be absent in all samples). Types and definitions of QLs and DLs are presented in the box on the next page. Before eliminating an air toxic because they are not detected (or conducting any other manipulation of the data), the following points should be considered:

- The sample quantitation limit (SQL) for a specific air toxic may be greater than corresponding standards, criteria, or concentrations against which the concentrations will be compared (e.g., RfCs, RfDs, or ecological benchmark levels). In this situation, the "undetected" air toxic may be present at levels greater than these benchmarks and their exclusion from the risk assessment may result in an underestimate of risk.

- A particular SQL may be significantly higher than positively detected values in other samples in a data set.

These two points are discussed in detail in the following two subsections. A third subsection provides guidance for situations where only some of the samples for a given medium test positive for a particular chemical. A fourth subsection addresses the special situation where SQLs are not available. The final subsection addresses the specific steps involved with elimination of air toxics from the quantitative risk assessment based on their QLs.

4.1 Sample Quantitation Limits (SQLs) That Are Greater Than Benchmark Concentrations

QLs needed for the sampling and analysis investigation should be specified in the sampling plan. For some air toxics, however, SQLs obtained from available analytical methods may exceed certain concentrations of potential concern (e.g., RfCs, tissue sample concentrations that might result in a dietary intake level that exceeds an RfD). Exhibits 10-10 and 10-11 identify some known deficiencies in available air monitoring methods and some air toxics for which improved monitoring methods are needed. Two points should be noted when considering this situation:

- Review of available information on sources and emissions, a preliminary determination of COPC, and/or the results of fate and transport modeling ***prior to sample collection*** may allow the risk assessor to identify when more sensitive sampling and/or analytical methods may be needed before an investigation begins. This is the most efficient way to minimize the problem of QLs exceeding levels of potential concern.

- Analytical laboratories may not be able to attain QLs in particular samples that meet data quality requirements using standardized, verified procedures.

If an air toxic is not detected in any sample from a particular medium at the QL and a more sensitive method is not available, then modeling data, as well as professional judgment, may be used to evaluate whether the chemical may be present above the concentrations of potential concern. If the available information indicates the chemical is not present, see Section 3.5 of this

Detection Limits and Quantitation Limits

Strictly interpreted, the detection limit (DL) is the lowest amount of a chemical that can be "seen" above the normal, random noise of an analytical instrument or method. A chemical present below that level cannot reliably be distinguished from noise. DLs are chemical-specific and instrument-specific and are determined by statistical treatment of multiple analyses in which the ratio of the lowest amount observed to the electronic noise level (i.e., the signal-to-noise ratio) is determined. On any given day in any given sample, the calculated limit may not be attainable; however, a properly calculated limit can be used as an overall general measure of laboratory performance.

Two types of DLs may be described: instrument DLs (IDLs) and method DLs (MDLs). The IDL is generally the lowest amount of a substance that can be detected by an instrument; it is a measure only of the DL for the instrument, and does not consider any effects that sample matrix, handling, and preparation may have. The MDL, on the other hand, takes into account the reagents, sample matrix, and preparation steps applied to a sample in specific analytical methods.

Due to the irregular nature of instrument or method noise, reproducible quantitation of a chemical is not possible at the DL. Generally, a factor of three to five is applied to the DL to obtain a quantitation limit (QL), which is considered to be the lowest level at which a chemical may be accurately and reproducibly quantitated. DLs indicate the level at which a small amount would be "seen," whereas QLs indicate the levels at which measurements of concentration can be "trusted."

Two types of QLs may be described: estimated quantitation limits (EQL - also sometimes referred to as a practical quantitation limit or PQL) and sample QLs (SQLs). EPA's Superfund Program maintains a Contract Laboratory Program (CLP) as a means to obtain reliable analytical results from many different laboratories. To participate in the CLP, a laboratory must be able to meet EPA's EQL. This EQL is established by contract and, thus, is called a contract required quantitation limit (CRQL). CRQLs are chemical-specific and vary depending on the medium analyzed and the amount of chemical expected to be present in the sample. As the name implies, CRQLs are not necessarily the lowest detectable levels achievable, but rather are levels that a CLP laboratory should routinely and reliably detect and quantitate in a variety of sample matrices. For most air toxics risk assessments, SQLs, not CRQLs, will be the QLs of interest for most samples. In fact, for the same chemical, a specific SQL may be higher than, lower than, or equal to SQL values for other samples. In addition, preparation or analytical adjustments such as dilution of a sample for quantitation of an extremely high level of only one compound could result in non-detects for all other compounds included as analytes for a particular method, even though these compounds may have been present at trace quantities in the environmental sample. Because SQLs take into account sample characteristics, sample preparation, and analytical adjustments, these values are the most relevant QLs for evaluating non-detected chemicals. Also note that because of the inability to accurately measure concentration at the MDL, the SQL is used as he starting point for developing exposure concentrations where some of the samples in a data set have detections of an analyte and others do not (see Appendix I).

appendix for guidance on eliminating chemicals. If there is some indication that the chemical is present, the only choices are to:

- Use modeling results in the risk assessment;
- Re-analyze selected samples using a more sensitive analytical method (if feasible); or
- Address the chemical qualitatively in the risk assessment.

In determining which option is most appropriate for an analysis, it may be helpful to assume the air toxic is present at the SQL for purposes of an initial (tier 1) screening risk assessment. In this way, risks that would be posed if the chemical is present at the SQL can be compared with risks posed by other air toxics in the analysis.

4.2 Unusually High SQLs

Due to one or more sample-specific problems (e.g., matrix interferences), SQLs for a particular chemical in some samples may be unusually high, sometimes greatly exceeding the positive results reported for the same chemical in other samples from the data set. Even if these SQLs do not exceed health-based standards or criteria, they may still present problems. If the SQLs cannot be reduced by re-analyzing the sample, consider excluding the samples from the quantitative risk assessment if they cause the calculated exposure concentration to exceed the maximum detected concentration for a particular sample set. Exhibit 2 presents an example of how to address a situation with unusually high QLs.

Exhibit 2. Example of Unusually High Quantitation Limits

In this hypothetical example, ambient air concentrations of benzene in air have been determined using the TO-1 method.

	Concentration (ppb)			
Chemical	**Sample 1**	**Sample 2**	**Sample 3**	**Sample 4**
benzene	50 U[(a)]	59	200 U	74

[(a)] U indicates that benzene was analyzed for, but not detected; the value presented (e.g., 50 U) is the SQL.

The ambient air concentrations presented in this example (i.e., 50 to 200 ppb) vary widely from sample to sample. Assume a more sensitive analytical method would not aid in reducing the unusually high QL of 200 ppb noted in Sample 3. In this case, the result for benzene in Sample 3 would be eliminated from the quantitative risk assessment because it would cause the calculated exposure concentrations to exceed the maximum detected concentration (in this case 74 ppb). Thus the data set would be reduced to three samples: the non-detect in Sample 1 and the two detected values in Samples 2 and 4.

4.3 When Only Some Samples in a Medium Test Positive For a Chemical

Most analytes are not positively detected in each sample collected and analyzed. Instead, for a particular chemical the data set generally will contain some samples with positive results and others with non-detected results. The non-detected results usually are reported as SQLs. These limits indicate that the chemical was not measured above certain levels, which may vary from sample to sample. The chemical may be present at a concentration just below the reported quantitation limit, or it may not be present in the sample at all (i.e., the concentration in the sample is zero). Appendix I provides a suggested methodology for combining the results of a dataset where some of the samples test positive for an analyte and others do not.

4.4 When SQLs Are Not Available

In some cases, laboratory data summaries may not provide the SQLs. Instead, MDLs, CRQLs, or even IDLs may have been substituted wherever a chemical was not detected. Sometimes, no detection or quantitation limits may be provided with the data. As a first step in these situations, always attempt to obtain the SQLs, because these are the most appropriate limits to consider when evaluating non-detected air toxics (i.e., they account for sample characteristics, sample preparation, or analytical adjustments that may differ from sample to sample). Good planning and clearly articulated directions to the laboratory will help ensure that the appropriate information is provided to the risk assessor. The problem associated with incorrectly reported data should only be an issue when evaluating historical data for which there was no pre-consultation with the laboratory about what is to be provided in the data package.

If SQLs cannot be obtained, the MDL may be used as the QL, with the understanding that in most cases this will underestimate the SQL (because the MDL is a measure of detection limits only and does not account for sample characteristics or matrix interferences). The IDL should rarely be used for non-detected air toxics since it is a measure only of the detection limit for a particular instrument and does not consider the effect of sample handling and preparation or sample characteristics.

4.5 When Air Toxics Are Not Detected in Any Samples in a Medium

After considering the discussion provided in the above subsections, generally eliminate those air toxics that have not been detected in any samples of a particular medium. If information exists to indicate that the air toxics are present, they should not be eliminated from the analysis. The outcome of this step is a data set that only contains air toxics for which positive data (i.e., analytical results for which measurable concentrations are reported) are available in at least one sample from each medium. Unless otherwise indicated, assume at this point in the evaluation of data that positive data to which no uncertainties are attached concerning either the assigned identity of the chemical or the reported concentration (i.e., data that are not “tentative,” “uncertain,” or “qualitative”) are appropriate for use in the quantitative risk assessment.

5.0 Step 4: Evaluate the Quality of Data with Respect to Qualifiers and Codes

Various qualifiers and codes (hereafter referred to as qualifiers) may be attached to certain data by either the laboratories conducting the analyses or by persons performing data validation. These qualifiers often pertain to QA/QC problems and generally indicate questions concerning

chemical identity, chemical concentration, or both. All qualifiers must be addressed before the chemical can be used in quantitative risk assessment. Qualifiers used by the laboratory may differ from those used by data validation personnel in either identity or meaning.

5.1 Types of Qualifiers

Exhibit 3 provides a list of the qualifiers that laboratories are permitted to use under the Superfund CLP, along with their potential use in risk assessment. Exhibit 4 provides a similar list addressing data validation qualifiers. (Note that the data qualifiers and their meanings provided here are not consistent across all laboratories. In all cases, it is critical to discuss with the lab what they mean by the data qualifiers they report.) In general, because the data validation process is intended to assess the effect of QC issues on data usability, validation data qualifiers are attached to the data after the laboratory qualifiers and supersede the laboratory qualifiers. If data have both laboratory and validation qualifiers and they appear contradictory, ignore the laboratory qualifier and consider only the validation qualifier. If qualifiers have been attached to certain data by the laboratory and have not been removed, revised, or superseded during data validation, then evaluate the laboratory qualifier itself. If it is unclear whether the data have been validated, contact the appropriate data validation and/or laboratory personnel.

The type of qualifier and other site-specific factors determine how qualified data are to be used in a risk assessment. As seen in Exhibits 3 and 4, the type of qualifier attached to certain data often indicates how that data should be used in a risk assessment. For example, most of the laboratory qualifiers for both inorganic chemical data and organic chemical data (e.g., J, E, N) indicate uncertainty in the reported concentration of the chemical, but not in its assigned identity. Therefore, these data can be used just as positive data with no qualifiers or codes. In general, include data with qualifiers that indicate uncertainties in concentrations but not in identification.

Exhibit 3. Example of Data Qualifiers and Their Potential Use in Quantitative Risk Assessment: Superfund Contract Laboratory Program (CLP)

Qualifier	Definition	Indicates:		
		Uncertain Identity?	Uncertain Concentration?	Include Data in Quantitative Risk Assessment?
Inorganic Chemical Data[a]				
B	Reported value is <CRDL, but >IDL.	No	No	Yes
U	Compound was analyzed for, but not detected.	Yes	Yes	?
E	Value is estimated due to matrix interferences.	No	Yes	Yes
M	Duplicate injection precision criteria not met.	No	Yes	Yes

Exhibit 3. Example of Data Qualifiers and Their Potential Use in Quantitative Risk Assessment: Superfund Contract Laboratory Program (CLP)				
Qualifier	Definition	Indicates:		
		Uncertain Identity?	Uncertain Concentration?	Include Data in Quantitative Risk Assessment?
N	Spiked sample recovery not within control limits.	No	Yes	Yes
S	Reported value was determined by the Method of Standard Additions (MSA).	No	No	Yes
W	Post-digestion spike for furnace AA analysis is out of control limits, while sample absorbance is <50% of spike absorbance.	No	Yes	Yes
*	Duplicate analysis was not within control limits.	No	Yes	Yes
+	Correlation coefficient for MSA was <0.995.	No	Yes	Yes
Organic Chemical Data[(b)]				
U	Compound was analyzed for, but not detected.	Yes	Yes	?
J	Value is estimated, either for a tentatively identified compound (TIC) or when a compound is present (spectral identification criteria are met, but the value is <CRQL).	No for TCL chemicals Yes for TICs	Yes	?
C	Pesticide results were confirmed by GC/MS.	No	No	Yes
B	Analyte found in associated blank as well as in sample.[©)]	No	Yes	Yes
E	Concentration exceeds calibration range of GC/MS instrument.	No	Yes	Yes
D	Compound identified in an analysis at a secondary dilution factor.	No	No	Yes
A	The TIC is a suspected aldol-condensation product.	Yes	Yes	No

Exhibit 3. Example of Data Qualifiers and Their Potential Use in Quantitative Risk Assessment: Superfund Contract Laboratory Program (CLP)				
Qualifier	Definition	Indicates:		
		Uncertain Identity?	Uncertain Concentration?	Include Data in Quantitative Risk Assessment?
X	Additional flags defined separately.	–(d)	--	--

(a) Source: U.S. EPA, 1988. *Contract Laboratory Program Statement of Work for Inorganics Analysis: Multi-media, Multi-concentration.* Office of Emergency and Remedial Response. SOW No. 788.
(b) Source: U.S. EPA, 1988. *Contract Laboratory Program Statement of Work for Organics Analysis: Multi-media, Multi-concentration.* Office of Emergency and Remedial Response. SOW No. 288.
©) See Section 6 for a discussion of blank contamination.
(d) Data will vary with laboratory conducting analyses.

Exhibit 4. Validation Data Qualifiers and Their Potential Use in Quantitative Risk Assessment				
Qualifier	Definition	Indicates:		
		Uncertain Identity?	Uncertain Concentration?	Include Data in Quantitative Risk Assessment?
Inorganic and Organic Chemical Data[a]				
U	The material was analyzed for, but not detected. The associated numerical value is the SQL.	Yes	Yes	?
J	The associated numerical value is an estimated quantity.	No	Yes	Yes
R	Quality control indicates that the data are unusable (compound may or may not be present). Re-sampling and/or re-analysis is necessary for verification.	Yes	Yes	No
Z	No analytical result (inorganic data only).	--	--	--
Q	No analytical result (organic data only).	--	--	--
N	Presumptive evidence of presence of material (tentative identification)[b]	Yes	Yes	?

Exhibit 4. Validation Data Qualifiers and Their Potential Use in Quantitative Risk Assessment
(a) Source: U.S. EPA. 1988. *Laboratory Data Validation Functional Guidelines for Evaluating Inorganics Analysis*. Office of Emergency and Remedial Response. U.S. EPA. 1988. *Laboratory Data Validation Functional Guidelines for Evaluating Organics Analysis (Functional Guidelines for Organics)*. Office of Emergency and Remedial Response. (b) Organic chemical data only

Exhibit 5 provides examples showing the use of two commonly encountered data qualifiers: the J qualifier, and the R qualifier. Basically, the suggestion is to use J-qualified concentrations the same way as positive data that do not have this qualifier. If possible, note potential uncertainties associated with the qualifier, so that if data qualified with a J contribute significantly to the risk, then appropriate caveats can be attached. The R data qualifier indicates that the sample result was rejected by the data validation personnel, and therefore this result should be eliminated from the risk assessment.

Exhibit 5. Example Use of "J" and "R" Data Qualifiers

In this example, concentrations of benzene in an air monitor have been determined using a hypothetical analytical method. Benzene was detected in these four samples at concentrations of 3,200 µg/l, 40 µg/l, and 20 µg/l; therefore, these concentrations – as well as the non-detect – should be used in determining representative concentrations.

Chemical	Sample 1	Sample 2	Sample 3	Sample 4
Benzene	3,200 J[(a)]	40	30 U[(b)]	20 J

(a) J = The numerical value is an estimated quantity
(b) U = Compound was analyzed for, but not detected. Value presented (e.g., 30 U) is the SQL.

In this example, concentrations of lead in surface water have been determined using a hypothetical analytical method. These data have been validated, and therefore the R qualifers indicate that the person conducting the data validation rejected the data for lead in samples 2 and 3. The "UR" qualifier means that lead was not detected in Sample 3; however, the data validator rejected the non-detected result. Eliminate these two samples so that the data set now consists of only two samples (Samples 1 and 4).

Chemical	Sample 1	Sample 2	Sample 3	Sample 4
Lead	310	500 R[(a)]	30 UR[(b)]	500

(a) R = Quality control indicates that the data are unusable (compound may not be present)
(b) U = Compound was analyzed for, but not detected. Value presented (e.g., 30 UR) is the SQL.

5.2 Using the Appropriate Qualifiers

The information presented in Exhibits 3 and 4 is based on 1988 EPA guidance documents concerning qualifiers. The types and definitions of qualifiers may be periodically updated within any analytical program, and EPA regions, states, and local governments may have their own data

qualifiers and associated definitions. In general, the risk assessor should clearly understand the specific data qualifiers used by a particular analytical program and use the resulting data appropriately in the risk assessment. Make sure that definitions of data qualifiers used in the data set for the analysis have been reported with the data and are current. Never guess about the definition of qualifiers.

6.0 Step 5: Evaluate the Quality of Data with Respect to Blanks

Blank samples provide a measure of contamination that has been introduced into a sample set either (1) in the field while the samples were being collected or transported to the laboratory, or (2) in the laboratory during sample preparation or analysis. To prevent the inclusion of non-site-related contaminants in the risk assessment, the concentrations of air toxics detected in blanks must be compared with concentrations of the same air toxics detected in site samples. Exhibit 6 provides detailed definitions of different types of blanks. Blank data should be compared with results from samples with which the blanks are associated. It is often impossible, however, to determine the association between certain blanks and data. In this case, compare the blank data with results from the entire sample data set. EPA's Superfund Program has developed guidelines for comparing sample concentrations with blank concentrations; **note that the requirements or practices for a given air toxic program may differ**.

- **Blanks containing common laboratory contaminants**. As discussed in the EPA documents cited in Exhibits 3 and 4, acetone, 2- butanone (or methyl ethyl ketone), methylene chloride, toluene, and the phthalate esters are considered by EPA to be common laboratory contaminants. If the blank contains detectable levels of common laboratory contaminants, EPA guidance indicates that the sample results should be considered as positive results only if the concentrations in the sample exceed **ten times** the maximum amount detected in any blank. If the concentration of a common laboratory contaminant is less than ten times the blank concentration, then EPA guidance indicates to conclude that the chemical was not detected in the particular sample and consider the blank-related concentrations of the chemical to be the quantitation limit for the chemical in that sample. Note that if all samples contain levels of a common laboratory contaminant that are less than ten times the level of contamination noted in the blank, then completely eliminate that chemical from the set of sample results.

- **Blanks containing chemicals that are not common laboratory contaminants**. As discussed in the previously referenced guidance, if the blank contains detectable levels of one or more organic or inorganic chemicals that are not considered by EPA to be common laboratory contaminants, then consider sample results as positive only if the concentration of the chemical in the sample exceeds **five times** the maximum amount detected in any blank. Treat samples containing less than five times the amount in any blank as non-detects, and consider the blank-related chemical concentration to be the quantitation limit for the chemical in that sample. Again, note that if all samples contain levels of a chemical that are less than five times the level of contamination noted in the blank, then completely eliminate that chemical from the set of sample results.

Exhibit 6. Types of Blanks
Blanks are analytical quality control samples analyzed in the same manner as site samples. They are used in the measurement of contamination that has been introduced into a sample either (1) in the field while the samples were being collected or transported to the laboratory or (2) in the laboratory during sample preparation or analysis. Four types of blanks – trip, field, laboratory calibration, and laboratory reagent (or method) – are described below. A discussion on the water used for the blank also is provided. **Trip Blank**. This type of blank is used to indicate potential contamination due to migration of volatile organic chemicals (VOCs) from the air on the site or in sample shipping containers, through the septum or around the lid of sampling vials, and into the sample. A trip blank consists of laboratory distilled, deionized water in a 40-ml glass vial sealed with a teflon septum. The blank accompanies the empty sample bottles to the field as well as the samples returning to the laboratory for analysis; it is not opened until it is analyzed in the lab with the actual site samples. The containers and labels for trip blanks should be the same as the containers and labels for actual samples, thus making the laboratory "blind" to the identity of the blanks. **Field Blank**. A field blank is used to determine if certain field sampling or cleaning procedures (e.g., insufficient cleaning of sampling equipment) result in cross-contamination of site samples. Like the trip blank, the field blank is a sample of distilled, deionized water taken to the field with empty sample bottles and is analyzed in the laboratory along with the actual samples. Unlike the trip blank, however, the field blank sample is opened in the field and used as a sample would be (e.g., it is poured through cleaned sampling equipment or it is poured from container to container in the vicinity of a gas-powered pump). As with trip blanks, the field blanks' containers and labels should be the same as for actual samples. **Laboratory Calibration Blank**. This type of blank is distilled, deionized water injected directly into an instrument without having been treated with reagents appropriate to the analytical method used to analyze actual site samples. This type of blank is used to indicate contamination in the instrument itself, or possibly in the distilled, deionized water. **Laboratory Reagent or Method Blank**. This blank results from the treatment of distilled, deionized water with all of the reagents and manipulations (e.g., digestions or extractions) to which site samples will be subjected. Positive results in the reagent blank may indicate either contamination of the chemical reagents or the glassware and implements used to store or prepare the sample and resulting solutions. Although a laboratory following good laboratory practices will have its analytical processes under control, in some instances method blank contamination cannot be entirely eliminated. **Water Used for Blanks**. For all the blanks described above, results are reliable only if the water comprising the blank was clean. For example, if the laboratory water comprising the trip blank was contaminated with VOCs prior to being taken to the field, then the source of VOC contamination in the trip blank cannot be isolated (see laboratory calibration blank).

7.0 Step 6: Evaluate Tentatively Identified Compounds

Both the identity and reported concentration of a tentatively identified compound (TIC) is questionable (see Exhibit 7). Two options for addressing TICs exist, depending on the relative number of TICs compared to non-TICs. If the risk assessment involves a regulatory decision, the risk assessor is strongly encouraged to consult the appropriate regulatory authorities about how to address TICs in the risk assessment.

- **When few TICs are present**. When only a few TICs are present, and either (a) no information indicates that either a particular TIC may indeed be present (e.g., it is not present in emissions from the source(s) being evaluated or other nearby sources), or (b) the estimated concentration is relatively low, and therefore, the risk estimate would likely not be dominated by the TIC, then generally do not include the TICs in the risk assessment.

- **When Many TICs are present**. If many TICs are present, or if TIC concentrations appear high or site information indicates that TICs are indeed present, then further evaluation of TICs is necessary. If sufficient time is available, use more sensitive analytical methods to confirm the identity and to positively and reliably measure the concentrations of TICs prior to their use in the risk assessment. If such methods are unavailable or impractical, then the TICs should be included as COPC in the risk assessment and (usually) discussed qualitatively in the risk characterization along with a discussion of the uncertainty in both identity and concentration.

Exhibit 7. Tentatively Identified Compounds (TICs)
The set of compounds analyzed in a particular laboratory protocol may be a limited subset of the organic air toxics that could actually be present in specific emissions being evaluated. Thus, a laboratory analysis may indicate the presence of additional organic compounds not being specifically evaluated. The presence of additional compounds may be indicated, for example, by"peaks" on a chromatogram (a chromatogram is a paper representation of the response of the instrument to the presence of a compound). The laboratory may be required to attempt to identify some of these compounds (e.g., the highest peaks) using computerized searches of a library containing mass spectra (essentially "fingerprints" for particular compounds). When the mass spectra match to a certain degree, the compound (or general class of compound) is named; however, the assigned identity is in most cases highly uncertain. These compounds are called tentatively identified compounds (TICs). The analytical protocols being used by the laboratory may include procedures to obtain a rough estimate of the concentrations of TICs. These estimates, however, generally are highly uncertain and could be orders of magnitude higher or lower than the actual concentration. For TICs, therefore, assigned identities may be inaccurate, and quantitation is certainly inaccurate. Due to these uncertainties, TIC information often is not provided with data summaries. Additional sampling and analysis using different or more sensitive methods may reduce the uncertainty associated with TICs and, therefore, TIC information should be sought even if it is absent from data summaries.

8.0 Step 7: Compare Potential Contamination with Background

In some cases, a comparison of sample concentrations with background concentrations is useful for identifying the relative contribution of the source(s) being evaluated and other potential sources to the total concentrations to which a population may be exposed. Often, however, the comparison of samples with background is unnecessary because the risk estimates resulting from other sources are very low compared to those resulting from the source(s) being evaluated.

Information collected during the risk assessment can provide information on two types of background chemicals: (1) naturally occurring chemicals that have not been influenced by humans and (2) chemicals that are present due to anthropogenic sources. Either type of background chemical can be either localized or widespread. Information on background chemicals may have been obtained by the collection of background samples and/or from other

sources (e.g., County Soil Conservation Service surveys, United States Geological Survey reports). Background concentrations should be from the vicinity of the location sampled. For example, background air samples are generally collected upwind from the study area to estimate concentrations of chemicals in the air mass that is moving into the study area. For water, samples are taken upstream of the area where deposition (or erosion of contaminated soils) is occurring.

Background samples collected during the monitoring effort should not be used if they were obtained from areas influenced or potentially influenced by the source(s) being evaluated. Instead, the literature sources mentioned in the previous paragraph may be consulted to determine expected background levels of air toxics in the study area. Care must be taken in using literature sources, because the data contained therein might represent nationwide variation in a particular parameter rather than variation typical of the geographic region or geological setting in which the site is located. For example, a literature source providing concentrations of chemicals in soil on a national scale may show a wide range of concentrations that is not representative of the variation in concentrations that would be expected within a particular study area.

Both the concentration of the chemical in the study-area and the concentration in background media should be clearly articulated in the risk assessment report. Background concentrations should generally not be subtracted from study-area specific concentrations; rather, they should be compared (e.g., as barcharts). Statistical analyses that indicate whether study-area and background concentrations are different may also be presented. (In cases where background comparisons will be made, the statistical methods that will be used to compare study-area concentrations to background concentrations should be identified prior to the collection of samples.)

As an example, chromium is present in air releases from a source in a study area and chromium is also naturally occurring in study area soils. In this case, it may be necessary to include a careful comparison of the relative magnitude of estimated exposure and risk due to background vs. estimated exposure and risk from total (i.e., deposited chromium + background chromium) . This can be done by the bar chart method mentioned above and may be augmented by statistical analyses that attempt to answer the question about whether study area soil concentrations of chromium are statistically different from background soils. Again, consultation with the appropriate decision making authorities is strongly encouraged to ensure that they get the type of information that they will need to make their risk management decisions. (Note that, in general, comparison with naturally occurring levels is commonly performed primarily for inorganic chemicals such as metals, because the majority of organic air toxics released to the environment are not naturally occurring (even though they may be ubiquitous). Similar to naturally occurring background concentrations, anthropogenic levels resulting from human sources (other than those being evaluated in the air toxics risk assessment) may also be present. For example, an assessment that is evaluating exposures to dioxin from a specific source may also have to contend with dioxin that is also present in the study area that has resulted from numerous other small sources in the area (and possibly also from naturally occurring sources such as forest fires and some amount of longer range transport). Similar to naturally occurring chemicals, some combination of background sampling, literature values, modeling, and statistical analysis can be performed to try and sort out how much of the concentrations and risk are due to the source(s) in question and how much is present due to other human (and non-human) influences.

9.0 Step 8: Develop a Set of Data for Use in the Risk Assessment

After the evaluation of data is complete as specified in previous sections, a list of the samples (by medium) is made that will be used to estimate exposure concentrations. In addition, a list of COPC (also by medium) will be needed for the quantitative risk assessment. This list should include chemicals that were:

- Positively detected in at least one sample in a given medium, including (a) chemicals with no qualifiers attached (excluding samples with unusually high detection limits), and (b) chemicals with qualifiers attached that indicate known identities but unknown concentrations (e.g., J-qualified data);

- Detected at levels significantly elevated above levels of the same chemicals detected in associated blank samples;

- Only tentatively identified but either may be associated with emissions from the source(s) being evaluated based on ancillary information or have been confirmed by additional analysis; and/or

- Transformation products of air toxics demonstrated to be present.

Air toxics that were not detected in samples from a given medium (i.e., non-detects) but that may be present at the site also may be included in the risk assessment if an evaluation of the risks potentially present at the detection limit is desired.

10.0 Step 9: Further Limit the Number of Chemicals to Be Carried Through the Risk Assessment, If Appropriate

For certain assessments, the list of air toxics potentially related to emissions from the source(s) being evaluated and remaining after quantitation limits, qualifiers, blank contamination, and background have been evaluated may be lengthy. ***Note, however, that often a modeling analysis can identify the subset of air toxics in the emissions being evaluated that are most likely to contribute significantly to risk, and therefore limit the scope of any subsequent sampling and analysis effort***. Carrying a large number of chemicals through a quantitative risk assessment may be complex, and it may consume significant amounts of time and resources. The resulting risk assessment report may be difficult to read and understand, and it may distract from the dominant risks. In these cases, the procedures discussed in this section – using chemical classes, frequency of detection, essential nutrient information, and a concentration toxicity screen – may be used to further reduce the number of COPC in each medium.

If conducting a risk assessment on a large number of chemicals is feasible (e.g., because of adequate computer capability), then the procedures presented in this section may be omitted. However, the most important chemicals (e.g., those presenting 99 percent of the risk) – identified after the risk assessment – may be the focus of the main text of the report, and the remaining chemicals could be presented in the appendices.

10.1 Conduct Initial Activities

There are several activities that are useful to conduct before implementing any of the procedures described in this section. The risk assessor is strongly encouraged to consult with appropriate decision making authorities prior to implementing these procedures to ensure that the resulting processed data will meet the decision makers' needs. These remaining initial activities include:

- **Considering how the rationale for the procedure should be documented**. The rationale for eliminating chemicals from the quantitative risk assessment based on the procedures discussed below should be clearly stated in the risk assessment report. This documentation, and its possible defense at a later date, could be fairly resource- intensive. If a continuing need to justify this step is expected, then any plans to eliminate chemicals should be reconsidered.

- **Examining historical information about the source(s) being evaluated**. Chemicals reliably associated with emissions from the source(s) being evaluated based on historical information generally should not be eliminated from the quantitative risk assessment (at least during the initial tiers of analysis), even if the results of the procedures given in this section indicate that such an elimination is possible.

- **Considering mobility, persistence, and bioaccumulation**. Three factors that should be considered are the mobility, persistence, and bioaccumulation of the chemicals. For example, a highly volatile (i.e., mobile) chemical such as benzene, a long-lived (i.e., persistent) chemical such as dioxin, or a readily bioaccumulated chemical such as the PB-HAPs, probably should remain in the risk assessment. These procedures do not explicitly include a mobility, persistence, or bioaccumulation component, and therefore the risk assessor must pay special attention to these factors.

- **Considering special exposure routes**. For some chemicals, certain exposure routes need to be considered carefully before using these procedures. For example, some air toxics may pose a significant risk in certain circumstances due to dermal contact. The procedures described in this section may not account for exposure routes such as this.

10.2 Group Chemicals by Class

Some dose-response values used in characterizing risks are available only for certain chemicals within a chemical class. For example, slope factors are available only for some of the polycyclic aromatic hydrocarbons (PAHs). In such cases, the information provided in Chapter 12 (toxicity evaluation) and information provided on EPA's FERA website (http://www.epa.gov/ttn/fera/).

10.3 Evaluate Frequency of Detection

Chemicals that are infrequently detected may be artifacts in the data due to sampling, analytical, or other problems, and therefore may not be related to the sources being evaluated. Consider the chemical as a candidate for elimination from the quantitative risk assessment if: (1) it is detected infrequently in one or perhaps two environmental media, (2) it is not detected in any other sampled media or at high concentrations, and (3) there is no reason to believe that the chemical may be present in emissions from the source(s) being evaluated. In particular, modeling results may indicate whether monitoring data that show infrequently detected chemicals are representative of only their sampling locations or of broader areas. Because chemical

concentrations within a broad assessment area are spatially variable, the risk assessor can use modeling results to compare infrequently detected chemical concentrations to those estimated over broader areas when determining whether the subject chemicals are relevant to the overall risk assessment. Judicious use of modeling to supplement available monitoring data often can minimize the need to resort to arbitrarily setting limits on inclusion of infrequently detected chemicals in the risk assessment.

In addition to available monitoring data and modeling results, the risk assessor should consider other relevant factors (e.g., presence of sensitive subpopulations) in recommending appropriate site-specific limits on inclusion of risk assessment.

The reported or modeled concentrations and locations of chemicals should be examined to check for "hotspots" (localized areas of particularly high concentrations), which may be especially important for short-term exposures and which therefore should not be eliminated from the risk assessment. For PB-HAPs, always consider detection of particular chemicals in all sampled media because some media may be sources of contamination for other media. In addition, infrequently detected chemicals with concentrations that greatly exceed reference concentrations should not be eliminated.

10.4 Use a Toxicity-Weighted or Risk-based Screening Analysis

The objective of this screening procedure is to identify the chemicals in a particular analysis that, based on concentration and toxicity, are most likely to contribute significantly to the resulting risk estimates. These procedures are described, along, with examples, in Chapter 6.

11.0 Summarize and Present Data

The section of the risk assessment report summarizing the results of the data collection and evaluation should be titled "Identification of COPC." Information in this section should be presented in ways that readily support the calculation of exposure concentrations in the exposure assessment portion of the risk assessment. Exhibits 8 and 9 present examples of tables to be included in this section of the risk assessment report.

11.1 Summarize Data Collection and Evaluation Results in Text

In the introduction for this section of the risk assessment report, clearly discuss in bullet form the steps involved in data evaluation. If the optional screening procedure described in Section 9 was used in determining COPC, these steps should be included in the introduction. If both historical data and current data were used in the data evaluation, state this in the introduction. Any special site-specific considerations in collecting and evaluating the data should be mentioned. General uncertainties concerning the quality associated with either the collection or the analysis of samples should be discussed so that the potential effects of these uncertainties on later sections of the risk assessment can be determined.

In the next part of the report, discuss the samples from each medium selected for use in quantitative risk assessment. Provide information concerning the sample collection methods used (e.g., grab, composite) as well as the number and location of samples. If any samples (e.g., field screening/analytical samples) were excluded specifically from the quantitative risk

assessment prior to evaluating the data, document this along with reasons for the exclusion. Again, remember that such samples, while not used in the quantitative risk assessment, may be useful for qualitative discussions and therefore should not be entirely excluded from the risk assessment.

Discuss the data evaluation within the appropriate context for the risk assessment. For example, the focus may be on a particular neighborhood within the assessment area; specific types of modeled receptors; or specific geographic features such as a water body. For PB-HAPs, the discussion should include those media (e.g., wastes, soils) that are potential sources of contamination for other media (e.g., surface water/sediments). If no samples or data were available for a particular medium, discuss this in the text. For soils data, discuss surface soil results separately from those of subsurface soils. Discuss surface water/sediment results by the specific surface water body sampled.

Exhibit 8. Example of Table Format for Presenting Air Toxics Sampled in Specific Media

Air Toxic	Concentration in Medium X			
	Frequency of Detection[a]	Range of Sample Quantitation Limits (SQLs) (units)	Range of Detected Concentrations (units)	Background Levels
Chemical A	3/25	2 - 30	320 - 4600	100 - 140
Chemical B[b]	25/25	1 - 32	17 - 72	--

-- Not sampled
[a] Number of samples in which the chemical was positively detected over the number of samples available
[b] Identified as a COPC based upon evaluation of data according to procedures described in text of report

For each medium, identify in the report the chemicals for which samples were analyzed, and list the analytes that were detected in at least one sample. If any detected chemicals were eliminated from the quantitative risk assessment based on evaluation of data (i.e., based on evaluation of data quality, background comparisons, and the optional screening procedures, if used), provide reasons for the elimination in the text (e.g., chemical was detected in blanks at similar concentrations to those detected in samples or chemical was infrequently detected).

Exhibit 9. Example of Table Format for Summarizing COPC in All Media Sampled				
Air Toxic	Concentration			
	Air ($\mu g/m^3$)	Soils (mg/kg)	Surface Water (µg/l)	Sediments (µg/l)
Chemical A	0.5 - 225	5 - 1,100	2 - 30	--
Chemical B	0.1 - 22	0.5 - 6.4	--	12 - 3650
Chemical C	0.01 - 2.2	--	50 - 440	100 - 11,000
Chemical D	3 - 854	2 - 12	--	
-- Not sampled				

The final subsection of the text is a discussion of general trends in the data results. For example, the text may mention (1) whether concentrations of COPC in most media were close to the detection limits or (2) trends concerning chemicals detected in more than one medium or in more than one operable unit at the site. In addition, the location of hot spots should be discussed, as well as any noticeable trends apparent from sampling results at different times.

11.2 Summarize Data Collection and Evaluation Results in Tables and Graphics

As shown in Exhibit 8, a separate table that includes all chemicals detected in a medium can be provided if appropriate. Chemicals that have been determined to be of potential concern based on the data evaluation should be designated in the table with an asterisk to the left of the chemical name.

For each chemical, present the frequency of detection in a certain medium (i.e., the number of times a chemical was detected over the total number of samples considered) and the range of detected or quantified values in the samples. Do not present the QL or similar indicator of a minimum level (e.g., <10 mg/L, ND) as the lower end of the range; instead, the lower and upper bound of the range should be the minimum and maximum detected values, respectively. The range of reported QLs obtained for each chemical in various samples should be provided in a separate column. Note that these QLs should be sample-specific; other types of non-sample-specific values (e.g., MDLs or CRQLs) should be provided only when SQLs are not available. Note that the range of QLs would not include any limit values (e.g., unusually high QLs) eliminated based on the guidance in Section 3. Finally, naturally occurring concentrations of chemicals used in comparing sample concentrations may be provided in a separate column. The source of these naturally occurring levels should be provided in a footnote. List the identity of the samples used in determining concentrations presented in the table in an appropriate footnote.

The final table in this section is a list of the COPC presented by medium at the site or by medium within each operable unit at the site. A sample table format is presented in Exhibit 9. Ths isopleth is another useful type of presentation of chemical concentration data (not shown). This

graphic characterizes the monitored or modeled concentrations of chemicals at a site and illustrates the spatial pattern of contamination.

References

1. U.S. Environmental Protection Agency. 1989. *Risk Assessment Guidance for Superfund: Volume I. Human Health Evaluation Manual (Part A).* Office of Emergency and Remedial Response. Washington, DC, EPA/541/1-89/002, available at: http://www.epa.gov/superfund/programs/risk/ragsa/index.htm

2. U.S. Environmental Protection Agency. 1992. Guidance for Data Useability in Risk Assessment (Part A). Office of Emergency and Remedial Response, Washington, DC. Publication 92857-09A, PB92-93356, available at: http://www.epa.gov/oerrpage/superfund/programs/risk/datause/parta.htm.

Appendix I Use of Air Monitoring Data to Develop Estimates of Exposure Concentration (Data Analysis and Reduction)

Table of Contents

1.0 Introduction 1

2.0 Data Treatment and Handling of Non-Detects 2

3.0 Statistical Methods: Characterization of Concentration Data 4

References 8

1.0 Introduction

This appendix discusses the process of air monitoring data analysis and reduction, the goals of which are to (1) extract and summarize air monitoring data needed for the risk assessment, (2) use the data to develop estimates of exposure concentration (EC), and (3) present the results of the air monitoring study in an informative and understandable format. In short, this Appendix describes how to take the refined air monitoring data sets developed according to the processes described in Appendix H and use them to develop estimates of exposure concentration. Standard computer software packages, such as Microsoft Excel® or the Statistical Analysis System,® may be used to generate summary statistics for each chemical and monitoring location. Summary statistics should include:

- The frequency of detection, or the proportion of total valid measurements collected which were present at or above the respective sample quantitation limit (SQL) and including detections marked with certain data qualifier (e.g., "J" values - see Appendix H);

- The range of concentrations detected (highest and lowest concentrations measured for each chemical at each monitoring site – including J values);

- The statistical description of the data (e.g., normally distributed, log-normally distributed), based on standardized statistical tests;

- The range of sample quantitation limits (SQLs); and

- An arithmetic mean value, the standard deviation, the median value (i.e., 50th percentile), and the 95th percentile upper confidence limit (95% UCL) of the arithmetic mean.

Tentatively Identified Compounds (TICs)

As noted in Appendix H, TICs are chemicals identified in the laboratory, but which cannot be identified with complete accuracy. Given that there is not certainty as to their identify (and because, there often is no toxicity data for them), TICs are often assess only qualitatively in the risk assessment. The level of detail applied to TICs depends on their tentative identification (are they known toxic compounds), their concentration, known sources, and frequency of detection. Depending on the answers to these questions, the analyst may recommend that re-sampling be performed to try to more accurately determine the nature of the TICs.

The mathematical formulas and procedures for calculating these summary statistics are provided in Section 3 below.

Statistical analysis of air monitoring data may be conducted using standard methods such as those outlined in EPA's *Guidance for Data Quality Assessment - Practical Methods for Data Analysis*.[(1)] This manual provides a detailed description of the formulae that should be used in estimating the parameters mentioned above, and reviews issues associated with data treatment (e.g., treatment of non-detects, use of J-qualified data). EPA's *Calculating Upper Confidence Limits for Exposure Point Concentrations at Hazardous Waste Sites* [(2)] is also an important reference to consider when evaluating air monitoring data for exposure assessments. Readers are encouraged to review both of these document prior to using monitoring data to calculate exposure concentrations.

Data Qualifiers

Having obtained a monitoring result, it is necessary to assign a qualifier to it so decision-makers can understand the quality of the result and, hence, the role the result might play in decisions (a more complete discussion of data qualifiers is provided in Appendix H).

- **U Flag**. If the value is below the MDL, the result should be flagged as <MDL or as U or "undetected." This indicates that it cannot be determined, within the limits described in the DQOs, that the compound is present in the sample. (Note, however, that some labs flag data below the SQL as U, even though they actually detect it. It is important to work through such details with a laboratory prior to analysis of samples.)

- **J Flag**. If the result is above the MDL, but less than the SQL, the result should be flagged as J or "estimated concentration." This indicates that the compound was detected in the sample, and can be quantified, but not within the limits on accuracy described in the DQOs.

- **R Flag**. If there are significant problems with the sample (e.g. improper calibration, or extensive holding time, or very low recovery efficiency), the result should be flagged as R or "unusable." This might occur, for example, if calibration procedures are judged inadequate. If the compound is of interest, and/or other results suggest the potential for significant concentration, then re-sampling or re-analysis is usually necessary.

Interpretation of these flagged results in the context of a risk assessment is described in Chapters 5 and 6 of the EPA document *Guidance for Data Usability in Risk Assessment, Part A* (Publication 9285.7-09A, Washington, DC, April 1992; available at www.epa.gov/superfund/programs/risk/datause/parta.htm). Another excellent source is Exhibits 5-4 and 5-5 in EPA's *Risk Assessment Guidance for Superfund (RAGS) Part A*, available at www.epa.gov/oerrpage/superfund/programs/risk/ragsa/index.htm. Appendix H of this volume also discusses this subject in some detail.

2.0 Data Treatment and Handling of Non-Detects

Calculation of summary descriptive statistics (arithmetic mean, standard deviation, median, 95% upper confidence limit) requires resolution of certain issues regarding the treatment of sampling data. Specifically, assumptions must be made regarding:

- Treatment of duplicate samples;
- Treatment of instances in which chemicals are not detected; and
- Use of measurements in which the identity of a chemical is certain but its concentration is estimated with some uncertainty (often reported as "J-qualified" data).

Duplicate samples refer to the simultaneous collection or analysis of multiple (usually two) samples under conditions that are kept as similar as possible. Field duplicates usually refer to separate samples collected side-by-side in the field, while laboratory duplicates involve separately analyzing portions of the contents of a single sample. Both types of duplicates serve the similar purpose of providing a sense of the reliability, reproducibility, and precision of measurements. Ideally, duplicate samples should yield the same results. Large differences in the results of duplicate measurements potentially indicate uncertainty in data quality.

In general, once it is clear that there are no issues with field duplicate samples, they should be treated as a single sample by simply averaging their results. In cases where a chemical is detected in one but not both duplicates (or the data is J-qualified), the chemical should be assumed to be present and the two values should be averaged using the procedure for handling non-detects as described below.

When a chemical is not detected in any sample at a monitor, that chemical can usually be removed from further consideration if there are no known problems with the method, the method meets DQOs, and there is no reason to suspect that the chemical should have been detected (e.g., there are no known sources, and the chemical was also not found at other monitors). In some instances, the monitoring methodology (or interferences by other substances) do not allow for the detection of a substance, even when it is present. The assessors must weigh these types of evidence when deciding to drop a chemical from further consideration.

Various procedures have been used in risk assessments to treat non-detects (i.e., samples in which the chemical concentration is not present at or higher than the sample quantification limit (SQL)), ranging from the assumption that the chemical is absent (i.e., the true concentration is zero) to the assumption that the chemical was present in a sample at a level infinitesimally beneath the SQL (i.e. very close to the SQL and so essentially equal to the SQL). Some algorithms differentiate assignment of values to non-detects based upon the frequency of a chemical's detection. For example, if a chemical is detected in almost all samples, a concentration equal to (or some fraction of) the analytical SQL is assigned to non-detects, but if the chemical is detected in few or no samples, a concentration of zero is assumed for non-detects. In general, the strategy described below may be used to address the issue of non-detects. References 1 and 2 provide more information on this subject and analysts are encouraged to become familiar with both of these documents prior to beginning data analysis. Also note that the generic upon which the procedure described below is based assumes approximately 30 or more samples collected over the course of a year are being averaged to develop an estimate of long term exposure concentration; however, air toxics monitoring sampling schemes usually collect samples on at least a one-in-six day schedule, giving the analyst approximately 60 or more samples to work with. Sampling frequencies are sometimes even greater.

The MDL or the SQL: Which One Should I Use for Risk Assessment?

When including non-detected data in the averaging processes described on this page, one may either include the non-detected sample as ½ the MDL or ½ the SQL. The MDL ***is not appropriate*** for this task because it is a statistical measure developed by each lab for each analytical instrument and can fluctuate from day to day. In other words, it is not a stable measure of true detection "limit." In addition, many labs that actually do detect a chemical in a sample at levels less than the quantitation limit do not routinely report the detection because they cannot accurately quantitate its concentration). It is for these two reasons that ½ the SQL is used when including nondetected samples in the averaging process. This holds even when the lab in question routinely reports J-valued data.

- If less than 15% of the monitored concentrations of a given chemical at a given location are below the SQL, then a value equal to ½ of the respective SQL is assigned to these concentrations and these values are used in the calculation of summary statistics as described below.

- If greater than 90% of the monitored concentrations of a given chemical at a given location are less than the respective SQL, no estimation of the statistical descriptors is undertaken initially. If concentrations were only detected on a limited number of days (i.e., 1 to 3 days) then an investigation may be undertaken to assess the potential sources for these chemicals and the validity of the measurements. A knowledgeable statistician can help determine an appropriate method for developing summary statistics from such a data set, if appropriate.

- If between 15% and 90% of the monitored concentrations of a given chemical at a given location are greater than the respective SQL, then a value equal to ½ of the respective SQL is assigned to these concentrations and these values are used in the calculation of summary statistics as described below. For chemicals in this group that end up contributing significantly to risk, a knowledgeable statistician may reevaluate the data according to the procedures in appropriate guidance (e.g., those provided in references 1 and 2).

3.0 Statistical Methods: Characterization of Concentration Data

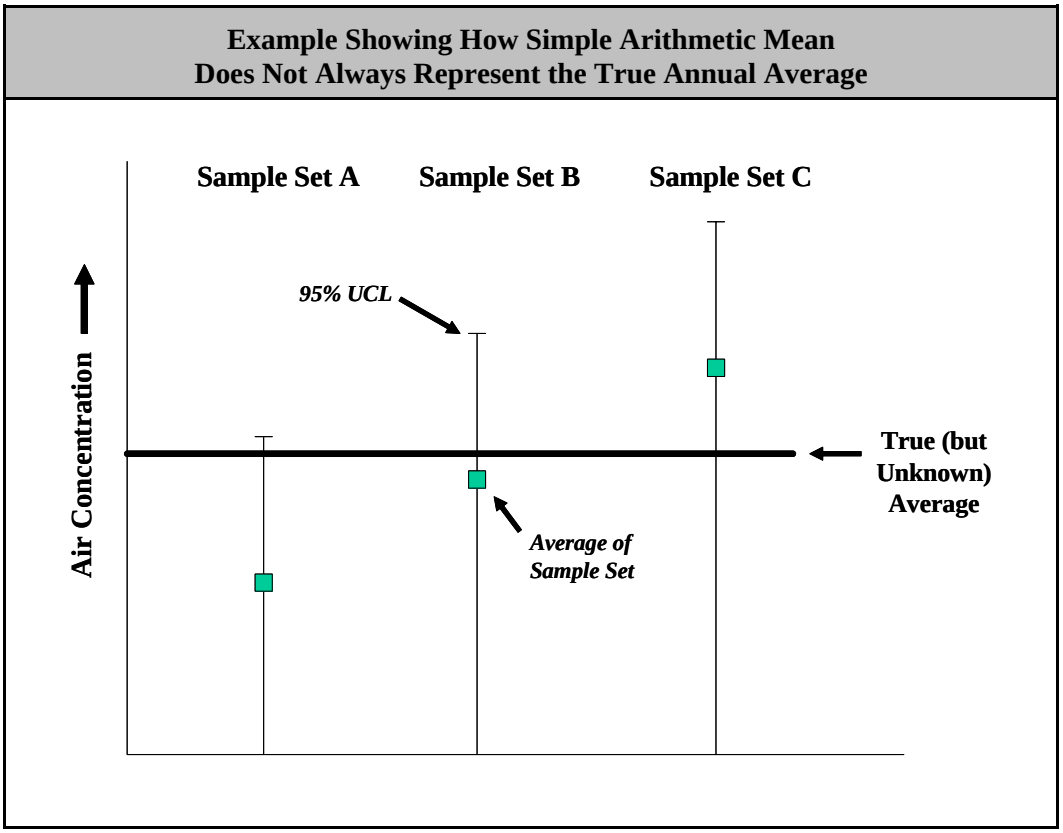
One method to estimate the long-term annual average concentration would be to calculate a simple arithmetic mean for each analyte/monitor combination. The arithmetic mean, or average is constructed from discrete sample measurements taken at the monitor over time. As noted previously, constraints on resources almost always place limits on the amount of sampling possible (e.g., air toxics samples usually cannot be collected every day). Instead, samples are usually collected roughly one out of every six days and in a manner to eliminate obvious sources of bias (e.g., samples are not uniformly collected on the same day of the week, or only on weekdays or only on weekends). In addition, collecting samples for a year allows for an evaluation of seasonal variability.

All factors being equal, one would expect the sampling results from such a monitoring program to contain equal probabilities of sampling on days when pollutant concentrations may have been relatively high as on days when pollutant concentrations may have been relatively low (or on days when meteorological conditions were conducive to high ground-level concentrations and days when they were not). Since samples are usually not collected every single day, however, one cannot be absolutely certain that all possible conditions were sampled equally. The arithmetic mean concentration is thus subject to uncertainty due to a number of factors, including:

- Daily variability in concentrations;
- The ability to measure only a finite number of instances from the distribution of concentrations over time; and
- Potential inaccuracy in individual measurements of concentrations.

This uncertainty produces a result in which the simple arithmetic mean of sampling results may underestimate, approach, or overestimate the true annual average. (The example below illustrates how three different monitoring data sets taken at the same monitor may result in an average concentration that underestimates, overestimates, or is close to the true long term average concentration.) Given this uncertainty in the use of the arithmetic mean concentration to describe “average” exposure concentration, the 95% Upper Confidence Limit of the mean (95% UCL) is commonly used as a public health protective estimate of the true annual average. Proceeding in this manner is likely to overestimate the true long-term average exposure;

however, this method virtually obviates the risk of underestimating the true exposure. EPA's Superfund program has routinely used this procedure to evaluate exposures at hazardous sites and this process has garnered long term acceptance as a public health protective approach, in light of the uncertainties.



Distributional Analysis

To calculate the 95% UCL for a chemical data set from a monitor, it is necessary to understand its underlying statistical distribution, including whether the sampling results are normally or lognormally distributed. Once the analysis goes beyond these commonly understood distributional types, the level of statistical sophistication can increase substantially. EPA's Office of Air Quality Planning and Standards (OAQPS) has developed the following pragmatic strategy to evaluate the distribution of monitoring data sets; however, other approaches are available (see references 1 and 2). Specifically, EPA suggests the following procedure:

- Inspect each data set for normality using standard test procedures (e.g., Shapiro-Wilk Test, Komolgorov-Smirnoff Test, or Filibens Test). If the assumption of normality holds, then the summary descriptive statistics, including the 95% UCL, should be calculated as described below with the equations based on the statistical assumption of a normal distribution.

- If the data are not normally distributed, then they are *presumed to be lognormal* and are log-transformed by taking the natural logarithm of the measured concentrations. The assumption of normality is then used to test the transformed data. If the assumption of normality holds for the transformed data, the summary descriptive statistics, including he

95% UCL, are developed with the transformed data using the equations based on the statistical assumption of a lognormal distribution.

- If the transformed data are not lognormal, they may be treated initially as lognormal. For chemicals in this group that significantly contribute to risk, a knowledgeable statistician may reevaluate the data (e.g., according to the procedures suggested in References 1 and 2).

The use of this simple and pragmatic approach to data analysis allows most scientists and engineers with a basic background in statistics to perform these analyses without access to advanced statistical analysis resources. Presuming a data set is lognormally distributed generally results in a 95% UCL that is conservative and, thus, public health protective. Only those chemicals that the initial risk characterization identifies as being significant risk drivers would be reevaluated with more robust statistical procedures, depending on the needs of the risk manager.

STATISTICAL FORMULAS

The following Exhibits provide the basic equations for developing the 95% UCL for chemical data sets that are either normally distributed (Exhibits 1 and 2) or lognormally – or presumed to be lognormally – distributed (Exhibits 3 and 4). The Students t and H statistics that are needed to perform these calculations are available in Gilbert's 1987 book *Statistical Methods for Environmental Pollution Monitoring*.[3]

Normally Distributed Data Sets

Exhibit 1. Directions for Computing UCL for the Mean of a Normal Distribution – Student's *t*

Let $X_1, X_2, \ldots, X_n$ represent the *n* randomly sampled concentrations.

STEP 1: Compute the sample mean $\overline{X} = \frac{1}{n}\sum_{i=1}^{n} X_i$

STEP 2: Compute the sample standard deviation $s = \sqrt{\frac{1}{n-1}\sum_{i=1}^{n}(X_i - \overline{X})^2}$

STEP 3: Use a table of quantiles of the Student's *t* distribution to find the $(1-\alpha)^{th}$ quantile of the Student's *t* distribution with *n*-1 degrees of freedom. For example, the value at the 0.05 level with 40 degrees of freedom is 1.684. A table of Student's *t* values can be found in Gilbert (1987, page 255, where the values are indexed by p = 1-α, rather than α level). The *t* value appropriate for computing the 95% UCL can be obtained in Microsoft Excel® with the formula TINV ((1-0.95)*2, *n*-1).

STEP 4: Compute the one-sided (1-α) upper confidence limit on the mean

$$UCL_{1-\alpha} = \overline{X} + t_{\alpha,n-1}\, s / \sqrt{n}$$

Exhibit 2. An Example Computation of UCL for a Normal Distribution – Student's *t*
25 VOC samples were collected from an air monitoring station and analyzed for a specific chemical. The values observed are 228, 552, 645, 208, 755, 553, 674, 151, 251, 315, 731, 466, 261, 240, 411, 368, 492, 302, 438, 751, 304, 368, 376, 634, and 810 µg/m^3. It seems reasonable that the data are normally distributed, and the Shapiro-Wilk *W* test for normality fails to reject the hypothesis that they are (*W* = 0.937). The UCL based on Student's *t* is computed as follows: STEP 1: The sample mean of the *n* = 25 values is $\bar{x} = 451$ STEP 2: The sample standard deviation of the values is $s = 198$ STEP 3: The *t*-value at the 0.05 level for 25-1 degrees of freedom is $t_{0.05,25-1} = 1.710$ STEP 4: The one-sided 95% upper confidence limit on the mean is therefore: $$95\%\ UCL = 451 + (1.710 \times 198 / \sqrt{25}) = 519$$

Lognormally Distributed Data

Exhibit 3. Directions for Computing UCL for the Mean of a Lognormal Distribution – Land Method
Let $X_1, X_2, \ldots, X_n$ represent the *n* randomly sampled concentrations. STEP 1: Compute the arithmetic mean of the log-transformed data $\overline{\ln X} = \frac{1}{n}\sum_{i-1}^{n} \ln(X_i)$ STEP 2: Compute the associated standard deviation $s_{\ln X} = \sqrt{\frac{1}{n-1}\sum_{i=1}^{n}(\ln(X_i) - \overline{\ln X})^2}$ STEP 3: Look up the $H_{1-\alpha}$ statistic for sample size *n* and the observed standard deviation of the log-transformed data. Tables of these values are given by Gilbert (1987, Tables A-10 and A-12) and Land (1975). STEP4: Compute the one-sided (1-α) upper confidence limit on the mean $$UCL_{1-\alpha} = \exp(\overline{\ln X} + s^2_{\ln X} / 2 + H_{1-\alpha}\ s_{\ln X} / \sqrt{n-1}$$

Exhibit 4. An Example Computation of UCL for a lognormal Distribution – Land Method
31 VOC samples were collected from an air monitoring stations and analyzed for a specific chemical. The values observed are 2.8, 22.9, 3.3, 4.6, 8.7, 30.4, 12.2, 2.5, 5.7, 26.3, 5.4, 6.1, 5.2, 1.8, 7.2, 3.4, 12.4, 0.8, 10.3, 11.4, 38.2, 5.6, 14.1, 12.3, 6.8, 3.3, 5.2, 2.1, 19.7, 3.9, and 2.8 μg/m^3. Because of their skewness, the data may be lognormally distributed. The Shapiro-Wilk *W* test for normality rejects the hypothesis, at both the 0.05 and 0.01 levels, that the distribution is normal. The same test fails to reject at either level the hypothesis that the distribution is lognormal. The UCL on the mean based on Land's H statistic is computed as follows:
STEP 1: Compute the arithmetic mean of the log-transformed data $\overline{\ln X} = 1.8797$
STEP 2: Compute the associated standard deviation $s_{\ln X} = 0.8995$
STEP 3: The H statistic for $n = 31$ and $s_{\ln X} = 0.90$ is 2.31
STEP4: The one-sided 95% upper confidence limit on the mean is therefore:
$$95\%\ UCL = \exp(1.8797 + 0.8995^2 / 2 + 2.31 \times 0.8995 / \sqrt{31-1}) = 14.4$$

It is statistically possible for the 95% UCL confidence limit of the mean to exceed the maximum measured concentration for a chemical. If this exceeding occurs, the maximum concentration of the chemical is commonly used in place of the 95th percentile upper confidence limit as the exposure concentration, with certain caveats (see reference 2).

References

1. U.S. Environmental Protection Agency. 1998. *Guidance for Data Quality Assessment – Practical Methods for Data Analysis*. EPA/QA-G9, QA97 Version, EPA 600/R96/O84, Washington, DC, January 1998; available at www.epa.gov/swerust1/cat/epaqag9.pdf.

2. U.S. Environmental Protection Agency. 2002. *Calculating Upper Confidence Limits for Exposure Point Concentrations at Hazardous Waste Sites*. Office of Emergency and Remedial Response, Washington, DC, December 2002. OSWER 9285.6-10, available at http://www.epa.gov/superfund/programs/risk/ragsa/ucl.pdf.

3. Gilbert, R.O. 1987. *Statistical Methods for Environmental Pollution Monitoring*. John Wiley & Sons, New York, NY.

Appendix J Air Monitoring and Sampling Methods

This appendix contains a summary of monitoring and sampling methods for a variety of organic and inorganic compounds in ambient air. Each approach is described briefly, with a listing of compounds for which it is appropriate, the detection limit, and a summary of advantages and disadvantages in using the approach. Descriptions of the methods can be downloaded from the EPA's Ambient Monitoring Technology Information Center (AMTIC) website (www.epa.gov/ttn/amtic/airtox.html).

The measurement process generally relies on collecting a sample in the field, followed by a return to the lab for analysis. A number of methods are used for initial collection of samples in the field:

1. **Sampling tubes**, in which air is drawn through a tube containing a sorbent specific to the compound being sampled, and the tube returned to the lab for analysis. Possible sorbents in the tube are organic polymers; carbon (molecular, activated, etc); polyurethane foam; silica gel; and dinitrophenylhydrazone (DNPH). Multi-sorbents also are available.

2. **Filters**, in which air is drawn through a fiber (often a glass fiber) filter, collecting the sampled compound, and returned to the lab for analysis. In some methods, air is drawn over an absorbent onto which the chemical sorbs. In some methods, a chemical reaction occurs that converts the air toxics to another material that is then analyzed.

3. **Cryogenic traps**, in which air is drawn into a chamber at low temperature, condensing the compound out of the air. The trap and condensate are returned to the lab for analysis.

4. **Evacuated chambers**, in which air is drawn into a chamber under vacuum. The chamber is returned to the lab for analysis.

An important consideration in the use of such methods is the available time between collection and analysis of samples. The compounds will degrade during the intervening holding period, and so this holding period should not exceed maximum allowed times (holding times depend on the method and compound (consult the AMTIC website for information on QA/QC for air monitoring).

Method Designation	Applicable Compounds	Approach	Detection Limit	Advantages	Disadvantages
TO-1	VOCs (80º to 200º C); e.g. benzene, toluene, xylenes.	Ambient air is drawn through organic polymer sorbent where certain compounds are trapped. The cartridge is transferred to the lab, thermally desorbed and analyzed using GC/MS or GC/FID.	0.01 to 100 ppbv	Good data base; large sample volume; water vapor not collected; wide variety of compounds collected; low detection limits; standard procedures available; practical for field use.	Highly volatile compounds and certain polar compounds not collected; rigorous clean-up of absorbent required; no possibility of multiple analyses; low breakthrough volume for some compounds; desorption of some compounds difficult; interference from structural isomers; possible contamination of sorbent and blank; artifact formation.
TO-2	Highly volatile VOCs (-15º to 120º C); e.g. vinyl chloride, chloroform, chlorobenzene.	Selected volatile organic compounds are captured on carbon molecular sieve absorbents. Compounds are thermally desorbed and analyzed by GC/MS or GC/FID techniques.	0.1 to 200 ppbv	Trace levels of VOCs are collected and concentrated; efficient collection of polar compounds; wide range of application; highly volatile compounds are absorbed; easy to use in field.	Some trace levels of organic species are difficult to recover from sorbent; interferences from structural isomers; water is collected and can de-activate absorption sites; thermal desorption of some compounds difficult.
TO-3	Nonpolar VOCs (-10º to 200º C); e.g. vinyl chloride, methylene chloride, acrylonitrile.	Vapor phase organics are condensed in a cryogenic trap. Carrier gas transfers the condensed sample to a GC column. Absorbed compounds are eluted from the GC column and measured by FID or ECD.	0.1 to 200 ppbv	Collects a wide variety of VOCs; standard procedures are available; contaminants common to absorbent materials are avoided; low blanks; consistent recovery; large data base.	Moisture levels in air can cause freezing problems in cryogenic trap; difficult to use in field; expensive; integrated sampling is difficult; compounds with similar retention times interfere.
TO-4	Pesticides and PCBs; e.g. PCBs, 4,4-DDE, DDT, DDD.	Pesticides/PCBs trap on filter and PUF absorbent trap. Trap is returned to lab, solvent extracted and analyzed by GC/FID/ECD or GC/MS.	0.2 pg/m^3 to 200 ng/m^3	Low detection limits; effective for broad range of pesticides and PCBs; PUF reusable; low blanks; excellent collection and retention efficiencies for common pesticides and PCBs.	Breakdown of PUF absorbent may occur with polar extraction solvents; contamination of glassware may increase detection limits; loss of some semi-volatile organics during storage; interference by extraneous organics; difficulty in identifying individual pesticides and PCBs if ECD used.
TO-5	Aldehydes and Ketones; e.g. formaldehyde, acetaldehyde, acrolein.	Air sample is drawn through DNPH impinger solution using a low volume pump. The solution is analyzed using HPLC with a UV detector.	1 to 50 ppbv	Specific for aldehydes and ketones; good stability for derivative compounds formed in the impingers; low detection limits.	Sensitivity limited by reagent purity; potential for evaporation of liquid over long term sampling; isomeric aldehydes and ketones may be unresolved by the HPLC system.

Method Designation	Applicable Compounds	Approach	Detection Limit	Advantages	Disadvantages
TO-6	Phosgene	Ambient air is drawn through a midget impinger containing 10 ml of 2/98 aniline/toluene (v/v). Phosgene reacts with aniline to form 1,3-diphenylurea and is analyzed using reverse-phase HPLC with a UV absorbance detector operating at 254 nm.	1 to 50 ppbv	Good specificity; good stability for derivative compounds formed in the impingers; low detection limits.	Chloroformates and acidic materials may interfere; contamination of aniline reagents may interfere; use of midget impingers in field application may not be practical.
TO-7	N-nitroso dimethylamine	Ambient air is drawn through a cartridge containing Thermosorb/N absorbant to trap N-nitrosodimethyl amine. The cartridge is returned to the lab and eluted with 5 ml of dichloromethane. The cartridge then is eluted in reverse direction with 2 ml of acetone. The N-nitrosodimethylamine is determined by GC/MS.	1 to 50 ppbv	Good specificity; good stability for derivative compounds formed on the cartridge; low detection limit for n-nitrosodimethylamine; placement of sorbent as first compound in sample train minimizes contamination; sampling system portable and lightweight.	Compounds with similar GC retention times and detectable MS ions may interfere; specificity is a limiting factor if looking for other organic amines.
TO-8	Cresol and phenol	Ambient air is drawn through two midget impingers. Phenols are trapped as phenolates in NaOH solution, which is returned to the lab and analyzed by HPLC.	1 to 250 ppbv	4,6-dinitro-2-methylphenol specific to class of compounds; good stability; detects non-volatile as well as volatile phenol compounds.	Compounds having the same HPLC retention times may interfere; phenolic compounds of interest may be oxidized; limited sensitivity.
TO-9A*	Dioxin, furan and PCBs	Ambient air is drawn through a glass fiber filter and a polyurethane foam (PUF) absorbent cartridge with a high volume sampler. The filter and PUF cartridge are returned to the lab and extracted using toluene. The extract is concentrated using the Kudrena-Danish technique, diluted with hexane, and cleaned up using column chromatography. The cleaned extract then is analyzed by high resolution GC/high resolution MS.	0.25 to 5000 pg/m^3	Cartridge is reusable; excellent detection limits; easy to preclean and extract; excellent collection and retention efficiencies; brad database; proven methodology.	Analytical interferences may occur from PCBs, methoxybiphenyls, chlorinated hydroxydiphenylethers, napthalenes, DDE and DDT with similar retention times and mass fractions; inaccurate measurement Ds/Fs are retained on particulate matter and may chemically change during sampling and storage; analytical equipment required (HRGC/HRMS) expensive and not readily available; operator skill level important; complex preparation and analysis process; can't separate particles from gas phase.

Method Designation	Applicable Compounds	Approach	Detection Limit	Advantages	Disadvantages
TO-10A	Pesticides; e.g. heptachlor, chlordane, dieldrin, aldrin	A low volume sample (1-5 L/min) is pulled through a PUF plug to trap organochlorine pesticides. After sampling, the plug is returned to the lab, extracted and analyzed by GC coupled to multi-detectors (ECID, PID, FID, etc).	1 to 100 ng/m^3	Easy field use; proven methodology; easy to clean; effective for broad range of compounds; portable; good retention of compounds.	ECD and other detectors (except MS) are subject to responses from a variety of compounds other than target analytes; PCBs, dioxins and furans may interfere; certain organochlorine pesticides (e.g. chlordane) are complex mixtures and can make accurate quantification difficult; may not be sensitive enough for all target analytes.
TO-11A	Formaldehyde, other aldehydes and ketones; e.g. formaldehyde, acetaldehyde, acrolein.	An ambient air sample is drawn through a DNPH cartridge at a rate of 500 to 1200 ml/minute. The cartridge is returned to the lab in screw-cap glass vials. The cartridge then is removed from the vial and washed with acetonitrile by gravity feed elution. The eluate is diluted volumetrically and an aliquot is removed for determination of the DNPH-formaldehyde derivative by isocratic reverse phase HPLC with UV detection at 350 nm.	0.5 to 100 ppbv	Placement of sorbent as first element in the sampling train minimizes contamination; large database; proven technology; sampling system is portable and lightweight.	Isometric aldehydes and ketones and other compounds with the same HPLC retention time as formaldehyde might interfere; Carbonyls on the DNPH cartridge may degrade if an ozone denuder is not used; liquid water captured on the DNPH cartridge during sampling may interfere; ozone and UV light deteriorates trapped carbonyls on cartridge.
TO-12	Non-methane organic compounds (NMOC)	Ambient air is drawn into a cryogenic trap, where the non-methane organic compounds (NMOCs) are concentrated. The trap is heated to move the NMOCs to the FID. Concentration of NMOCs is determined by integrating under the broad peak. Water correction is necessary.	0.1 to 200 ppmvC	Standard procedures are available; contaminants common to absorbent materials are avoided; low blanks; consistent recoveries; large data base; good sensitivity; useful for screening areas or samples; analysis much faster than GC.	Moisture levels in air can cause freezing problems; non-speciated measurement; precision is limited.
TO-15	VOCs (polar and non-polar); methanol, benzene, xylene, nitrobenzene	Whole air samples are collected in a specifically-prepared canister. VOCs are concentrated on a solid sorbent trap or other arrangement, separated on a GC column, and passed to an MS detector for identifaction and quantification.	0.2 to 25 ppbv	Incorporates a multi-sorbent/dry purge technique to manage water; has established methods performance criteria; provides enhanced provisions for QC; unique water management approach allows analysis of polar VOCs.	Expensive analytical equipment; depends critically on operator skill level.

Method Designation	Applicable Compounds	Approach	Detection Limit	Advantages	Disadvantages
TO-16	Polar and non-polar VOCs; e.g. alcohols, ketones, benzene, toluene, o-xylene, chlorobenzene.	VOCs are monitored using real-time long-path open-path Fourier transform infrared spectroscopy (FTIR).	25 to 500 ppbv	Open path analysis maintains integrity of samples; multi-gas analysis saves money and time; path-integrated pollutant concentration measurement minimizes possible sample contamination and provides real-time pollutant concentration; applicable for special survey monitoring; monitoring at inaccessible areas possible using open-path FTIR.	High levels of operator skill required; requires spectra interpretation; Limited spectral library available; higher detection limits than most alternatives; must be skilled in computer operation; substantial limitations from ambient CO_2 and humidity levels associated with spectral analysis.
TO-17	Polar and non-polar VOCs; e.g. alcohols, ketones, benzene, toluene, o-xylene, chlorobenzene.	Ambient air is drawn through a multi-bed sorbent tube where VOCs are trapped. The cartridge is returned to the lab, thermally desorbed and analyzed by GC/MS or other methods.	0.2 to 25 ppbv	Placement of the sorbent as the first element minimizes contamination from other sample train components; large selection of sorbents to match with target analyte list; includes polar VOCs; better water management using hydrophobic sorbents than Compendium Method TO-14A; large database; proven technology; size and cost advantages in sampling equipment.	Distributed volume pairs required for quality assurance; rigorous clean-up of sorbent required; no possibility of multiple analysis; must purchase thermal desorption unit for analysis; desorption of some VOCs is difficult; contamination of absorbent can be a problem.
IO-1	Suspended particulate matter (SPM); continuous measurement.	Ambient air is drawn at a rate of approximately 16 to 17 L/minute through a virtual impact or cyclonic flow filter. Particle build-up on a filter tape is determined continuously either through measurement of attenuation of beta particles incident on the tape or through an oscillating pendulum.	3 micrograms/m^3.	Less sensitive to temperature, pressure and humidity fluctuations than other continuous methods.	Results can be biased by water collection on the filter tape; oscillator must be isolated from external noise and vibrations.
IO-2	Suspended particulate matter (SPM); integrated measurement.	Ambient air is drawn through a filter with a high volume sampler, with large (> 10 micron) particles removed prior to the filter. The filter is weighed before and after sampling, with dessication to remove water vapor. Mean particulate concentration is determined from mass gain and air flow rate.	1 microgram/m^3	Well established methodology; relatively simply technique to employ	Balance used in measurement must be precise; subject to bias due to collection of water vapor if complete dessication is not obtained;

Method Designation	Applicable Compounds	Approach	Detection Limit	Advantages	Disadvantages
IO-3	Chemical species analysis of filter-collected SPM.	Ambient air is drawn through a filter with a high volume sampler, with large (> 10 micron) particles removed prior to the filter. The filter is weighed before and after sampling, with dessication to remove water vapor. The filter then is subsampled and strips digested using a microwave or hot acid extraction technique. Specific extracts are analyzed by the appropriate method.	Depends on compound considered.	Advantages depend on chemical species analyzed, but particle collection has the advantages noted in IO-2.	Disadvantages depend on chemical species analyzed.
IO-4	Reactive acidic and basic gases; strong acidity of atmospheric fine particles. HNO_3, NH_3, HCL, SO_2, NH_4, SO_4, NO_3	Based on measurement of the fine particle strong acidity component of the atmosphere. Air is drawn through an annular denuder followed by a 37 mm Teflon filter to trap the fine particle acid aerosol. The filter is returned to the lab for extraction and analysis using an aequeous solution of perchloric acid followed by titration or pH determination.		Simple method of analysis; well established methodology.	Without denuders employed to remove ammounia and other acid gases, interference can occur.
IO-5	Atmospheric mercury	Low flow (for vapor phase) or higher flow (for particulate phase) ambient air stream is flowed over gold coated bead traps and glass fiber filters. Mercury content is determined by cold-vapor atomic fluorescence spectrometry after thermal desorption.	30 pg/m^3 (particulate phase) or 45 pg/m^3 for vapor phase.	No known positive interferences using the 253.7 nm wavelength to excite the mercury atoms.	Possible interferences from PAHs and water vapor; excessive water quenches signal; free halogens can degrade trap.

Appendix K Equations For Estimating Concentrations of PB-HAP Compounds in Food and Drinking Water

Table of Contents

1.0 Introduction 1

2.0 Calculation of PB-HAP Compound Concentrations in Soil 1

2.1 Calculating Cumulative Soil Concentration (*Cs*) 2

2.2 Calculating the PB-HAP compound Soil Loss Constant (*ks*) 3
- 2.2.1 PB-HAP compound Loss Constant Due to Biotic and Abiotic Degradation *(ksg)* 4
- 2.2.2 PB-HAP compound Loss Constant Due to Soil Erosion (*kse*) 5
- 2.2.3 PB-HAP compound Loss Constant Due to Runoff (*ksr*) 6
- 2.2.4 PB-HAP compound Loss Constant Due to Leaching (*ksl*) 6
- 2.2.5 PB-HAP compound Loss Constant Due to Volatilization (*ksv*) 7

2.3 Calculating the Deposition Term (*Ds*) 8

2.4 Universal Soil Loss Equation (USLE) 9

2.5 Site-Specific Parameters for Calculating Cumulative Soil Concentration 9
- 2.5.1 Soil Mixing Zone Depth (Z_s) 9
- 2.5.2 Soil Dry Bulk Density (*BD*) 9
- 2.5.3 Available Water ($P + I - RO - E_v$) 10
- 2.5.4 Soil Volumetric Water Content (θ_{sw}) 10

3.0 Calculation of PB-HAP Compound Concentrations in Produce 10

3.1 Aboveground Produce Concentration Due to Direct Deposition (*Pd*) 11
- 3.1.1 Interception Fraction of the Edible Portion of Plant (*Rp*) 12
- 3.1.2 Plant Surface Loss Coefficient (*kp*) 12
- 3.1.3 Length of Plant Exposure to Deposition per Harvest of Edible Portion of Plant (*Tp*) . 12
- 3.1.4 Standing Crop Biomass (Productivity) (*Yp*) 12

3.2 Aboveground Produce Concentration Due to Air-to-Plant Transfer (*Pv*) 12

3.3 Produce Concentration Due to Root Uptake (*Pr*) 13

4.0 Calculation of PB-HAP Compound Concentrations in Beef and Dairy Products 14

4.1 Forage and Silage Concentrations Due to Direct Deposition (*Pd*) 15
4.1.1 Interception Fraction of the Edible Portion of Plant (*Rp*) 15
4.1.2 Plant Surface Loss Coefficient (*kp*) 15
4.1.3 Length of Plant Exposure to Deposition per Harvest of the Edible Portion of Plant (*Tp*) 16
4.1.4 Standing Crop Biomass (Productivity) (Yp) 16

4.2 Forage and Silage Concentrations Due to Air-to-Plant Transfer (*Pv*) 16

4.3 Forage, Silage, and Grain Concentrations Due to Root Uptake (*Pr*) 16

4.4 Beef Concentration Resulting from Plant and Soil Ingestion (A_{beef}) 17
4.4.1 Fraction of Plant Type *i* Grown on Contaminated Soil and Eaten by the Animal (Cattle)(F_i) 17
4.4.2 Quantity of Plant Type *i* Eaten by the Animal (Cattle) Each Day (Qp_i) 17
4.4.3 Concentration of PB-HAP compound in Plant Type *i* Eaten by the Animal (Cattle) (P_i) 18
4.4.4 Quantity of Soil Eaten by the Animal (Cattle) Per Day (*Qs*) 18
4.4.5 Average Soil Concentration Over Exposure Duration (*Cs*) 18
4.4.6 Soil Bioavailability Factor (*Bs*) 18
4.4.7 Metabolism Factor (*MF*) 18

4.5 PB-HAP compound Concentration In Milk Due to Plant and Soil Ingestion (A_{milk}) 19
4.5.1 Fraction of Plant Type *i* Grown on Contaminated Soil and Eaten by the Animal (Dairy Cattle) (F_i) 19
4.5.2 Quantity of Plant Type *i* Eaten by the Animal (Dairy Cattle) Per Day (Qp_i) 19
4.5.3 Concentration of PB-HAP compound in Plant Type *i* Eaten by the Animal (Dairy Cattle) (P_i) 20
4.5.4 Quantity of Soil Eaten by the Animal (Dairy Cattle) Per Day (*Qs*) 20
4.5.5 Average Soil Concentration Over Exposure Duration (*Cs*) 20
4.5.6 Soil Bioavailability Factor (*Bs*) 20
4.5.7 Metabolism Factor (*MF*) 20

5.0 Calculation of PB-HAP Compound Concentrations in Pork 20

5.1 Concentration of PB-HAP compound In Pork 20
5.1.1 Fraction of Plant Type *i* Grown on Contaminated Soil and Eaten by the Animal (Swine) (F_i) 21
5.1.2 Quantity of Plant Type *i* Eaten by the Animal (Swine) Each Day (Qp_i) 21
5.1.3 Concentration of PB-HAP compound in Plant Type *i* Eaten by the Animal (Swine) (P_i) 22
5.1.4 Quantity of Soil Eaten by the Animal (Swine) Each Day (Q_s) 22
5.1.5 Average Soil Concentration Over Exposure Duration (*Cs*) 22
5.1.6 Soil Bioavailability Factor (*Bs*) 22
5.1.7 Metabolism Factor (*MF*) 22

6.0 Calculation of PB-HAP Compound Concentrations in Chicken and Eggs 22

6.1 Concentration of PB-HAP compound in Chicken and Eggs 23
6.1.1 Fraction of Plant Type *i* Grown on Contaminated Soil and Eaten by the Animal (Chicken)(F_i) 23
6.1.2 Quantity of Plant Type *i* Eaten by the Animal (Chicken) Each Day (Qp_i) 23
6.1.3 Concentration of PB-HAP compound in Plant Type *i* Eaten by the Animal (Chicken) (P_i) 24
6.1.4 Quantity of Soil Eaten by the Animal (Chicken) Each Day (Qs) 24
6.1.5 Average Soil Concentration Over Exposure Duration (Cs) 24
6.1.6 Soil Bioavailability Factor (Bs) 24

7.0 Calculation of PB-HAP Compound Concentrations in Drinking Water and Fish 24

7.1 Total PB-HAP compound Load to the Water Body (L_T) 26
7.1.1 Total (Wet and Dry) Particle Phase and Vapor Phase PB-HAP compound Direct Deposition Load to Water Body (L_{DEP}) 26
7.1.2 Vapor Phase PB-HAP compound Diffusion Load to Water Body (L_{dif}) 27
7.1.3 Runoff Load from Impervious Surfaces (L_{RI}) 27
7.1.4 Runoff Load from Pervious Surfaces (L_R) 28
7.1.5 Soil Erosion Load (L_E) 29

7.2 Universal Soil Loss Equation - USLE 29

7.3 Sediment Delivery Ratio (SD) 30

7.4 Total Water Body PB-HAP compound Concentration (C_{wtot}) 30
7.4.1 Fraction of Total Water Body PB-HAP compound Concentration in the Water Column (f_{wc}) and Benthic Sediment (f_{bs}) 31
7.4.2 Overall Total Water Body PB-HAP compound Dissipation Rate Constant (k_{wt}) 33
7.4.3 Water Column Volatilization Rate Constant (k_v) 33
7.4.4 Overall PB-HAP compound Transfer Rate Coefficient (K_v) 34
7.4.5 Liquid Phase Transfer Coefficient (K_L) 35
7.4.6 Gas Phase Transfer Coefficient (K_G) 36
7.4.7 Benthic Burial Rate Constant (k_b) 36
7.4.8 Total PB-HAP compound Concentration in Water Column (C_{wctot}) 38
7.4.9 Dissolved Phase Water Concentration (C_{dw}) 38
7.4.10 PB-HAP compound Concentration Sorbed to Bed Sediment (C_{sb}) 39

7.5 Concentration of PB-HAP compound in Fish (C_{fish}) 39
7.5.1 Fish Concentration (C_{fish}) from Bioconcentration Factors Using Dissolved Phase Water Concentration 40
7.5.2 Fish Concentration (C_{fish}) from Bioaccumulation Factors Using Dissolved Phase Water Concentration 40
7.5.3 Fish Concentration (C_{fish}) from Biota-To-Sediment Accumulation Factors Using PB-HAP compound Sorbed to Bed Sediment 40

8.0 Concentrations of Dioxins in Breast Milk 41

References 42

1.0 Introduction

This Appendix describes equations used by some multimedia models to estimate media concentrations for the recommended exposure scenarios presented in Part III. Most risk assessments will use a multimedia fate and transport model to perform these calculations; the particular equations used in a given model may differ slightly from those presented here, which are taken largely from EPA's 1998 *Peer Review Draft Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities, Volume I.*[1] The equations, and descriptions of the associated parameters, are presented here simply as a general reference, and are not intended to imply a recommendation over other equations, methods, or values for describing these processes. EPA's 1998 *Risk Assessment Protocol for Hazardous Waste Combustion Facilities* provides a more detailed discussion of the origin and development of each of these equations and many of their specific parameters. It should be noted that reference made throughout this chapter to "particle phase" is generic and made without distinction between particle and particle-bound. The remainder of this chapter is divided into seven sections:

- Section 2 describes the estimating media concentration equations for soils contaminated by PB-HAP compounds.
- Section 3 describes the estimating media concentration equations used to determine PB-HAP compound concentrations in produce.
- Sections 4 through 6 describe equations used to determine PB-HAP compound concentrations in animal products (such as milk, beef, pork, poultry, and eggs) resulting from animal ingestion of contaminated feed and soil.
- Section 7 describes equations used to determine PB-HAP compound concentrations in fish through bioaccumulation (or, for some compounds, bioconcentration) from the water column, dissolved water concentration, or bed sediment – depending on the PB-HAP compound.
- Section 8 describes equations for estimating the concentrations of doxins in breast milk.

2.0 Calculation of PB-HAP Compound Concentrations in Soil

PB-HAP compound concentrations in soil are calculated by summing the vapor phase and particle phase deposition of PB-HAP compounds to the soil. Wet and dry deposition of particles and vapors are considered, with dry deposition of vapors calculated from the vapor air concentration and the dry deposition velocity. The calculation of soil concentration incorporates a term that accounts for loss of PB-HAP compounds by several mechanisms, including leaching, erosion, runoff, degradation (biotic and abiotic), and volatilization. These loss mechanisms all lower the soil concentration associated with the deposition rate.

Soil concentrations may require many years to reach steady state. As a result, the equations used to calculate the average soil concentration over the period of deposition were derived by integrating the instantaneous soil concentration equation over the period of deposition. For carcinogenic PB-HAP compounds, EPA (1998)[1] recommends using two variations of the equation (average soil concentration over exposure duration). One form should be used if the exposure duration is greater than or equal to the operating lifetime of the emission source(s), and the other should be used if the exposure duration is less than the operating lifetime of the emission source(s).

For noncarcinogenic PB-HAP compounds, EPA (1998)[1] recommends using the second form of the carcinogenic equation to calculate the highest annual average PB-HAP compound soil concentration occurring during the exposure duration. These equations are described in more detail in Section 2.1.

Soil conditions such as pH, structure, organic matter content, and moisture content affect the distribution and mobility of PB-HAP compounds. Loss of PB-HAP compounds from the soil is modeled by using rates that depend on the physical and chemical characteristics of the soil. These variables and their use are described in the following subsections, along with the recommended equations.

2.1 Calculating Cumulative Soil Concentration (*Cs*)

EPA (1998)[1] recommends the use of Equations 1A, 1B, and 1C to calculate the cumulative soil concentration (*Cs*).

Carcinogens:

For $T_2 \leq tD$

$$Cs = \frac{Ds}{k_s \cdot (tD - T_1)} \cdot \left[\left(tD + \frac{\exp(-ks \cdot tD)}{ks} \right) - \left(T_1 + \frac{\exp(-ks \cdot T_1)}{ks} \right) \right] \qquad \text{(Equation 1A)}$$

For $T_1 < tD < T_2$

$$Cs = \frac{\left(\frac{Ds \cdot tD - Cs_{tD}}{ks} \right) + \left(\frac{Cs_{tD}}{ks} \right) \cdot \left(1 - \exp\left[-ks \cdot (T_2 - tD) \right] \right)}{(T_2 - T_1)} \qquad \text{(Equation 1B)}$$

Noncarcinogens:

$$Cs_{tD} = \frac{Ds \cdot \left[1 - \exp(-ks \cdot tD) \right]}{ks} \qquad \text{(Equation 1C)}$$

where

Cs	=	Average soil concentration over exposure duration (mg PB-HAP compound/kg soil)
Ds	=	Deposition term (mg PB-HAP compound/kg soil/yr)
T_1	=	Time period at the beginning of emissions (yr)
ks	=	PB-HAP compound soil loss constant due to all processes (yr^{-1})
tD	=	Time period over which deposition occurs (time period of emissions) (yr)
Cs_{tD}	=	Soil concentration at time *tD* (mg/kg)
T_2	=	Length of exposure duration (yr)

EPA (1998)[1] recommends Equation 1C when an exposure duration that is less than or equal to the operating lifetime of the emission source(s) ($T_2 \leq tD$); when an exposure duration greater than the operating lifetime of the emissions source(s) ($T_1 < tD < T_2$), Equation 1B is recommended. For noncarcinogenic PB-HAP compounds, Equation 1C is recommended.

The PB-HAP compound soil concentration averaged over the exposure duration, represented by *Cs*, can be used for carcinogenic compounds, where risk is averaged over the lifetime of an individual. Because the hazard quotient associated with noncarcinogenic PB-HAP compounds is based on a threshold dose rather than a lifetime exposure, the highest annual average PB-HAP compound soil concentration occurring during the exposure duration period is recommended to be used for noncarcinogenic PB-HAP compounds. The highest annual average PB-HAP compound soil concentration, Cs_{tD}, will typically occur at the end of the operating life of the emission source(s).

EPA (1998) [1] recommends using the highest 1-year annual average soil concentration, determined by using Equation 1C, to evaluate risk from noncarcinogenic PB-HAP compounds.

2.2 Calculating the PB-HAP compound Soil Loss Constant (*ks*)

Organic and inorganic PB-HAP compounds may be lost from the soil by several processes that may or may not occur simultaneously. The rate at which a PB-HAP compound is lost from the soil is known as the soil loss constant (*ks*). The constant *ks* is determined by using the soil's physical, chemical, and biological characteristics to consider the loss resulting from leaching, runoff, erosion, biotic and abiotic degradation, and volatilization. EPA (1998)[1] recommends that Equation 2 be used to calculate the PB-HAP compound soil loss constant (*ks*).

$$ks = ksg + kse + ksr + ksl + ksv \qquad \text{(Equation 2)}$$

where

ks	=	PB-HAP compound soil loss constant due to all processes (yr^{-1})
ksg	=	PB-HAP compound loss constant due to biotic and abiotic degradation (yr^{-1})
kse	=	PB-HAP compound loss constant due to soil erosion (yr^{-1})
ksr	=	PB-HAP compound loss constant due to surface runoff (yr^{-1})
ksl	=	PB-HAP compound loss constant due to leaching (yr^{-1})
ksv	=	PB-HAP compound loss constant due to volatilization (yr^{-1})

As highlighted in Section 2.1, the use of Equation 2 in Equations 1A and 1B assumes that PB-HAP compound loss can be defined by using first-order reaction kinetics. First-order reaction rates depend on the concentration of one reactant.[2] The loss of a PB-HAP compound by a first-order process depends only on the concentration of the PB-HAP compound in the soil, and a constant fraction of the PB-HAP compound is removed from the soil over time. Those processes that apparently exhibit first-order reaction kinetics without implying a mechanistic dependence on a first-order loss rate are termed "apparent first-order" loss rates.[3] The assumption that PB-HAP compound loss follows first-order reaction kinetics may be an oversimplification because – at various concentrations or under various environmental conditions – the loss rates from soil systems will resemble different kinetic expressions. However, at low concentrations, a

first-order loss constant may be adequate to describe the loss of the PB-HAP compound from soil (EPA 1990)[4].

PB-HAP compound loss in soil can also follow zero or second-order reaction kinetics. Zero-order reaction kinetics are independent of reactant concentrations (Bohn, McNeal, and O'Connor 1985).[2] Zero-order loss rates describe processes in which the reactants are present at very high concentrations. Under zero-order kinetics, a constant amount of a PB-HAP compound is lost from the soil over time, independent of its concentration. Processes that follow second-order reaction kinetics depend on the concentrations of two reactants or the concentration of one reactant squared (Bohn, McNeal, and O'Connor 1985)[2]. The loss constant of a PB-HAP compound following a second-order process can be contingent on its own concentration, or on both its concentration and the concentration of another reactant, such as an enzyme or catalyst.

Because PB-HAP compound loss from soil depends on many complex factors, it may be difficult to model the overall rate of loss. In addition, because the physical phenomena that cause PB-HAP compound loss can occur simultaneously, the use of Equation 2 may also overestimate loss rates for each process (Valentine 1986).[5] When possible, the common occurrence of all loss processes should be taken into account. Combined rates of soil loss by these processes can be derived experimentally; values for some PB-HAP compounds are presented in EPA (1986).[6]

Sections 2.2.1 through 2.2.5 discuss issues associated with the calculation of the *ksl*, *kse*, *ksr*, *ksg*, and *ksv* variables.

2.2.1 PB-HAP compound Loss Constant Due to Biotic and Abiotic Degradation *(ksg)*

Soil losses resulting from biotic and abiotic degradation (*ksg*) are determined empirically from field studies and should be addressed in the literature (EPA 1990).[4] Lyman et al. (1982)[7] states that degradation rates can be assumed to follow first order kinetics in a homogenous medium. Therefore, the half-life of a compound can be related to the degradation rate constant. Ideally, *ksg* is the sum of all biotic and abiotic rate constants in the soil media. Therefore, if the half-life of a compound (for all of the mechanisms of transformation) is known, the degradation rate can be calculated. However, literature sources do not provide sufficient data for all such mechanisms, especially for soil. EPA (1994a)[8] recommends that *ksg* values for all PB-HAP compounds other than polycyclic organic matter (specifically 2,3,7,8-TCDD) should be set equal to zero. EPA (1998) [1] presents EPA recommended values for this compound-specific variable.

The rate of biological degradation in soils depends on the concentration and activity of the microbial populations in the soil, the soil conditions, and the PB-HAP compound concentration (Jury and Valentine 1986).[9] First-order loss rates often fail to account for the high variability of these variables in a single soil system. However, the use of simple rate expressions may be appropriate at low chemical concentrations (e.g., nanogram per kilogram soil) at which a first-order dependence on chemical concentration may be reasonable. The rate of biological degradation is PB-HAP compound-specific, depending on the complexity of the PB-HAP compound and the usefulness of the PB-HAP compound to the microorganisms. Some substrates, rather than being used by the organisms as a nutrient or energy source, are simply degraded with other similar PB-HAP compounds, which can be further utilized. Environmental and PB-HAP compound-specific factors that may limit the biodegradation of PB-HAP compounds in the soil environment (Valentine and Schnoor 1986)[10] include (1) availability of

the PB-HAP compound, (2) nutrient limitations, (3) toxicity of the PB-HAP compound, and (4) inactivation or nonexistence of enzymes capable of degrading the PB-HAP compound.

Chemical degradation of organic compounds can be a significant mechanism for removal of PB-HAP compounds in soil (EPA 1990).[4] Hydrolysis and oxidation-reduction reactions are the primary chemical transformation processes occurring in the upper layers of soils (Valentine 1986).[5] General rate expressions describing the transformation of some PB-HAP compounds by all non-biological processes are available, and these expressions are helpful when division into component reactions is not possible.

Hydrolysis in aqueous systems is characterized by three processes: acid-catalyzed, base-catalyzed, and neutral reactions. The overall rate of hydrolysis is the sum of the first-order rates of these processes (Valentine 1986).[5] In soil systems, sorption of the PB-HAP compound can increase, decrease, or not affect the rate of hydrolysis, as numerous studies cited in Valentine (1986)[5] have shown. The total rate of hydrolysis in soil can be predicted by adding the rates in the soil and water phases, which are assumed to be first-order reactions at a fixed pH (Valentine 1986).[5] Methods for estimating these hydrolysis constants are described by Lyman et al. (1982).[7]

Organic and inorganic compounds also undergo oxidation-reduction (redox) reactions in the soil (Valentine 1986).[5] Organic redox reactions involve the exchange of oxygen and hydrogen atoms by the reacting molecules. Inorganic redox reactions may involve the exchange of atoms or electrons by the reactants. In soil systems where the identities of oxidant and reductant species are not specified, a first-order rate constant can be obtained for describing loss by redox reactions (Valentine 1986).[5] Redox reactions involving metals may promote losses from surface soils by making metals more mobile (e.g., leaching to subsurface soils).

2.2.2 PB-HAP compound Loss Constant Due to Soil Erosion (*kse*)

EPA (1998) [1] recommends that the constant for the loss of soil resulting from erosion (*kse)* is recommended to be set equal to zero in most cases. If soil erosion is a significant issue in the assessment area, EPA (1993b)[11] recommends the use of Equation 3 to calculate the constant for soil loss resulting from erosion (*kse*).

$$kse = \frac{0.1 \cdot X_e \cdot SD \cdot ER}{BD \cdot Z_s} \cdot \frac{Kd_s \cdot BD}{\theta_{sw} + \left(Kd_s \cdot BD\right)} \qquad \text{(Equation 3)}$$

where

kse	=	PB-HAP compound soil loss constant due to soil erosion
0.1	=	Units conversion factor (1,000 g-kg/10,000 cm^2-m^2)
X_e	=	Unit soil loss (kg/m^2-yr)
SD	=	Sediment delivery ratio (unitless)
ER	=	Soil enrichment ratio (unitless)
Kd_s	=	Soil-water partition coefficient (mL water/g soil)
BD	=	Soil bulk density (g soil/cm^3 soil)
Z_s	=	Soil mixing zone depth (cm)
θ_{sw}	=	Soil volumetric water content (mL water/cm^3 soil)

Unit soil loss (X_e) is calculated by using the Universal Soil Loss Equation (USLE) (See Section 7.2). Soil bulk density (*BD*) is described in Section 2.4.2. Soil volumetric water content (θ_{sw}) is described in Section 2.5.4.

For additional information on addressing *kse*, EPA (1998)[1] recommends consulting the methodologies described in EPA NCEA document, *Methodology for Assessing Health Risks Associated with Multiple Pathways of Exposure to Combustor Emissions* (EPA 1998)[12].

2.2.3 PB-HAP compound Loss Constant Due to Runoff (*ksr*)

EPA (1998)[1] recommends that Equation 4 be used to calculate the constant for the loss of soil resulting from surface runoff (*ksr*).

$$ksr = \frac{RO}{\theta_{sw} \cdot Z_s} \cdot \left(\frac{1}{1 + \left(Kd_s \cdot BD / \theta_{sw} \right)} \right) \qquad \text{(Equation 4)}$$

where

ksr	=	PB-HAP compound loss constant due to runoff (yr^{-1})
RO	=	Average annual surface runoff from pervious areas (cm/yr)
θ_{sw}	=	Soil volumetric water content (mL water/cm^3 soil)
Z_s	=	Soil mixing zone depth (cm)
Kd_s	=	Soil-water partition coefficient (mL water/g soil)
BD	=	Soil bulk density (g soil/cm^3 soil)

Soil bulk density (*BD*) is described in Section 2.5.2. Soil volumetric water content (θ_{sw}) is described in Section 2.5.4.

2.2.4 PB-HAP compound Loss Constant Due to Leaching (*ksl*)

Losses of soil PB-HAP compounds due to leaching (*ksl*) depend on the amount of water available to generate leachate and soil properties such as bulk density, soil moisture, soil porosity, and soil sorption properties. EPA (1998)[1] recommends that Equation 5 be used to calculate the PB-HAP compound loss constant due to leaching (*ksl)* to account for runoff.

$$ksl = \frac{P + I - RO + E_v}{\theta_{sw} \cdot Z_s \cdot \left[1.0 + \left(BD \cdot Kd_s / \theta_{sw} \right) \right]} \qquad \text{(Equation 5)}$$

where

ksl	=	PB-HAP compound loss constant due to leaching (yr^{-1})
P	=	Average annual precipitation (cm/yr)
I	=	Average annual irrigation (cm/yr)
RO	=	Average annual surface runoff from pervious areas (cm/yr)
E_v	=	Average annual evapotranspiration (cm/yr)
θ_{sw}	=	Soil volumetric water content (mL water/cm^3 soil)

Z_s = Soil mixing zone depth (cm)
Kd_s = Soil-water partition coefficient (cm^3 water/g soil)
BD = Soil bulk density (g soil/cm^3 soil)

The average annual volume of water ($P + I - RO - E_v$) available to generate leachate is the mass balance of all water inputs and outputs from the area under consideration. These variables are described in Section 2.5.3. Soil bulk density (*BD*) is described in Section 2.5.2. Soil volumetric water content (θ_{sw}) is described in Section 2.5.4.

2.2.5 PB-HAP compound Loss Constant Due to Volatilization (*ksv*)

Semi-volatile and volatile PB-HAP compounds emitted in high concentrations may become adsorbed to soil particles and exhibit volatilization losses from soil. The loss of a PB-HAP compound from the soil by volatilization depends on the rate of movement of the PB-HAP compound to the soil surface, the chemical vapor concentration at the soil surface, and the rate at which vapor is carried away by the atmosphere (Jury 1986).[13]

EPA (1998)[1] recommends that in cases where high concentrations of volatile organic compounds are expected to be present in the soil that Equation 6A be used to calculate the constant for the loss of soil resulting from volatilization (*ksv)*.

$$ksv = \left(\frac{3.1536 \times 10^7 \cdot H}{Z_s \cdot Kd_s \cdot R \cdot T_a \cdot BD}\right) \cdot \left(\frac{D_a}{Z_s}\right) \cdot \left[1 - \left(\frac{BD}{\rho_{soil}}\right) - \theta_{sw}\right] \qquad \text{(Equation 6A)}$$

where

ksv = PB-HAP compound loss constant due to volatization (yr^{-1})
3.1536×10^7 = Units conversion factor (s/yr)
H = Henry's Law constant (atm-m^3/mol)
Z_s = Soil mixing zone depth (cm)
Kd_s = Soil-water partition coefficient (mL/g)
R = Universal gas constant (atm-m^3/mol-K)
T_a = Ambient air temperature (K) = 298.1 K
BD = Soil bulk density (g soil/cm^3 soil) = 1.5 g/cm^3
D_a = Diffusivity of PB-HAP compound in air (cm^2/s)
θ_{sw} = Soil volumetric water content (mL/cm^3 soil) = 0.2 mL/cm^3
ρ_{soil} = Solids particle density (g/cm^3) = 2.7 g/cm^3

The gas-phase mass transfer coefficient, K_t, based on general soil properties, can also be written as follows (Hillel 1980; Miller and Gardiner 1998)[14]:

$$K_t = \frac{D_a \cdot \theta_v}{Z_s} \qquad \text{(Equation 6B)}$$

where

K_t = Gas phase mass transfer coefficient (cm/s)
Z_s = Soil mixing zone depth (cm)
D_a = Diffusivity of PB-HAP compound in air (cm^2/s)
θ_v = Soil void fraction (cm^3/cm^3)

The soil void fraction (θ_v) is the volumetric fraction of a soil that does not contain solids or water and can be expressed as:

$$\theta_v = 1 - \left(\frac{BD}{\rho_{soil}}\right) - \theta_{sw} \qquad \text{(Equation 6C)}$$

where

θ_v = Soil void fraction (cm^3/cm^3)
θ_{sw} = Soil volumetric water content (mL water/cm^3 soil) = 0.2 mL/cm^3
BD = Soil bulk density (g/cm^3) = 1.5 g/cm^3
ρ_{soil} = Solids particle density (g/cm^3) = 2.7 g/cm^3

The expression containing bulk density (*BD*) divided by solids particle density (ρ_{soil}) gives the volume of soil occupied by pore space or voids (Miller and Gardiner 1998).[14] Soil bulk density is affected by the soil structure, such as looseness or compaction of the soil, depending on the water and clay content of the soil (Hillel 1980)[14]; a range of 0.83 to 1.84 was originally cited in Hoffman and Baes (1979).[15] A default soil bulk density value of 1.5 g/cm^3 is recommended based on a mean value for loam soil from Carsel et al. (1988).[16] Blake and Hartge (1996)[17] and Hillel (1980)[14] both suggests that the mean density of solid particles is about 2.7 gm/cm^3. The soil water content depends on both the available water and the soil structure of a particular soil. Values for θ_{sw} range from 0.03 to 0.40 mL/cm^3 depending on soil type (Hoffman and Baes 1979).[15] The lower values are typical of sandy soils, which cannot retain much water; the higher values are typical of soils such as clay or loam soils which can retain water. A mid-point default value of 0.2 mL water/cm^3 soil is recommended as a default in the absence of site-specific information. However, since the soil water content of soil is unique for each soil type, site-specific information is highly recommended.

2.3 Calculating the Deposition Term (*Ds*)

EPA (1998)[1] recommends that Equation 7 be used to calculate the deposition term *(Ds).*

$$Ds = \left[\frac{100 \cdot Q}{Z_s \cdot BD}\right] \cdot \left[F_v \cdot \left(Dydv + Dywv\right) + \left(Dydp + Dywp\right) \cdot \left(1 - F_v\right)\right] \qquad \text{(Equation 7)}$$

where

Ds = Deposition term (mg PB-HAP compound/kg soil/yr)
100 = Units conversion factor (mg-m^2/kg-cm^2)
Q = PB-HAP compound emission rate (g/s)
Z_s = Soil mixing zone depth (cm)

BD = Soil bulk density (g soil/cm^3 soil)
F_v = Fraction of PB-HAP compound air concentration in vapor phase (unitless)
Dydv = Unitized yearly average dry deposition from vapor phase (s/m^2-yr)
Dywv = Unitized yearly average wet deposition from vapor phase (s/m^2-yr)
Dydp = Unitized yearly average dry deposition from particle phase (s/m^2-yr)
Dywp = Unitized yearly average wet deposition from particle phase (s/m^2-yr)

2.4 Universal Soil Loss Equation (USLE)

EPA (1998)[1] recommends that the universal soil loss equation (USLE) be used to calculate the unit soil loss (X_e). This equation is further described in Section 7.2.

2.5 Site-Specific Parameters for Calculating Cumulative Soil Concentration

Calculating average soil concentration over the exposure duration (*Cs*) requires the use of site-specific parameters including the following:

- Soil mixing zone depth (Z_s)
- Soil bulk density (*BD*)
- Available water ($P + I - RO - E_v$)
- Soil volumetric water content (q_{sw})

Determination of values for these parameters is further described in the following subsections.

2.5.1 Soil Mixing Zone Depth (Z_s)

When exposures to PB-HAP compounds in soils are modeled, the depth of contaminated soils is important in calculating the appropriate soil concentration. PB-HAP compounds deposited onto soil surfaces may be moved into lower soil profiles by tilling, whether manually in a garden or mechanically in a large field.

EPA (1998)[1] recommends the following values for the soil mixing zone depth (Z_s):

- 2 cm for untilled soils; and
- 20 cm for tilled soils.

The assumption made to determine the value of Z_s may affect the outcome of the risk assessment, because soil concentrations that are based on soil depth are used to calculate exposure via several pathways: (1) ingestion of plants contaminated by root uptake; (2) direct ingestion of soil by humans, cattle, swine, or chicken; and (3) surface runoff into water bodies.

2.5.2 Soil Dry Bulk Density (*BD*)

Soil dry bulk density (*BD*) is the ratio of the mass of soil to its total volume. EPA (1998)[1] recommends the value of 1.50 g/cm^3 for the soil dry bulk density (*BD*). EPA (1994c)[18] recommended that wet soil bulk density be determined by weighing a thin-walled, tube soil sample (e.g., a Shelby tube) of known volume and subtracting the tube weight (ASTM Method

D2937).[19] Moisture content can then be calculated (ASTM Method 2216)[20] to convert wet soil bulk density to dry soil bulk density.

2.5.3 Available Water ($P + I - RO - E_v$)

The average annual volume of water available ($P + I - RO - E_v$) for generating leachate is the mass balance of all water inputs and outputs from the area under consideration. A wide range of values for these site-specific parameters may apply in the various EPA regions.

The average annual precipitation (P), irrigation (I), runoff (RO), and evapotranspiration (E_v) rates and other climatological data may be obtained from either data recorded on site or from the Station Climatic Summary for a nearby airport.

Meteorological variables such as the evapotranspiration rate (E_v) and the runoff rate (RO) may also be found in resources such as Geraghty, Miller, van der Leeden, and Troise (1973).[21] Surface runoff may also be estimated by using the Curve Number Equation developed by the U.S. Soil Conservation Service (EPA 1990).[4] EPA (1985)[22] cited isopleths of mean annual cropland runoff corresponding to various curve numbers developed by Stewart, Woolhiser, Wischmeier, Caro, and Frere (1975).[23] Curve numbers are assigned to an area on the basis of soil type, land use or cover, and the hydrologic conditions of the soil (EPA 1990).[4]

Using these different references, however, introduces uncertainties and limitations. For example, Geraghty, Miller, van der Leeden, and Troise (1973)[21] presented isopleths for annual surface water contributions that include interflow and ground water recharge. As noted in EPA (1994a)[8], these values are recommended to be adjusted downward to reflect surface runoff only. EPA (1994a)[8] recommended that these values be reduced by 50 percent.

2.5.4 Soil Volumetric Water Content (θ_{sw})

The soil volumetric water content (θ_{sw}) depends on the available water and the soil structure. A wide range of values for these variables may apply in the various EPA regions. EPA (1998)[1] recommends a value for θ_{sw} of 0.2 ml/cm^3.

3.0 Calculation of PB-HAP Compound Concentrations in Produce

Indirect exposure resulting from ingestion of produce depends on the total concentration of PB-HAP compounds in the leafy, fruit, and tuber portions of the plant. Because of general differences in contamination mechanisms, consideration of indirect exposure separates produce into two broad categories: aboveground produce and belowground produce. In addition, aboveground produce can be further subdivided into exposed and protected aboveground produce for consideration of contamination as a result of indirect exposure.

Aboveground Produce

Aboveground exposed produce is assumed to be contaminated by three possible mechanisms:

- **Direct deposition of particles—**wet and dry deposition of particle phase PB-HAP compounds on the leaves and fruits of plants (Section 3.1).

- **Vapor transfer—**uptake of vapor phase PB-HAP compounds by plants through their foliage (Section 3.2).
- **Root uptake—**root uptake of PB-HAP compounds available from the soil and their transfer to the aboveground portions of the plant (Section 3.3).

The total PB-HAP compound concentration in aboveground exposed produce is calculated as a sum of contamination occurring through all three of these mechanisms. However, edible portions of aboveground protected produce, such as peas, corn, and melons, are covered by a protective covering; hence, they are protected from contamination through deposition and vapor transfer. Therefore, root uptake of PB-HAP compounds is the primary mechanism through which aboveground protected produce becomes contaminated (Section 3.3).

Belowground Produce

For belowground produce, contamination is assumed to occur only through one mechanism – root uptake of PB-HAP compounds available from soil (Section 3.3). Contamination of belowground produce via direct deposition of particles and vapor transfer are not considered because the root or tuber is protected from contact with contaminants in the vapor phase.

3.1 Aboveground Produce Concentration Due to Direct Deposition (*Pd*)

EPA (1998)[1] recommends the use of Equation 8 to calculate PB-HAP compound concentration in exposed and aboveground produce due to direct deposition.

$$Pd = \frac{1{,}000 \cdot Q \cdot \left(1 - F_v\right) \cdot \left[Dydp + \left(Fw \cdot Dywp\right)\right] \cdot Rp \cdot \left[1.0 - \exp\left(-kp \cdot T_p\right)\right]}{Yp \cdot kp} \qquad \text{(Equation 8)}$$

where

Pd	=	Plant (aboveground produce) concentration due to direct (wet and dry) deposition (mg PB-HAP compound/kg DW)
1,000	=	Units conversion factor (mg/g)
Q	=	PB-HAP compound emission rate (g/s)
F_v	=	Fraction of PB-HAP compound air concentration in vapor phase (unitless)
Dydp	=	Unitized yearly average dry deposition from particle phase (s/m^2-yr)
Fw	=	Fraction of PB-HAP compound wet deposition that adheres to plant surfaces (unitless)
Dywp	=	Unitized yearly wet deposition from particle phase (s/m^2-yr)
Rp	=	Interception fraction of the edible portion of plant (unitless)
kp	=	Plant surface loss coefficient (yr^{-1})
Tp	=	Length of plant exposure to deposition per harvest of the edible portion of the *i*th plant group (yr)
Yp	=	Yield or standing crop biomass of the edible portion of the plant (productivity) (kg DW/m^2)

3.1.1 Interception Fraction of the Edible Portion of Plant (*Rp*)

EPA (1998)[1] recommends the use of the weighted average *Rp* value of 0.39 as a default *Rp* value because it represents the most current parameters including standing crop biomass and relative ingestion rates.

3.1.2 Plant Surface Loss Coefficient (*kp*)

EPA (1998)[1] recommends use of a plant surface loss coefficient (*kp*) value of 18. The primary uncertainty associated with this variable is that the calculation of *kp* does not consider chemical degradation processes. However, information regarding chemical degradation of contaminants on plant surfaces is limited. The inclusion of chemical degradation processes would result in decreased half-life values and thereby increase *kp* values. Note that effective plant concentration decreases as *kp* increases. Therefore, use of a *kp* value that does not consider chemical degradation processes is protective.

3.1.3 Length of Plant Exposure to Deposition per Harvest of Edible Portion of Plant (*Tp*)

This value represents the time required from when a plant first emerges until harvest. EPA (1998)[1] recommends using a *Tp* value of 0.164 year as the best available default value. The primary uncertainty associated with the use of this value is that it is based on the growing season for hay rather than aboveground produce. The average period between successive hay harvests (60 days) may not reflect the length of the growing season or the period between successive harvests for aboveground produce at specific sites. To the extent that information documenting the growing season or period between successive harvests for aboveground produce is available, this information may be used to estimate a site-specific *Tp* value. Calculated plant concentrations will be affected most if the site-specific value of *Tp* is significantly less than 60 days.

3.1.4 Standing Crop Biomass (Productivity) (*Yp*)

EPA (1998)[1] recommends the use of the weighted average *Yp* value of 2.24 as a default *Yp* value based on this value representing the most complete and thorough information available. The primary uncertainty associated with this variable is that the harvest yield (*Yh*) and area planted (*Ah*) may not reflect site-specific conditions. To the extent to which site-specific information is available, the magnitude of the uncertainty introduced by the default *Yp* value can be estimated.

3.2 Aboveground Produce Concentration Due to Air-to-Plant Transfer (*Pv*)

The methodology used to estimate PB-HAP compound concentration in exposed and aboveground produce due to air-to-plant transfer (*Pv*) considers limitations of PB-HAP compounds concentrations to transfer from plant surfaces to the inner portions of the plant. These limitations result from mechanisms responsible for inhibiting the transfer of the lipophilic PB-HAP compound (e.g., the shape of the produce) and the removal of the PB-HAP compounds from the edible portion of the produce (e.g., washing, peeling, and cooking). EPA (1998)[1] recommends the use of Equation 9 to calculate aboveground produce concentration due to air-to-plant transfer (*Pv*).

$$Pv = Q \cdot F_v \cdot \frac{Cyv \cdot Bv_{ag} \cdot VG_{ag}}{\rho_a} \qquad \text{(Equation 9)}$$

where

Pv	=	Concentration of PB-HAP compound in the plant resulting from air-to-plant transfer (µg PB-HAP compound/g DW)
Q	=	PB-HAP compound emission rate (g/s)
F_v	=	Fraction of PB-HAP compound air concentration in vapor phase (unitless)
Cyv	=	Unitized yearly average air concentration from vapor phase (µg-s/g-m^3)
Bv_{ag}	=	PB-HAP compound air-to-plant biotransfer factor ([mg PB-HAP compound/g DW plant]/[mg PB-HAP compound/g air]) (unitless)
VG_{ag}	=	Empirical correction factor for aboveground produce (unitless)
ρ_a	=	Density of air (g/m^3)

As discussed below in Section 3.2.1, the parameter VG_{ag} is dependent on lipophilicity of the PB-HAP compound, and assigned a value of 0.01 for lipophilic PB-HAP compounds (log K_{ow} greater than 4) or a value of 1.0 for PB-HAP compounds with a log K_{ow} less than 4.

Empirical Correction Factor for Aboveground Produce (VG_{ag})

The parameter VG_{ag} has been incorporated into Equation 9 to address the potential overestimation for lipophilic PB-HAP compounds to be transferred to the inner portions of bulky produce, such as apples. Because of the protective outer skin, size, and shape of bulky produce, transfer of lipophilic PB-HAP compounds (log K_{ow} greater than 4) to the center of the produce is not as likely as for non-lipophilic PB-HAP compounds and, as a result, the inner portions will be less affected. EPA (1998)[1] recommends the following empirical VG_{ag} values for aboveground produce:

- 0.01 for lipophilic PB-HAP compounds (log K_{ow} greater than 4); and
- 1.0 for PB-HAP compounds with a log K_{ow} less than 4 (these PB-HAP compounds are assumed pass more easily through the skin of produce).

Uncertainty may be introduced by the assumption of VG_{ag} values for leafy vegetables (such as lettuce) and for legumes (such as snap beans). Underestimation may be introduced by assuming a VG_{ag} value of 0.01 for legumes and leafy vegetables because these species often have a higher ratio of surface area to mass than other bulkier fruits and fruiting vegetables, such as tomatoes.

3.3 Produce Concentration Due to Root Uptake (*Pr*)

Root uptake of contaminants from soil may also result in PB-HAP compound concentrations in aboveground exposed produce, aboveground protected produce, and belowground produce. EPA (1998)[1] recommends the use of Equations 10A and 10B to calculate PB-HAP compound concentration aboveground and belowground produce due to root uptake (*Pr*).

Exposed and protected aboveground produce:

$$Pr = Cs \cdot Br \qquad \text{(Equation 10A)}$$

Belowground produce:

$$Pr = \frac{Cs \cdot RCF \cdot VG_{rootveg}}{Kd_s \cdot 1\ kg\ /\ L} \qquad \text{(Equation 10B)}$$

where

Pr	=	Concentration of PB-HAP compound in produce due to root uptake (mg/kg)
Br	=	Plant-soil bioconcentration factor for produce (unitless)
$VG_{rootveg}$	=	Empirical correction factor for belowground produce (unitless)
Kd_s	=	Soil-water partition coefficient (L/kg)
Cs	=	Average soil concentration over exposure duration (mg PB-HAP compound/kg soil)
RCF	=	Root concentration factor (unitless)

Equation 10A is appropriate for evaluation of exposed and protected aboveground produce; however, it may not be appropriate for soil-to-belowground plant transfers. For belowground produce, Equation 10B includes a root concentration factor (RCF) developed by Briggs et al. (1982).[24] RCF is the ratio of PB-HAP compound concentration in the edible root to the PB-HAP compound concentration in the soil water. Since Briggs et al. (1982)[24] conducted their experiments in a growth solution, the PB-HAP compound soil concentration (Cs) must be divided by the PB-HAP compound-specific soil-water partition coefficient (Kd_s) (EPA 1994b).[25]

Similar to VG_{ag} and as discussed in Section 3.2.1, $VG_{rootveg}$ is based on the lipophilicity of the PB-HAP compound. EPA (1998)[1] recommends the following empirical values for $VG_{rootveg}$:

- 0.01 for lipophilic PB-HAP compounds (log K_{ow} greater than 4) based on root vegetables like carrots and potatoes; and
- 1.0 for PB-HAP compounds with a log K_{ow} less than 4.

4.0 Calculation of PB-HAP Compound Concentrations in Beef and Dairy Products

PB-HAP compound concentrations in beef tissue and milk products are estimated on the basis of the amount of PB-HAP compounds that cattle are assumed to consume through their diet. The cattle's diet is assumed to consist of forage (primarily pasture grass and hay); silage (forage that has been stored and fermented), and grain. Additional contamination may occur through the cattle’s ingestion of soil. The total PB-HAP compound concentration in the feed items (e.g., forage, silage, and grain) is calculated as a sum of contamination occurring through the following mechanisms:

- **Direct deposition of particles**—wet and dry deposition of particle phase PB-HAP compounds onto forage and silage (Section 4.1).

- **Vapor transfer**—uptake of vapor phase PB-HAP compounds by forage and silage through foliage (Section 4.2).

- **Root uptake**—root uptake of PB-HAP compounds available from the soil and their transfer to the aboveground portions of forage, silage, and grain (Section 4.3).

Feed items consumed by animals can be classified as exposed and protected, depending on whether it has a protective outer covering. Because the outer covering on the protected feed acts as a barrier, it is assumed that there is negligible contamination of protected feed through deposition of particles and vapor transfer. In this analysis, grain is classified as protected feed. As a result, grain contamination is assumed to occur only through root uptake. Contamination of exposed feed items, including forage and silage, is assumed to occur through all three mechanisms.

The amount of grain, silage, forage, and soil consumed is assumed to vary between dairy and beef cattle. Sections 4.4 (beef) and 4.5 (dairy) describe methods for estimating consumption rates and subsequent PB-HAP compound concentrations in cattle. EPA (1998)[1] recommends that 100 percent of the plant materials eaten by cattle be assumed to have been grown on soil contaminated by emission sources. Therefore, 100 percent of the feed items consumed are assumed to be contaminated.

4.1 Forage and Silage Concentrations Due to Direct Deposition (*Pd*)

PB-HAP compound concentrations in forage and silage result from wet and dry deposition onto exposed plant surfaces; similar to aboveground produce (Section 3.1). Equation 8, described in Section 3.1, is recommended for calculation of PB-HAP compound concentrations resulting from direct deposition onto plant surfaces of leafy plants and exposed produce (*Pd*). Therefore, EPA (1998)[1] recommends that Equation 8 also be used in calculating forage and silage concentrations due to direct deposition.

4.1.1 Interception Fraction of the Edible Portion of Plant (*Rp*)

EPA (1998)[1] recommends use of the *Rp* value of 0.5 for forage and the *Rp* value of 0.46 for silage. Note that the empirical relationships used to develop the default values for silage may not accurately represent site-specific silage types. However, the range of empirical constants used to develop the default value for forage is fairly small, and therefore the use of the midpoint should not significantly affect the *Rp* value and the resulting estimate of plant PB-HAP compound concentration.

4.1.2 Plant Surface Loss Coefficient (*kp*)

Section 3.1.2 presents the recommended value for plant surface loss coefficient *kp* for aboveground produce. The *kp* factor is derived in exactly the same manner for cattle forage and silage, and the uncertainties of *kp* for cattle forage and silage are similar to its uncertainties for aboveground produce.

4.1.3 Length of Plant Exposure to Deposition per Harvest of the Edible Portion of Plant (*Tp*)

As discussed in Section 3.1.3, *Tp* is treated as a constant, based on the average period between successive hay harvests. This periodrepresents the length of time that aboveground vegetation (in this case, hay) would be exposed to particle deposition before being harvested. EPA (1998)[1] recommends the following *Tp* values: 0.12 year for forage; and 0.16 year for silage. The primary uncertainties associated with *Tp* are similar to those for aboveground produce, and are discussed in Section 3.1.3.

4.1.4 Standing Crop Biomass (Productivity) (Yp)

As discussed in Section 3.1.4, the best estimate of *Yp* is productivity, requires consideration of dry harvest yield (*Yh*) and area harvested (*Ah*). EPA (1998)[1] recommends that forage *Yp* be calculated as a weighted average of the calculated pasture grass and hay *Yp* values. Weightings are assumed to be 0.75 for forage and 0.25 for hay, based on the fraction of a year that cattle are assumed to be pastured and eating grass (9 months per year) or not pastured and fed hay (3 months per year). The resulting value of 0.24 kg DW/m^2 is recommended as the *Yp* for forage. For silage, EPA (1998)[1] recommends that a production-weighted U.S. average *Yp* of 0.8 kg DW/m^2 be assumed. The primary uncertainty associated with this variable is that the harvest yield (*Yh*) and area planted (*Ah*) may not reflect site-specific conditions. To the extent that site-specific information is available, the magnitude of the uncertainty introduced by the default *Yp* value can be estimated. In addition, the weightings assumed in this discussion for the amount of time that cattle are pastured (and foraging) or stabled (and being fed silage) should be adjusted to reflect site-specific conditions, as appropriate.

4.2 Forage and Silage Concentrations Due to Air-to-Plant Transfer (*Pv*)

PB-HAP compound concentration in aboveground produce resulting from air-to-plant transfer (*Pv*), is calculated by using Equation 9 (Section 3.2). *Pv* is calculated for cattle forage and silage similarly to the way that it is calculated for aboveground produce. A detailed discussion of *Pv* is provided in Section 3.2. Differences in VG_{ag} values for forage and silage, as compared to the values for aboveground produce described in Section 3.2.1, are presented below in Section 4.2.1.

Empirical Correction Factor for Forage and Silage (VG_{ag})

EPA (1998)[1] recommends the use of VG_{ag} values of 1.0 for forage and 0.5 for silage. As discussed, the primary uncertainty associated with this variable is the lack of specific information on the proportions of each vegetation type of which silage may consist, leading to the default assumption of 0.5.

4.3 Forage, Silage, and Grain Concentrations Due to Root Uptake (*Pr*)

PB-HAP compound concentration in aboveground and belowground produce resulting from root uptake is calculated by using Equations 10A and 10B (Section 3.3). *Pr* is also calculated for cattle forage, silage, and grain in exactly the same way that it is calculated for aboveground produce. A detailed discussion describing calculation of *Pr* is provided in Section 3.3.

4.4 Beef Concentration Resulting from Plant and Soil Ingestion (A_{beef})

EPA (1998)[1] recommends that PB-HAP compound concentration in beef tissue (A_{beef}) be calculated by using Equation11. Equation 11 calculates the daily amount of a PB-HAP compound that is consumed by cattle through the ingestion of contaminated feed items (plant) and soil. The equation includes biotransfer and metabolism factors to transform the daily animal intake of a PB-HAP compound (mg/day) into an animal PB-HAP compound tissue concentration (mg PB-HAP compound/kg tissue).

$$A_{beef} = \left(\sum\left(F_i \cdot Qp_i \cdot P_i\right) + Qs \cdot Cs \cdot Bs\right) \cdot Ba_{beef} \cdot MF \qquad \text{(Equation 11)}$$

where

A_{beef}	=	Concentration of PB-HAP compound in beef (mg PB-HAP compound/kg FW tissue)
F_i	=	Fraction of plant type *i* grown on contaminated soil and ingested by the animal (cattle) (unitless)
Qp_i	=	Quantity of plant type *i* eaten by the animal (cattle) per day (kg DW plant/day)
P_i	=	Concentration of PB-HAP compound in each plant type *i* eaten by the animal (cattle) (mg/kg DW)
Qs	=	Quantity of soil eaten by the animal (cattle) each day (kg/day)
Cs	=	Average soil concentration over exposure duration (mg PB-HAP compound/kg soil)
Bs	=	Soil bioavailability factor (unitless)
Ba_{beef}	=	PB-HAP compound biotransfer factor for beef (day/kg FW tissue)
MF	=	Metabolism factor (unitless)

The parameters F_i, Qp_i, P_i, Qs, Cs, Bs, and MF are described in Sections 4.4.1 through 4.4.7, respectively.

4.4.1 Fraction of Plant Type *i* Grown on Contaminated Soil and Eaten by the Animal (Cattle)(F_i)

EPA (1998)[1] recommends that 100 percent of the plant materials eaten by cattle be assumed to have been grown on soil contaminated by the emission sources being evaluated and therefore recommends a default value of 1.0 for F_i.

4.4.2 Quantity of Plant Type *i* Eaten by the Animal (Cattle) Each Day (Qp_i)

EPA (1998)[1] recommends the following beef cattle ingestion rates of forage, silage, and grain. These values are based on the total daily intake rate of about 12 kg DW/day.

- Forage = 8.8 kg DW/day;
- Silage = 2.5 kg DW/day; and
- Grain = 0.47 kg DW/day.

The principal uncertainty associated with Qp_i is the variability between forage, silage, and grain ingestion rates for cattle.

4.4.3 Concentration of PB-HAP compound in Plant Type *i* Eaten by the Animal (Cattle) (P_i)

The total PB-HAP compound concentration in forage, silage, and grain are recommended to be calculated by using Equation 12. Values for *Pd*, *Pv*, and *Pr* can be derived for each type of feed by using Equations 8, 17, and 10, respectively.

$$P_i = \sum_i \left(Pd + Pv + Pr \right) \qquad \text{(Equation 12)}$$

where

P_i	=	Concentration of PB-HAP compound in each plant type *i* eaten by the animal (mg PB-HAP compound/kg DW)
Pd	=	Plant concentration due to direct deposition (mg PB-HAP compound/kg DW)
Pv	=	Plant concentration due to air-to-plant transfer (mg PB-HAP compound/kg DW)
Pr	=	Plant concentration due to root uptake (mg PB-HAP compound/kg DW)

4.4.4 Quantity of Soil Eaten by the Animal (Cattle) Per Day (*Qs*)

Additional cattle contamination occurs through ingestion of soil. EPA (1998)[1] recommends a value of 0.5 kg/day for the quantity of soil ingested by the animal (cattle).

4.4.5 Average Soil Concentration Over Exposure Duration (*Cs*)

PB-HAP compound concentration in soil is recommended to be calculated as discussed in Section 2.1, by using Equations 1A, 1B, and 1C.

4.4.6 Soil Bioavailability Factor (*Bs*)

The efficiency of transfer from soil may differ from efficiency or transfer from plant material for some PB-HAP compounds. If the transfer efficiency is lower for soils, than this ratio would be less than 1.0. If it is equal or greater than that of vegetation, the *Bs* value would be equal to or greater than 1.0. Until more PB-HAP compound-specific data becomes available for this parameter, EPA (1998)[1] recommends a default value of 1 for *Bs*.

4.4.7 Metabolism Factor (*MF*)

The metabolism factor (*MF*) represents the estimated amount of PB-HAP compound that remains in fat and muscle. EPA (1998)[1] recommends a *MF* of 1.0 for all PB-HAP compounds. Considering the recommended values for this variable, *MF* has no quantitative effect on A_{beef}. *MF* applies only to mammalian species, including beef cattle, dairy cattle, and pigs. It does not relate to metabolism in produce, chicken, or fish. In addition, since exposures evaluated in this chapter are intake driven, the use of a metabolism factor applies only to ingestion of beef, milk, and pork. In summary, use of a *MF* does not apply for direct exposures to soil or water, or to ingestion of produce, chicken, or fish.

4.5 PB-HAP compound Concentration In Milk Due to Plant and Soil Ingestion (A_{milk})

Equation 11 (Section 4.4) describes the calculation of PB-HAP compound concentrations in beef cattle (A_{beef}). Equation 11 can be modified to calculate PB-HAP compound milk concentrations (A_{milk}), as follows:

$$A_{milk} = \left(\sum\left(F_i \cdot Q_{pi} \cdot P_i\right) + Qs \cdot Cs \cdot Bs\right) \cdot Ba_{milk} \cdot MF \qquad \text{(Equation 13)}$$

where

A_{milk}	=	Concentration of PB-HAP compound in milk (mg PB-HAP compound/kg milk)
F_i	=	Fraction of plant type *i* grown on contaminated soil and ingested by the animal (dairy cattle) (unitless)
Qp_i	=	Quantity of plant type *i* eaten by the animal (dairy cattle) each day (kg DW plant/day)
P_i	=	Concentration of PB-HAP compound in plant type *i* eaten by the animal (dairy cattle) (mg/kg DW)
Qs	=	Quantity of soil eaten by the animal (dairy cattle) each day (kg soil/day)
Cs	=	Average soil concentration over exposure duration (mg PB-HAP compound/kg soil)
Bs	=	Soil bioavailability factor (unitless)
Ba_{milk}	=	PB-HAP compound biotransfer factor for milk (day/kg WW tissue)
MF	=	Metabolism factor (unitless)

EPA (1998)[1] recommends the use of Equation 13 to estimate dairy cattle milk PB-HAP compound concentration (A_{milk}). The discussion in Section 4.4 of the variables F_i, Qp_i, P_i, Qs, Cs, and MF for beef cattle generally applies to the corresponding variables for dairy cattle. However, there are some differences in assumptions made for dairy cattle; these differences are summarized in the following subsections.

4.5.1 Fraction of Plant Type *i* Grown on Contaminated Soil and Eaten by the Animal (Dairy Cattle) (F_i)

The calculation of F_i for dairy cattle is identical to that for beef cattle (Section 4.4.1).

4.5.2 Quantity of Plant Type *i* Eaten by the Animal (Dairy Cattle) Per Day (Qp_i)

As discussed in Section 4.4.2, the daily quantity of forage, silage, and grain feed consumed by cattle is estimated for each category of feed material. However, daily ingestion rates for dairy cattle are estimated differently than for beef cattle. The daily quantity of feed consumed by cattle is recommended to be estimated on a dry weight basis for each category of plant feed.

EPA (1998)[1] recommends a default total ingestion rate of 20 kg DW/day for dairy cattle, divided among forage, silage, and grain, as follows:

- Forage = 13.2 kg DW/day;
- Silage = 4.1 kg DW/day; and
- Grain = 3.0 kg DW/day

Uncertainties associated with the estimation of Qp_i include the estimation of forage, grain, and silage ingestion rates, which will vary from site to site. The assumption of uniform contamination of plant materials consumed by cattle also introduces uncertainty.

4.5.3 Concentration of PB-HAP compound in Plant Type *i* Eaten by the Animal (Dairy Cattle) (P_i)

The estimation of P_i for dairy cattle is identical to that for beef cattle (Section 4.4.3).

4.5.4 Quantity of Soil Eaten by the Animal (Dairy Cattle) Per Day (*Qs*)

As discussed in Section 4.4.4, contamination of dairy cattle also results from the ingestion of soil. EPA (1998)[1] recommends a soil ingestion rate of 0.4 kg/day for dairy cattle. Uncertainties associated with *Qs* include the lack of current empirical data to support soil ingestion rates for dairy cattle. The assumption of uniform contamination of soil ingested by cattle also adds uncertainty.

4.5.5 Average Soil Concentration Over Exposure Duration (*Cs*)

The calculation of *Cs* for dairy cattle is the same as for beef cattle (Section 4.4.5).

4.5.6 Soil Bioavailability Factor (*Bs*)

The calculation of *Bs* for dairy cattle is the same as for beef cattle (Section 4.4.6).

4.5.7 Metabolism Factor (*MF*)

The recommended values for *MF* are identical to those recommended for beef cattle (Section 4.4.7).

5.0 Calculation of PB-HAP Compound Concentrations in Pork

PB-HAP compound concentrations in pork tissue are estimated on the basis of the amount of PB-HAP compounds that swine are assumed to consume through their diet; assumed to consist of silage and grain. Additional PB-HAP compound contamination of pork tissue may occur through the ingestion of soil by swine.

5.1 Concentration of PB-HAP compound In Pork

Equation 11 (Section 4.4) describes the calculation of PB-HAP compound concentration in beef cattle (A_{beef}). Equation 11 can be modified to calculate PB-HAP compound concentrations in swine (A_{pork}), as follows:

$$A_{pork} = \left(\sum\left(F_i \cdot Q_{pi} \cdot P_i\right) + Qs \cdot Cs \cdot Bs\right) \cdot Ba_{pork} \cdot MF \qquad \text{(Equation 14)}$$

where

A_{pork} = Concentration of PB-HAP compound in pork (mg PB-HAP compound/kg FW tissue)
F_i = Fraction of plant type *i* grown on contaminated soil and ingested by the animal (swine)(unitless)
Qp_i = Quantity of plant type *i* eaten by the animal (swine) each day (kg DW plant/day)
P_i = Concentration of PB-HAP compound in plant type *i* eaten by the animal (swine) (mg/kg DW)
Qs = Quantity of soil eaten by the animal (swine) (kg/day)
Cs = Average soil concentration over exposure duration (mg PB-HAP compound/kg soil)
Bs = Soil bioavailability factor (unitless)
Ba_{pork} = PB-HAP compound biotransfer factor for pork (day/kg FW tissue)
MF = Metabolism factor (unitless)

EPA (1998)[1] recommends that Equation 14 be used to calculate PB-HAP compound pork concentrations (A_{pork}). The discussion in Section 4.5 of the variables F_i, Qp_i, P_i, Qs, Cs and MF for beef cattle generally applies to the corresponding variables for pork. However, different assumptions are made for pork. These differences are summarized in the following subsections.

5.1.1 Fraction of Plant Type *i* Grown on Contaminated Soil and Eaten by the Animal (Swine) (F_i)

The calculation of F_i for pork is identical to that for beef cattle (Section 4.4.1).

5.1.2 Quantity of Plant Type *i* Eaten by the Animal (Swine) Each Day (Qp_i)

As discussed in Section 4.4.2, the daily quantity of forage, silage, and grain feed consumed by beef cattle is estimated for each category of feed material. However, daily ingestion rates for pork are estimated differently than for beef cattle. Because swine are not grazing animals, they are assumed not to eat forage, and EPA (1998)[1] recommends that the daily quantity of plant feeds (kilograms of DW) consumed by swine be estimated for each category of plant feed.

EPA (1990)[4] and NC DEHNR (1997)[26] did not differentiate between subsistence and typical hog farmers as for cattle. EPA (1990)[4] and NC DEHNR (1997)[26] recommended grain and silage ingestion rates for swine as 3.0 and 1.3 kg DW/day, respectively. NC DEHNR (1997)[26] references EPA (1990)[4] as the source of these ingestion rates. EPA (1990)[4] reported total dry matter ingestion rates for hogs and lactating sows as 3.4 and 5.2 kg DW/day, respectively. EPA (1990)[4] cites Boone, Ng, and Palm (1981)[27] as the source of the ingestion rate for hogs, and NAS (1987)[28] as the source of the ingestion rate for a lactating sow. Boone, Ng, and Palm (1981)[27] reported a grain ingestion rate of 3.4 kg DW/day for a hog. NAS (1987)[28] reported an average ingestion rate of 5.2 kg DW/day for a lactating sow. EPA (1990)[4] recommended using the average of these two rates (4.3 kg DW/day). EPA (1990)[4] assumed that 70 percent of the swine diet is grain and 30 percent silage to obtain the grain ingestion rate of 3.0 kg DW/day and the silage ingestion rate of 1.3 kg DW/day. EPA (1990)[4] cited EPA (1982)[29] as the source of the grain and silage dietary fractions. EPA (1995)[30] recommended an ingestion rate of 4.7 kg DW/day for a swine, referencing NAS (1987).[28] NAS (1987)[28] reported an average daily intake of 4.36 kg DW/day for a gilt (young sow) and a average daily intake of 5.17 kg DW/day for a sow, which averages out to 4.7 kg/DW/day. Assuming the 70 percent grain to 30 percent silage diet noted above, estimated ingestion rates of 3.3 kg DW/day (grain) and 1.4 kg DW/day (silage) are derived.

EPA (1998)[1] recommends the use of the following Qp_i values for pork:

- Grain = 3.3 kg DW/day; and
- Silage = 1.4 kg DW/day.

Uncertainties associated with this variable include the variability of actual grain and silage ingestion rates from site to site. Site-specific data can be used to mitigate this uncertainty. In addition, the assumption of uniform contamination of plant materials consumed by swine produces some uncertainty.

5.1.3 Concentration of PB-HAP compound in Plant Type *i* Eaten by the Animal (Swine) (P_i)

The calculation of P_i for pork is identical to that for beef cattle (Section 4.4.3).

5.1.4 Quantity of Soil Eaten by the Animal (Swine) Each Day (Q_s)

As discussed in Section 4.4.4, additional contamination of swine results from ingestion of soil. EPA (1998)[1] recommends the following soil ingestion rate for swine: 0.37 kg DW/day. Uncertainties associated with this variable include the lack of current empirical data to support soil ingestion rates for swine, and the assumption of uniform contamination of soil ingested by swine.

5.1.5 Average Soil Concentration Over Exposure Duration (*Cs*)

The calculation of *Cs* for pork is the same as for beef cattle (Section 4.4.5).

5.1.6 Soil Bioavailability Factor (*Bs*)

The calculation of *Bs* for pork is the same as for beef cattle (Section 4.4.6)

5.1.7 Metabolism Factor (*MF*)

The recommended values for MF are identical to those recommended for beef cattle (Section 4.4.7).

6.0 Calculation of PB-HAP Compound Concentrations in Chicken and Eggs

Estimates of the PB-HAP compound concentrations in chicken and eggs are based on the amount of PB-HAP compounds that chickens consume through ingestion of grain and soil. The uptake of PB-HAP compounds via inhalation and via ingestion of water is assumed to be insignificant relative to other pathways. Chickens are assumed to be housed in a typical manner that allows contact with soil; and therefore, are assumed to consume 10 percent of their diet as soil. The remainder of the diet (90 percent) is assumed to consist of grain. Grain ingested by chickens is assumed to have originated from the exposure scenario location; therefore, 100 percent of the grain consumed is assumed to be contaminated. The uptake of PB-HAP compounds via ingestion of contaminated insects and other organisms (e.g., worms, etc.), which may also

contribute to the ingestion of PB-HAP compounds, is not accounted for in the equations and may be a limitation depending on the site-specific conditions under which the chickens are raised.

The PB-HAP compound concentration in grain is estimated by using the algorithm for aboveground produce described in Section 3. Grain is considered to be a feed item that is protected from deposition of particles and vapor transfer. As a result, only contamination due to root uptake of PB-HAP compounds is considered in the calculation of PB-HAP compound concentration in grain.

6.1 Concentration of PB-HAP compound in Chicken and Eggs

EPA (1998)[1] recommends the use of Equation 15 to calculate PB-HAP compound concentrations in chicken and eggs. It is recommended that PB-HAP compound concentrations in chicken and eggs be determined separately.

$$A_{chicken} \text{ or } A_{egg} = \left(\sum\left[F_i \cdot Qp_i \cdot P_i\right] + Qs \cdot Cs \cdot Bs\right) \cdot \left(Ba_{egg} \text{ or } Ba_{chicken}\right) \qquad \text{(Equation 15)}$$

where

$A_{chicken}$	=	Concentration of PB-HAP compound in chicken (mg PB-HAP compound/kg FW tissue)
A_{egg}	=	Concentration of PB-HAP compound in eggs (mg PB-HAP compound/kg FW tissue)
F_i	=	Fraction of plant type *i* (grain) grown on contaminated soil and ingested by the animal (chicken)(unitless)
Qp_i	=	Quantity of plant type *i* (grain) eaten by the animal (chicken) each day (kg DW plant/day)
P_i	=	Concentration of PB-HAP compound in plant type *i* (grain) eaten by the animal (chicken) (mg/kg DW)
Qs	=	Quantity of soil eaten by the animal (chicken) (kg/day)
Cs	=	Average soil concentration over exposure duration (mg PB-HAP compound/kg soil)
Bs	=	Soil bioavailability factor (unitless)
$Ba_{chicken}$	=	PB-HAP compound biotransfer factor for chicken (day/kg FW tissue)
Ba_{egg}	=	PB-HAP compound biotransfer factor for eggs (day/kg FW tissue)

EPA (1998)[1] describes determination of compound specific parameters $Ba_{chicken}$ and Ba_{egg}. The remaining parameters are discussed in the following subsections.

6.1.1 Fraction of Plant Type *i* Grown on Contaminated Soil and Eaten by the Animal (Chicken)(F_i)

The calculation of F_i for chicken is identical to that for beef cattle (Section 4.4.1).

6.1.2 Quantity of Plant Type *i* Eaten by the Animal (Chicken) Each Day (Qp_i)

Because chickens are not grazing animals, they are assumed not to eat forage. Chickens are assumed not to consume any silage. The daily quantity of plant feeds (kilograms of DW)

consumed by chicken only should be estimated for grain feed. EPA (1998)[1] recommends the use of the following ingestion rate (Qp_i): Grain = 0.2 kg DW/day. Uncertainties associated with this variable include the variability of actual grain ingestion rates from site to site. In addition, the assumption of uniform contamination of plant materials consumed by chicken produces some uncertainty.

6.1.3 Concentration of PB-HAP compound in Plant Type *i* Eaten by the Animal (Chicken) (P_i)

The total PB-HAP compound concentration is the PB-HAP compound concentration in grain and can be calculated by using Equation 16. Values for *Pr* can be derived by using Equation 10.

$$P_i = \sum_i \left(Pr \right) \qquad \text{(Equation 16)}$$

where

P_i = Concentration of PB-HAP compound in each plant type *i* eaten by the animal (mg PB-HAP compound/kg DW)
Pr = Plant concentration due to root uptake (mg PB-HAP compound/kg DW)

6.1.4 Quantity of Soil Eaten by the Animal (Chicken) Each Day (*Qs*)

PB-HAP compound concentration in chickens also results from intake of soil. As discussed earlier, chickens are assumed to consume 10 percent of their total diet as soil. EPA (1998)[1] recommends the following soil ingestion rate for chicken: 0.022 kg DW/day. Uncertainties associated with this variable include the lack of current empirical data to support soil ingestion rates for chicken, and the assumption of uniform contamination of soil ingested by chicken.

6.1.5 Average Soil Concentration Over Exposure Duration (*Cs*)

The calculation of *Cs* for chicken is the same as for beef cattle (Section 4.4.5).

6.1.6 Soil Bioavailability Factor (*Bs*)

The calculation of *Bs* for chicken is the same as for beef cattle (Section 4.4.6)

7.0 Calculation of PB-HAP Compound Concentrations in Drinking Water and Fish

PB-HAP compound concentrations in surface water are calculated for all water bodies selected for evaluation in the risk assessment; specifically, evaluation of the drinking water and/or fish ingestion exposure pathways. Mechanisms considered for determination of PB-HAP compound loading of the water column are:

(1) Direct deposition,
(2) Runoff from impervious surfaces within the watershed,
(3) Runoff from pervious surfaces within the watershed,
(4) Soil erosion over the total watershed,
(5) Direct diffusion of vapor phase PB-HAP compounds into the surface water, and
(6) Internal transformation of compounds chemically or biologically.

Other potential mechanisms may need consideration on a case-by-case basis (e.g., tidal influences), however, contributions from other potential mechanisms are assumed to be negligible in comparison with those being evaluated.

The USLE and a sediment delivery ratio are used to estimate the rate of soil erosion from the watershed. In the ISCST3 model, surface water concentration algorithms include a sediment mass balance, in which the amount of sediment assumed to be buried and lost from the water body is equal to the difference between the amount of soil introduced to the water body by erosion and the amount of suspended solids lost in downstream flow. As a result, the assumptions are made that sediments do not accumulate in the water body over time, and an equilibrium is maintained between the surficial layer of sediments and the water column. The total water column PB-HAP compound concentration is the sum of the PB-HAP compound concentration dissolved in water and the PB-HAP compound concentration associated with suspended solids. Partitioning between water and sediment varies with the PB-HAP compound. The total concentration of each PB-HAP compound is partitioned between the sediment and the water column. The assumptions for other multimedia models may differ.

To evaluate the PB-HAP compound loading to a water body from its associated watershed, it is recommended that the PB-HAP compound concentration in watershed soils be calculated. As described in Section 2, the equation for PB-HAP compound concentration in soil includes a loss term that considers the loss of contaminants from the soil after deposition. These loss mechanisms all lower the soil concentration associated with a specific deposition rate.

The ISCST3 model approach for modeling PB-HAP compound loading to a water body represents a simple steady-state model to solve for a water column in equilibrium with the upper sediment layer. This approach may be limited in addressing the dynamic exchange of contaminants between the water body and the sediments following changes in external loadings. While appropriate for calculating risk under long-term average conditions, the evaluation of complex water bodies or shorter term loading scenarios may be improved through the use of a dynamic modeling framework [e.g., Exposure Analysis Modeling System (EXAMS)]. Although typically more resource intensive, such analysis may offer the ability to refine modeling of contaminant loading to a water body. Additionally, the computations may better represent the exposure scenario being evaluated.

For example, EXAMS allows computations to be performed for each defined segment or compartment of a water body or stream. These compartments are considered physically homogeneous and are connected via advective and dispersive fluxes. Compartments can be defined as littoral, epilimnion, hypolimnion, or benthic. Such resolution also makes it possible to assign receptor locations specific to certain portions of a water body where evaluation of exposure is of greatest interest.

Some considerations regarding the selection and use of a dynamic modeling framework or simulation model to evaluate water bodies may include the following:

- Will a complex surface water modeling effort provide enhanced results over the use of the more simplistic steady-state equations;

- Are the resources needed to conduct, as well as review, a more complex modeling effort justified in comparison to the refinement to results provided;

- Has the model been used previously for regulatory purposes, and therefore, already has available documentation to support such uses;

- Can the model conduct steady-state and dynamic analysis; and

- Does the model require calibration with field data, and if so, are there sufficient quantity and quality of site-specific data available to support calibration.

7.1 Total PB-HAP compound Load to the Water Body (L_T)

EPA (1998)[1] recommends the use of Equation 17 to calculate the total PB-HAP compound load to a water body (L_T).

$$L_T = L_{DEP} + L_{dif} + L_{RI} + L_R + L_E + L_I \qquad \text{(Equation 17)}$$

where

L_T	=	Total PB-HAP compound load to the water body (including deposition, runoff, and erosion) (g/yr)
L_{DEP}	=	Total (wet and dry) particle phase and vapor phase PB-HAP compound direct deposition load to water body (g/yr)
L_{dif}	=	Vapor phase PB-HAP compound diffusion load to water body (g/yr)
L_{RI}	=	Runoff load from impervious surfaces (g/yr)
L_R	=	Runoff load from pervious surfaces (g/yr)
L_E	=	Soil erosion load (g/yr)
L_I	=	Internal transfer (g/yr)

Due to the limited data and uncertainty associated with the chemical or biological internal transfer, L_I, of compounds into daughter products, EPA (1998)[1] recommends a default value for this variable of zero. However, if a permitting authority determines that site-specific conditions indicate calculation of internal transfer may need to be considered, EPA (1998)[1] recommends following the methodologies described in EPA NCEA document, *Methodology for Assessing Health Risks Associated with Multiple Pathways of Exposure to Combustor Emissions* (EPA 1998).[12] Calculation of each of the remaining variables (L_{DEP}, L_{dif}, L_{RI}, L_R, and L_E) is discussed in the following subsections.

7.1.1 Total (Wet and Dry) Particle Phase and Vapor Phase PB-HAP compound Direct Deposition Load to Water Body (L_{DEP})

EPA (1998)[1] recommends Equation 18 to calculate the load to the water body from the direct deposition of wet and dry particles and vapors onto the surface of the water body (L_{DEP}).

$$L_{DEP} = Q \cdot \left[F_v \cdot Dytwv + \left(1 - F_v\right) \cdot Dytyp \right] \cdot A_W \qquad \text{(Equation 18)}$$

where

L_{DEP} = Total (wet and dry) particle phase and vapor phase PB-HAP compound direct deposition load to water body (g/yr)
Q = PB-HAP compound emission rate (g/s)
F_v = Fraction of PB-HAP compound air concentration in vapor phase (unitless)
$Dytwv$ = Unitized yearly (water body or watershed) average total (wet and dry) deposition from vapor phase (s/m^2-yr)
$Dytwp$ = Unitized yearly (water body or watershed) average total (wet and dry) deposition from particle phase (s/m^2-yr)
A_W = Water body surface area (m^2)

7.1.2 Vapor Phase PB-HAP compound Diffusion Load to Water Body (L_{dif})

EPA (1998)[1] recommends using Equation 19 to calculate the vapor phase PB-HAP compound diffusion load to the water body (L_{dif}).

$$L_{dif} = \frac{K_v \cdot Q \cdot F_v \cdot Cyvw \cdot A_W \cdot 1 \times 10^{-6}}{\dfrac{H}{R \cdot T_{wk}}} \qquad \text{(Equation 19)}$$

where

L_{dif} = Vapor phase PB-HAP compound diffusion load to water body (g/yr)
K_v = Overall PB-HAP compound transfer rate coefficient (m/yr)
Q = PB-HAP compound emission rate (g/s)
F_v = Fraction of PB-HAP compound air concentration in vapor phase (unitless)
$Cywv$ = Unitized yearly (water body or watershed) average air concentration from vapor phase (µg-s/g-m^3)
A_W = Water body surface area (m^2)
10^{-6} = Units conversion factor (g/µg)
H = Henry's Law constant (atm-m^3/mol)
R = Universal gas constant (atm-m^3/mol-K)
T_{wk} = Water body temperature (K)

The overall PB-HAP compound transfer rate coefficient (K_v) is calculated by using Equation 29 (see section 7.4.4). EPA (1998)[1] recommends a water body temperature (T_{wk}) of 298 K (or 25°C).

7.1.3 Runoff Load from Impervious Surfaces (L_{RI})

In some watershed soils, a fraction of the total (wet and dry) deposition in the watershed will be to impervious surfaces. This deposition may accumulate and be washed off during rain events. EPA (1998)[1] recommends the use of Equation 20 to calculate impervious runoff load to a water body (L_{RI}).

$$L_{RI} = Q \cdot \left[F_v \cdot Dytwv + \left(1.0 - F_v\right) \cdot Dytwp \right] \cdot A_I \qquad \text{(Equation 20)}$$

where

L_{RI} = Runoff load from impervious surfaces (g/yr)
Q = PB-HAP compound emission rate (g/s)
F_v = Fraction of PB-HAP compound air concentration in vapor phase (unitless)
$Dytwv$ = Unitized yearly (water body or watershed) average total (wet and dry) deposition from vapor phase (s/m^2-yr)
$Dytwp$ = Unitized yearly (water body or watershed) average total (wet and dry) deposition from particle phase (s/m^2-yr)
A_I = Impervious watershed area receiving PB-HAP compound deposition (m^2)

Impervious watershed area receiving PB-HAP compound deposition (A_I) is the portion of the total effective watershed area that is impervious to rainfall (such as roofs, driveways, streets, and parking lots) and drains to the water body.

7.1.4 Runoff Load from Pervious Surfaces (L_R)

EPA (1998)[1] recommends the use of Equation 21 to calculate the runoff dissolved PB-HAP compound load to the water body from pervious soil surfaces in the watershed (L_R).

$$L_R = RO \cdot \left(A_L - A_I\right) \cdot \frac{Cs \cdot BD}{\theta_{sw} + Kd_s + BD} \cdot 0.01 \qquad \text{(Equation 21)}$$

where

L_R = Runoff load from pervious surfaces (g/yr)
RO = Average annual surface runoff from pervious areas (cm/yr)
A_L = Total watershed area receiving PB-HAP compound deposition (m^2)
A_I = Impervious watershed area receiving PB-HAP compound deposition (m^2)
Cs = Average soil concentration over exposure duration (in watershed soils) (mg PB-HAP compound/kg soil)
BD = Soil bulk density (g soil/cm^3 soil)
θ_{sw} = Soil volumetric water content (mL water/cm^3 soil)
Kd_s = Soil-water partition coefficient (cm^3 water/g soil)
0.01 = Units conversion factor (kg-cm^2/mg-m^2)

The calculation of the PB-HAP compound concentration in watershed soils (Cs) are discussed in Section 2.1. Soil bulk density (BD) is described in Section 2.5.2. Soil water content (θ_{sw}) is described in Section 2.5.4.

7.1.5 Soil Erosion Load (L_E)

EPA (1998)[1] recommends the use of Equation 22 to calculate soil erosion load (L_E).

$$L_E = X_e \cdot \left(A_L - A_I\right) \cdot SD \cdot ER \cdot \frac{Cs \cdot Kd_s \cdot BD}{\theta_{sw} + Kd_s + BD} \cdot 0.001 \qquad \text{(Equation 22)}$$

where

L_E	=	Soil erosion load (g/yr)
X_e	=	Unit soil loss (kg/m^2-yr)
A_L	=	Total watershed area (evaluated) receiving PB-HAP compound deposition (m^2)
A_I	=	Impervious watershed area receiving PB-HAP compound deposition (m^2)
SD	=	Sediment delivery ratio (watershed) (unitless)
ER	=	Soil enrichment ratio (unitless)
Cs	=	Average soil concentration over exposure duration (in watershed soils) (mg PB-HAP compound/kg soil)
BD	=	Soil bulk density (g soil/cm^3 soil)
θ_{sw}	=	Soil volumetric water content (mL water/cm^3 soil)
Kd_s	=	Soil-water partition coefficient (mL water/g soil)
0.001	=	Units conversion factor (k-cm^2/mg-m^2)

Unit soil loss (X_e) is described in Section 7.2. Watershed sediment delivery ratio (SD) is calculated as described in Section 7.3. PB-HAP compound concentration in soils (Cs) is described in Section 2.1. Soil bulk density (BD) is described in Section 2.5.2. Soil water content (θ_{sw}) is described in Section 2.5.4.

7.2 Universal Soil Loss Equation - USLE

EPA (1998)[1] recommends that the universal soil loss equation (USLE), Equation 22A, be used to calculate the unit soil loss (X_e) specific to each watershed.

$$X_e = RF \cdot K \cdot LS \cdot C \cdot PF \cdot \frac{907.18}{4047} \qquad \text{(Equation 22A)}$$

where

X_e	=	Unit soil loss (kg/m^2-yr)
RF	=	USLE rainfall (or erosivity) factor (yr^{-1})
K	=	USLE erodibility factor (ton/acre)
LS	=	USLE length-slope factor (unitless)
C	=	USLE cover management factor (unitless)
PF	=	USLE supporting practice factor (unitless)
907.18	=	Units conversion factor (kg/ton)
4047	=	Units conversion factor (m^2/acre)

The USLE *RF* variable, which represents the influence of precipitation on erosion, is derived from data on the frequency and intensity of storms. This value is typically derived on a storm-by-storm basis, but average annual values have been compiled (U.S. Department of Agriculture 1982).[31] Information on determining site-specific values for variables used in calculating X_e is provided in U.S. Department of Agriculture (U.S. Department of Agriculture 1997)[32] and EPA guidance (EPA 1985).[22]

7.3 Sediment Delivery Ratio (*SD*)

EPA (1998)[1] recommends the use of Equation 23 to calculate sediment delivery ratio (*SD*).

$$SD = a \cdot \left(A_L\right)^{-b} \qquad \text{(Equation 23)}$$

where

SD = Sediment delivery ratio (watershed) (unitless)
a = Empirical intercept coefficient (unitless)
b = Empirical slope coefficient (unitless)
A_L = Total watershed area (evaluated) receiving PB-HAP compound deposition (m^2)

A_L is the total watershed surface area evaluated that is affected by deposition and drains to the body of water (see Chapter 2). In assigning values to the watershed surface area affected by deposition, the following may be a consideration:

- Distance from the emission source;
- Location of the area affected by deposition fallout with respect to the point at which drinking water is extracted or fishing occurs; and
- The watershed hydrology.

7.4 Total Water Body PB-HAP compound Concentration (C_{wtot})

EPA (1998)[1] recommends the use of Equation 24 to calculate total water body PB-HAP compound concentration (C_{wtot}). The total water body concentration includes both the water column and the bed sediment.

$$C_{wtot} = \frac{L_T}{Vf_x \cdot f_{wc} \cdot k_{wt} \cdot A_W \cdot \left(d_{wc} + d_{bs}\right)} \qquad \text{(Equation 24)}$$

where

C_{wtot} = Total water body PB-HAP compound concentration (including water column and bed sediment) (g PB-HAP compound/m^3 water body)
L_T = Total PB-HAP compound load to the water body (including deposition, runoff, and erosion) (g/yr)
Vf_x = Average volumetric flow rate through water body (m^3/yr)
f_{wc} = Fraction of total water body PB-HAP compound concentration in the water column (unitless)
k_{wt} = Overall total water body PB-HAP compound dissipation rate constant (yr^{-1})

A_W = Water body surface area (m^2)
d_{wc} = Depth of water column (m)
d_{bs} = Depth of upper benthic sediment layer (m)

The total PB-HAP compound load to the water body (L_T) – including deposition, runoff, and erosion – is described in Section 7.1. The depth of the upper benthic layer (d_{bs}), which represents the portion of the bed that is in equilibrium with the water column, cannot be precisely specified; however, EPA (1998)[1] recommends a default value of 0.03. Issues related to the remaining parameters are summarized in the following subsections.

7.4.1 Fraction of Total Water Body PB-HAP compound Concentration in the Water Column (f_{wc}) and Benthic Sediment (f_{bs})

EPA (1998)[1] recommends using Equation 25A to calculate fraction of total water body PB-HAP compound concentration in the water column (f_{wc}), and Equation 25B to calculate total water body contaminant concentration in benthic sediment (f_{bs}).

$$f_{wc} = \frac{\left(1 + kd_{sw} \cdot TSS \cdot 1 \times 10^{-6}\right) \cdot d_{wc} / d_z}{\left(1 + kd_{sw} \cdot TSS \cdot 10^{-6}\right) \cdot d_{wc} / d_z + \left(\theta_{bs} + Kd_{bs} \cdot C_{BS}\right) \cdot d_{bs} / d_z} \qquad \text{(Equation 25A)}$$

$$f_{bs} = 1 - f_{wc} \qquad \text{(Equation 25B)}$$

where

f_{wc} = Fraction of total water body PB-HAP compound concentration in the water column (unitless)
f_{bs} = Fraction of total water body PB-HAP compound concentration in benthic sediment (unitless)
Kd_{sw} = Suspended sediments/surface water partition coefficient (L water/kg suspended sediment)
TSS = Total suspended solids concentration (mg/L)
1×10^{-6} = Units conversion factor (kg/mg)
d_z = Total water body depth (m)
θ_{bs} = Bed sediment porosity ($L_{water}/L_{sediment}$)
Kd_{bs} = Bed sediment/sediment pore water partition coefficient (L water/kg bottom sediment)
C_{BS} = Bed sediment concentration (g/cm^3 [equivalent to kg/L])
d_{wc} = Depth of water column (m)
d_{bs} = Depth of upper benthic sediment layer (m)

The partition coefficient Kd_{sw} describes the partitioning of a contaminant between sorbing material, such as soil, surface water, suspended solids, and bed sediments. Due to variability in water body specific values, EPA (1998)[1] recommends the use of water body-specific measured total suspended solids (*TSS*) values representative of long-term average annual values for the water body of concern. Average annual values for *TSS* are generally expected to be in the range

of 2 to 300 mg/L; with additional information on anticipated *TSS* values available in the EPA NCEA document, *Methodology for Assessing Health Risks Associated with Multiple Pathways of Exposure to Combustor Emissions* (EPA 1998).[12] If measured data are not available, or of unacceptable quality, a calculated *TSS* value can be obtained for non-flowing water bodies using Equation 25C.

$$TSS = \frac{X_e \cdot \left(A_L - A_I\right) \cdot SD \cdot 1 \times 10^3}{Vf_x + D_{ss} \cdot A_W} \qquad \text{(Equation)25C}$$

where

TSS	=	Total suspended solids concentration (mg/L)
X_e	=	Unit soil loss (kg/m^2-yr)
A_L	=	Total watershed area (evaluated) receiving PB-HAP compound deposition (m^2)
A_I	=	Impervious watershed area receiving PB-HAP compound deposition (m^2)
SD	=	Sediment delivery ratio (watershed) (unitless)
Vf_x	=	Average volumetric flow rate through water body (value should be 0 for quiescent lakes or ponds) (m^3/yr)
D_{ss}	=	Suspended solids deposition rate (a default value of 1,825 for quiescent lakes or ponds) (m/yr)
A_W	=	Water body surface area (m^2)
$1x10^{-3}$	=	Units conversion factor (g/kg)

The default value of 1,825 m/yr provided for D_{ss} is characteristic of Stoke's settling velocity for an intermediate (fine to medium) silt.

Also, to evaluate the appropriateness of watershed-specific values used in calculating the unit soil loss (X_e), as described in Section 7.2, the water-body specific measured *TSS* value can be compared to the calculated *TSS* value obtained using Equation 25C. If the measured and calculated *TSS* values differ significantly, parameter values used in calculating X_e can be re-evaluated. This re-evaluation of *TSS* and X_e can also be conducted if the calculated *TSS* value is outside of the normal range expected for average annual measured values, as discussed above.

Bed sediment porosity (θ_{bs}) can be calculated from the bed sediment concentration by using Equation 26 (EPA 1993b)[11]:

$$\theta_{bs} = 1 - \frac{C_{BS}}{\rho_s} \qquad \text{(Equation 26)}$$

where

θ_{bs}	=	Bed sediment porosity ($L_{water}/L_{sediment}$)
ρ_s	=	Bed sediment density (kg/L)
C_{BS}	=	Bed sediment concentration (kg/L)

EPA (1998)[1] recommends the default value of 0.6 $L_{water}/L_{sediment}$ for bed sediment porosity (θ_{bs}). This assumes a bed sediment density (ρ_s) of 2.65 kg/L and a bed sediment concentration (C_{BS}) of 1.0 kg/L.

7.4.2 Overall Total Water Body PB-HAP compound Dissipation Rate Constant (k_{wt})

EPA (1998)[1] recommends the use of Equation 27 to calculate the overall dissipation rate of PB-HAP compounds in surface water, resulting from volatilization and benthic burial.

$$k_{wt} = f_{wc} \cdot k_v + f_{bs} \cdot k_b \qquad \text{(Equation 27)}$$

where

k_{wt} = Overall total water body dissipation rate constant (yr^{-1})
f_{wc} = Fraction of total water body PB-HAP compound concentration in the water column (unitless)
k_v = Water column volatilization rate constant (yr^{-1})
f_{bs} = Fraction of total water body PB-HAP compound concentration in benthic sediment (unitless)
k_b = Benthic burial rate constant (yr^{-1})

The variables f_{wc} and f_{bs} are discussed in Section 7.4.1, and Equations 25A and 25B.

7.4.3 Water Column Volatilization Rate Constant (k_v)

EPA (1998)[1] recommends using Equation 28 to calculate water column volatilization rate constant.

$$k_v = \frac{K_v}{d_z \cdot \left(1 + kd_{sw} \cdot TSS \cdot 1 \times 10^{-6}\right)} \qquad \text{(Equation 28)}$$

where

k_v = Water column volatilization rate constant (yr^{-1})
K_v = Overall PB-HAP compound transfer rate coefficient (m/yr)
d_z = Total water body depth (m)
Kd_{sw} = Suspended sediments/surface water partition coefficient (L water/kg suspended sediments)
TSS = Total suspended solids concentration (mg/L)
1×10^{-6} = Units conversion factor (kg/mg)

Total water body depth (d_z), suspended sediment and surface water partition coefficient (Kd_{sw}), and total suspended solids concentration (TSS), are described in Section 7.4.1. The overall transfer rate coefficient (K_v) is described in Section 7.4.4.

7.4.4 Overall PB-HAP compound Transfer Rate Coefficient (K_v)

Volatile organic chemicals can move between the water column and the overlying air. The overall transfer rate K_v, or conductivity, is determined by a two-layer resistance model that assumes that two "stagnant films" are bounded on either side by well-mixed compartments. Concentration differences serve as the driving force for the water layer diffusion. Pressure differences drive the diffusion for the air layer. From balance considerations, the same mass must pass through both films; the two resistances thereby combine in series, so that the conductivity is the reciprocal of the total resistance.

EPA (1998)[1] recommends the use of Equation 29 to calculate the overall transfer rate coefficient (K_v).

$$K_v = \left(K_L^{-1} + \left(K_G \cdot \frac{H}{R \cdot T_{wk}} \right)^{-1} \right)^{-1} \cdot \theta^{T_{wk}-293} \qquad \text{(Equation 29)}$$

where

K_v	=	Overall PB-HAP compound transfer rate coefficient (m/yr)
K_L	=	Liquid phase transfer coefficient (m/yr)
K_G	=	Gas phase transfer coefficient (m/yr)
H	=	Henry's Law constant (atm-m^3/mol)
R	=	Universal gas constant (atm-m^3/mol-K)
T_{wk}	=	Water body temperature (K)
θ	=	Temperature correction factor (unitless)

The value of the conductivity K_v depends on the intensity of turbulence in the water body and the overlying atmosphere. As Henry's Law constant increases, the conductivity tends to be increasingly influenced by the intensity of turbulence in water. Conversely, as Henry's Law constant decreases, the value of the conductivity tends to be increasingly influenced by the intensity of atmospheric turbulence.

The liquid and gas phase transfer coefficients, K_L and K_G, respectively, vary with the type of water body. The liquid phase transfer coefficient (K_L) is calculated by using Equations 30A and 30B (described in Section 7.4.5). The gas phase transfer coefficient (K_G) is calculated by using Equations 31A and 31B (described in Section 7.4.6).

Henry's Law constants generally increase with increasing vapor pressure of a PB-HAP compound and generally decrease with increasing solubility of a PB-HAP compound. Henry's Law constants are compound-specific and are presented in Appendix D. The universal ideal gas constant, R, is 8.205×10^{-5} atm-m^3/mol-K, at 20°C. The temperature correction factor (θ), which is equal to 1.026, is used to adjust for the actual water temperature. Volatilization is assumed to occur much less readily in lakes and reservoirs than in moving water bodies.

7.4.5 Liquid Phase Transfer Coefficient (K_L)

EPA (1998)[1] recommends using Equations 30A and 30B to calculate liquid phase transfer coefficient. (K_L).

$$K_L = \sqrt{\frac{\left(1 \times 10^{-4}\right) \cdot D_w \cdot u}{d_z}} \cdot 3.1536 \times 10^7 \qquad \text{(Equation 30A)}$$

$$K_L = \left(C_d^{0.5} \cdot W\right) \cdot \left(\frac{\rho_a}{\rho_w}\right)^{0.5} \cdot \frac{k^{0.33}}{\lambda_z} \cdot \frac{\mu_w}{\rho_w \cdot D_w} \cdot 3.1536 \times 10^7 \qquad \text{(Equation 30B)}$$

where

K_L	=	Liquid phase transfer coefficient (m/yr)
D_w	=	Diffusivity of PB-HAP compound in water (cm^2/s)
u	=	Current velocity (m/s)
1×10^{-4}	=	Units conversion factor (m^2/cm^2)
d_z	=	Total water body depth (m)
C_d	=	Drag coefficient (unitless)
W	=	Average annual wind speed (m/s)
ρ_a	=	Density of air (g/cm^3)
ρ_w	=	Density of water (g/cm^3)
k	=	von Karman's constant (unitless)
λ_z	=	Dimensionless viscous sublayer thickness (unitless)
μ_w	=	Viscosity of water corresponding to water temperature (g/cm-s)
3.1536×10^7	=	Units conversion factor (s/yr)

For a flowing stream or river, the transfer coefficients are controlled by flow-induced turbulence. For these systems, the liquid phase transfer coefficient is calculated by using Equation 30A. For a stagnant system (quiescent lake or pond), the transfer coefficient is controlled by wind-induced turbulence, and the liquid phase transfer coefficient can be calculated by using Equation 30B. The total water body depth (d_z) for liquid phase transfer coefficients is discussed in Section 7.4.1. EPA (1998)[1] recommends the use of the following default values:

- A diffusivity of chemical in water ranging (D_w) from 1.0×10^{-5} to 8.5×10^{-2} cm^2/s;
- A dimensionless viscous sublayer thickness (λ_z) of 4;
- A von Karman's constant (k) of 0.4;
- A drag coefficient (C_d) of 0.0011;
- An air density (ρ_a) of 0.0012 g/cm^3 at standard conditions (temperature = 20°C or 293 K, pressure = 1 atm or 760 millimeters of mercury);
- A water density of(ρ_w) of 1 g/cm^3; and
- A water viscosity(μ_w) of a 0.0169 g/cm-s corresponding to water temperature.

7.4.6 Gas Phase Transfer Coefficient (K_G)

EPA (1998)[1] recommends using Equations 31A and 31B to calculate gas phase transfer coefficient (K_G).

For flowing streams or rivers:

$$K_G = 36{,}500\ m/yr \qquad \text{(Equation 31A)}$$

For quiescent lakes or ponds:

$$K_G = \left(C_d^{0.5} \cdot W\right) \cdot \frac{k^{0.33}}{\lambda_z} \cdot \left(\frac{\mu_a}{\rho_a \cdot D_a}\right)^{-0.67} \cdot 3.1536 \times 10^7 \qquad \text{(Equation 31B)}$$

where

K_G	=	Gas phase transfer coefficient (m/yr)
C_d	=	Drag coefficient (unitless)
W	=	Average annual wind speed (m/s)
k	=	von Karman's constant (unitless)
λ_z	=	Dimensionless viscous sublayer thickness (unitless)
μ_a	=	Viscosity of air corresponding to air temperature (g/cm-s)
ρ_a	=	Density of air corresponding to water temperature (g/cm^3)
D_a	=	Diffusivity of PB-HAP compound in air (cm^2/s)
3.1536×10^7	=	Units conversion factor (s/yr)

EPA (1998)[1] recommends 1.81 x 10^{-4} g/cm-s for the viscosity of air corresponding to air temperature.

7.4.7 Benthic Burial Rate Constant (k_b)

EPA (1998)[1] recommends using Equation 32 to calculate benthic burial rate (k_b).

$$k_b = \left(\frac{X_e \cdot A_L \cdot SD \cdot 1 \times 10^3 - Vf_x \cdot TSS}{A_W \cdot TSS}\right) \cdot \left(\frac{TSS \cdot 1 \times 10^{-6}}{C_{BS} \cdot d_{bs}}\right) \qquad \text{(Equation 32)}$$

where

k_b	=	Benthic burial rate constant (yr^{-1})
X_e	=	Unit soil loss (kg/m^2-yr)
A_L	=	Total watershed area (evaluated) receiving deposition (m^2)
SD	=	Sediment delivery ratio (watershed) (unitless)
Vf_x	=	Average volumetric flow rate through water body (m^3/yr)
TSS	=	Total suspended solids concentration (mg/L)
A_W	=	Water body surface area (m^2)

C_{BS}	=	Bed sediment concentration (g/cm^3)
d_{bs}	=	Depth of upper benthic sediment layer (m)
1 x 10^{-6}	=	Units conversion factor (kg/mg)
1 x 10^3	=	Units conversion factor (g/kg)

The benthic burial rate constant (k_b), can also be expressed in terms of the rate of burial (Wb) (Equation 33):

$$Wb = k_b \cdot d_{bs} \qquad \text{(Equation 33)}$$

where

Wb	=	Rate of burial (m/yr)
k_b	=	Benthic burial rate constant (yr^{-1})
d_{bs}	=	Depth of upper benthic sediment layer (m)

EPA (1998)[1] recommends the following default value of 1.0 kg/L for bed sediment concentration (C_{BS}).

Section 7.2 discusses the unit soil loss (X_e). Section 7.3 discusses sediment delivery ratio (SD) and watershed area evaluated receiving PB-HAP compound deposition (A_L). Section 7.4 discusses the depth of the upper benthic sediment layer (d_{bs}). Average volumetric flow rate through the water body (Vf_x) and water body surface area (A_w) are discussed further in EPA (1998).[1] Section 7.4.1 discusses total suspended solids concentration (TSS).

The calculated value for k_b is expected to range from 0 to 1.0; with low k_b values expected for water bodies characteristic of no or limited sedimentation (rivers and fast flowing streams), and k_b values closer to 1.0 expected for water bodies characteristic of higher sedimentation (lakes). This range of values is based on the relation between the benthic burial rate and rate of burial expressed in Equation 33; with the depth of upper benthic sediment layer held constant. For k_b values calculated as a negative (water bodies with high average annual volumetric flow rates in comparison to watershed area evaluated), EPA (1998)[1] recommends assigning a k_b value of 0 for use in calculating the total water body PB-HAP compound concentration (C_{wtot}) in Equation 34 (see next section). If the calculated k_b value exceeds 1.0, re-evaluation of the parameter values used in calculating X_e is recommended to be conducted.

7.4.8 Total PB-HAP compound Concentration in Water Column (C_{wctot})

EPA (1998)[1] recommends using Equation 34 to calculate total PB-HAP compound concentration in water column (C_{wctot}).

$$C_{wctot} = f_{wc} \cdot C_{wtot} \cdot \frac{d_{wc} + d_{bs}}{d_{wc}} \qquad \text{(Equation 34)}$$

where

C_{wctot}	=	Total PB-HAP compound concentration in water column (mg PB-HAP compound/L water column)
f_{wc}	=	Fraction of total water body PB-HAP compound concentration in the water column (unitless)
C_{wtot}	=	Total water body PB-HAP compound concentration, including water column and bed sediment (mg PB-HAP compound/L water body)
d_{wc}	=	Depth of water column (m)
d_{bs}	=	Depth of upper benthic sediment layer (m)

Total water body PB-HAP compound concentration – including water column and bed sediment (C_{wtot}) and fraction of total water body PB-HAP compound concentration in the water column (f_{wc}) – can be calculated by using Equation 34 and Equation 35 (see next section). Depth of upper benthic sediment layer (d_{bs}) is discussed in Section 7.4.1.

7.4.9 Dissolved Phase Water Concentration (C_{dw})

EPA (1998)[1] recommends the use of Equation 35 to calculate the concentration of PB-HAP compound dissolved in the water column (C_{dw}).

$$C_{dw} = \frac{C_{wctot}}{1 + Kd_{sw} \cdot TSS \cdot 1 \times 10^{-6}} \qquad \text{(Equation 35)}$$

where

C_{dw}	=	Dissolved phase water concentration (mg PB-HAP compound/L water)
C_{wctot}	=	Total PB-HAP compound concentration in water column (mg PB-HAP compound/L water column)
Kd_{sw}	=	Suspended sediments/surface water partition coefficient (L water/kg suspended sediment)
TSS	=	Total suspended solids concentration (mg/L)
1×10^{-6}	=	Units conversion factor (kg/mg)

The total PB-HAP compound concentration in water column (C_{wctot}) is calculated by using the Equation 34. Section 7.4.1 discusses the surface water partition coefficient (Kd_{sw}) and total suspended solids concentration (TSS).

7.4.10 PB-HAP compound Concentration Sorbed to Bed Sediment (C_{sb})

EPA (1998)[1] recommends the use of Equation 36 to calculate PB-HAP compound concentration sorbed to bed sediment (C_{sb}).

$$C_{sb} = f_{bs} \cdot C_{wtot} \cdot \left(\frac{Kd_{bs}}{\theta_{bs} + Kd_{bs} \cdot C_{BS}} \right) \cdot \left(\frac{d_{wc} + d_{bs}}{d_{bs}} \right) \qquad \text{(Equation 36)}$$

where

C_{sb}	=	PB-HAP compound concentration sorbed to bed sediment (mg PB-HAP compound/kg sediment)
f_{bs}	=	Fraction of total water body PB-HAP compound concentration in benthic sediment (unitless)
C_{wtot}	=	Total water body PB-HAP compound concentration, including water column and bed sediment (mg PB-HAP compound/L water body)
Kd_{bs}	=	Bed sediment/sediment pore water partition coefficient (L PB-HAP compound/kg water body)
θ_{bs}	=	Bed sediment porosity ($L_{pore\ water}/L_{sediment}$)
C_{BS}	=	Bed sediment concentration (g/cm^3)
d_{wc}	=	Depth of water column (m)
d_{bs}	=	Depth of upper benthic sediment layer (m)

Bed sediment porosity (θ_{bs}) and bed sediment concentration (C_{BS}) are discussed in Section 7.4.1. Depth of water column (d_{wc}) and depth of upper benthic layer (d_{bs}) are discussed in Section 7.4.

7.5 Concentration of PB-HAP compound in Fish (C_{fish})

The PB-HAP compound concentration in fish is calculated using either a PB-HAP compound-specific bioconcentration factor (*BCF*), a PB-HAP compound-specific bioaccumulation factor (*BAF*), or a PB-HAP compound-specific biota-sediment accumulation factor (*BSAF*). For compounds with a $log\ K_{ow}$ less than 4.0, *BCF*s are used. Compounds with a $log\ K_{ow}$ greater than 4.0 (except for extremely hydrophobic compounds such as polycyclic organic matter and PCBs), are assumed to have a high tendency to bioaccumulate, therefore, *BAF*s are used. While extremely hydrophobic PB-HAP compounds are also assumed to have a high tendency to bioaccumulate, they are expected to be sorbed to the bed sediments more than associated with the water phase. Therefore, for polycyclic organic matter and PCBs, EPA (1998)[1] recommends using *BSAF*s to calculate concentrations in fish.

BCF and *BAF* values are generally based on dissolved water concentrations. Therefore, when *BCF* or *BAF* values are used, the PB-HAP compound concentration in fish is calculated using dissolved water concentrations. *BSAF* values are based on benthic sediment concentrations. Therefore, when *BSAF* values are used, PB-HAP compound concentration in fish is calculated using benthic sediment concentrations. The equations used to calculate fish concentrations are described in the subsequent subsections.

7.5.1 Fish Concentration (C_{fish}) from Bioconcentration Factors Using Dissolved Phase Water Concentration

EPA (1998)[1] recommends the use of Equation 37 to calculate fish concentration from *BCF*s using dissolved phase water concentration.

$$C_{fish} = C_{dw} \cdot BCF_{fish} \qquad \text{(Equation 37)}$$

where

C_{fish} = Concentration of PB-HAP compound in fish (mg PB-HAP compound/kg FW tissue)
C_{dw} = Dissolved phase water concentration (mg PB-HAP compound/L)
BCF_{fish} = Bioconcentration factor for PB-HAP compound in fish (L/kg)

The dissolved phase water concentration (C_{dw}) is calculated by using Equation 35.

7.5.2 Fish Concentration (C_{fish}) from Bioaccumulation Factors Using Dissolved Phase Water Concentration

EPA (1998)[1] recommends the use of Equation 38 to calculate fish concentration from *BAF*s using dissolved phase water concentration.

$$C_{fish} = C_{dw} \cdot BAF_{fish} \qquad \text{(Equation 38)}$$

where

C_{fish} = Concentration of PB-HAP compound in fish (mg PB-HAP compound/kg FW tissue)
C_{dw} = Dissolved phase water concentration (mg PB-HAP compound/L)
BAF_{fish} = Bioaccumulation factor for PB-HAP compound in fish (L/kg FW tissue)

The dissolved phase water concentration (C_{dw}) is calculated by using Equation 35.

7.5.3 Fish Concentration (C_{fish}) from Biota-To-Sediment Accumulation Factors Using PB-HAP compound Sorbed to Bed Sediment

EPA (1998)[1] recommends the use of Equation 39 to calculate fish concentration from *BSAF*s using PB-HAP compound sorbed to bed sediment for very hydrophobic compounds (polycyclic organic matter and PCBs).

$$C_{fish} = \frac{C_{sb} \cdot f_{lipid} \cdot BSAF}{OC_{sed}} \qquad \text{(Equation 39)}$$

where

C_{fish} = Concentration of PB-HAP compound in fish (mg PB-HAP compound/kg FW tissue)

C_{sb} = Concentration of PB-HAP compound sorbed to bed sediment (mg PB-HAP compound/kg bed sediment)
f_{lipid} = Fish lipid content (unitless)
$BSAF$ = Biota-to-sediment accumulation factor (unitless)
OC_{sed} = Fraction of organic carbon in bottom sediment (unitless)

The concentration of PB-HAP compound sorbed to bed sediment (C_{sb}) is calculated by using Equation 36. EPA recommended default values for the fish lipid content (f_{lipid}) and for the fraction of organic carbon in bottom sediment (OC_{sed}) are given in EPA (1998).[1]

Values for the fraction of organic carbon in bottom sediment recommended by EPA (1993b)[11] range from 0.03 to 0.05 (Oc_{sed}). These values are based on an assumption of a surface soil OC content of 0.01. This document states that the organic carbon content in bottom sediments is higher than the organic carbon content in soils because (1) erosion favors lighter-textured soils with higher organic carbon contents, and (2) bottom sediments are partially comprised of detritus materials.

The fish lipid content (f_{lipid}) value is site-specific and dependent on the type of fish. As stated in EPA (1998)[1], a default range of 0.03 to 0.07 is recommended specific to warm or cold water fish species. EPA (2000)[33] provides information supporting a value of 0.03 (3 percent lipid content of the edible portion). EPA (1993a)[34] recommended a default value of 0.04 for OC_{sed}, which is the midpoint of the specified range. EPA (1993b; 1993a)[11][34] recommended the use of 0.07, which was originally cited in Cook et al. (1991).[35]

8.0 Concentrations of Dioxins in Breast Milk

EPA (1998)[1] recommends the use of Equation 40 to estimate the concentrations of dioxins in breast milk.

$$C_{milkfat} = \frac{m \cdot 1 \times 10^9 \cdot h \cdot f_1}{0.6932 \cdot f_2} \qquad \text{(Equation 40)}$$

where

$C_{milkfat}$ = Concentration of dioxin in milk fat of breast milk for a specific exposure scenario (pg dioxin/kg milk fat)
m = Average maternal intake of dioxin for each adult exposure scenario (mg dioxin/kg BW-day)
1×10^9 = Units conversion factor (pg/mg)
h = Half-life of dioxin in adults (days)
f_1 = Fraction of ingested dioxin that is stored in fat (unitless)
f_2 = Fraction of mother's weight that is fat (unitless)

The values of m, h, f_1, and f_2 are site-specific and dependent on the specific species of dioxin present. EPA (1998)[1] recommends a default value of 2,555 days for h, a default value of 0.9 for f_1, and a default value of 0.3 for f_2 . Additional references for the derivation of this equation and these default values are given in EPA (1998).[1]

Uncertainties associated with this equation include:

- The most significant uncertainties are associated with the variable *m*. Because *m* is calculated as the sum of numerous potential intakes, estimates of *m* incorporate uncertainties associated with each exposure pathway. Therefore, *m* may be under- or over-estimated.

- This equation assumes that the concentration of dioxin in breast milk fat is the same as in maternal fat. To the extent that this is not the case, uncertainty is introduced.

References

1. U.S. Environmental Protection Agency. 1998. *Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities, Volume 1, Peer Review Draft*. Office of Solid Waste and Emergency Response, July 1998, available at: http://www.epa.gov/epaoswer/hazwaste/combust/risk.htm

2. Bohn H., B.L. McNeal, and G.A. O'Connor. 1985. *Soil Chemistry*. John Wiley and Sons, Inc. New York. Second Edition.

3. Sparks, D.L. 1989. *Kinetics of Soil Chemical Processes*. Academic Press, Inc. San Diego, California.

4. U.S. Environmental Protection Agency. 1990. *Interim Final Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions*. Environmental Criteria and Assessment Office. ORD. EPA-600-90-003. January.

5. Valentine, R.L. 1986. "Nonbiological Transformation." *Vadose Zone Modeling of Organic Pollutants*. S.C. Hern and S.M. Melacon, Editors. Lewis Publishers, Inc. Chelsea, Michigan.

6. U.S. Environmental Protection Agency. 1986. *Superfund Public Health Evaluation Manual*. Office of Emergency and Remedial Response, Washington, D.C. EPA/540/1-86/060.

7. Lyman, W.J., W.F. Reehl, and D.H. Rosenblatt. 1982. *Handbook of Chemical Property Estimation Methods*. McGraw-Hill Book Company. New York, New York.

8. U.S. Environmental Protection Agency. 1994a. *Draft Guidance for Performing Screening Level Risk Analyses at Combustion Facilities Burning Hazardous Wastes. Attachment C, Draft Exposure Assessment Guidance for RCRA Hazardous Waste Combustion Facilities.* April 15.

9. Jury, W.A., and R.L. Valentine. 1986. "Transport Mechanisms and Loss Pathways for Chemicals in Soils." *Vadose Zone Modeling of Organic Pollutants*. S.C. Hern and S.M. Melancorn, Editors. Lewis Publishers, Inc. Chelsea, Michigan.

10. Valentine, R.L., and Schnoor. 1986. "Biotransformation." *Vadose Zone Modeling of Organic Pollutants*. S.C. Hern and S.M. Melancon, Editors. Lewis Publishers, Inc. Chelsea, Michigan.

11. U.S. Environmental Protection Agency. 1993b. *Review Draft Addendum to the Methodology for Assessing Health Risks Associated with Indirect Exposure to Combustor Emissions*. OHEA. ORD. EPA/600-AP-93-003. November 10, 1993.

12. U.S. Environmental Protection Agency. 1998. *Methodology for Assessing Health Risks Associated with Multiple Pathways of Exposure to Combustor Emissions (MPE).* Update to EPA/600/6-90/003. Office of Research and Development, National Center for Environmental Assessment, U.S. EPA. EPA/600/R-98/137. December.

13. Jury, W.A. 1986. "Volatilization from the Soil." *Vadose Zone Modeling of Organic Pollutants.* S.C. Hern and S.M. Melancorn, Editors. Lewis Publishers, Inc. Chelsea, Michigan.

14. Hillel, D. 1980. *Fundamentals of Soil Physics*. Academic Press, Inc. New York, New York.

Miller, R.W., and D.T. Gardiner.1889. *In: Soils in Our Environment*. J.U. Miller, Ed. Prentice Hall. Upper Saddle River, N.J. Pages 80-123.

15. Hoffman, F.O., and C.F. Baes. 1979. *A Statistical Analysis of Selected Parameters for Predicting Food Chain Transport and Internal Dose of Radionuclides*. ORNL/NUREG/TM-882.

16. Carsel, R.F., R.S, Parrish, R.L. Jones, J.L. Hansen, and R.L. Lamb. 1988. "Characterizing the Uncertainty of Pesticide Leaching in Agricultural Soils." *Journal of Contaminant Hydrology*. Vol. 2. Pages 11-24.

17. Blake, G.R. and K.H. Hartge. 1996. *Particle Density. Methods of Soil Analysis, Part 1: Physical and Mineralogical Methods, 2nd ed.* Arnold Klute, Ed. American Society of Agronomy, Inc. Madison, WI. Page 381.

18. U.S. Environmental Protection Agency. 1994c. *Revised Draft Guidance for Performing Screening Level Risk Analyses at Combustion Facilities Burning Hazardous Wastes. Attachment C, Draft Exposure Assessment Guidance for RCRA Hazardous Waste Combustion Facilities*. OERR. OSW. December 14, 1994.

19. (ASTM Method D2937)

20. (ASTM Method 2216)

21. Geraghty, J.J., D.W. Miller, F. Van Der Leeden, and F.L. Troise. 1973. *Water Atlas of the United States*. Water Information Center, Inc. New York.

22. U.S. Environmental Protection Agency. 1985. *Water Quality Assessment: A Screening Procedure for Toxic and Conventional Pollutants in Surface and Ground Water—Part I (Revised).* ORD. Athens, Georgia. EPA/600/6-85/002a.

23. Stewart, B.A, D.A. Woolhiser, W.H. Wischmeier, J.H. Caro, and M.H. Frere. 1975. *Control of Water Pollution from Croplands. Volume I.* EPA/600/2-75/026a.

24. Briggs, G.G., R.H. Bromilow, and A.A. Evans. 1982. "Relationships Between Lipophilicity and Root Uptake and Translocation of Nonionized Chemicals by Barley." *Pesticide Science*. Volume 13. Pages 495-504.

25. U.S. Environmental Protection Agency. 1994b. *Estimating Exposure to Dioxin-Like Compounds. Volume III: Site-Specific Assessment Procedures. Review Draft*. ORD. Washington, D.C. EPA/600/6-88/005Cc. June.

26. North Carolina Department of Health, Environment, and Natural Resources (NC DEHNR). 1997. *North Carolina Protocol for Performing Indirect Exposure Risk Assessments for Hazardous Waste Combustion Units*. January.

27. Boone, F.W., Y.C. Ng, and J.M. Palm. 1981. "Terrestrial Pathways of Radionuclide Particulates." *Health Physics*. 41:735-747.

28. National Academy of Sciences (NAS). 1983. *Risk Assessment in the Federal Government: Managing the Process.* National Research Council, Committee on the Institutional Means for Assessment of Risks to Public Health, Washington, D.C.

29. U.S. Environmental Protection Agency. 1982. *Pesticides Assessment Guidelines Subdivision O: Residue Chemistry*. Office of Pesticides and Toxic Substances. Washington, D.C. EPA-600-90-003. January.

30. U.S. Environmental Protection Agency. 1995. *Further Issues for Modeling the Indirect Exposure Impacts from Combustor Emissions*. ORD. Washington, D.C. January 20.

31. U.S. Department of Agriculture (USDA). 1982. *Average Annual Values for the Rainfall Factor, R*. Soil Conservation Service Technical Guide, Section I-C-2. Columbia, Missouri. July.

32. U.S. Department of Agriculture (USDA). 1997. *Predicting Soil Erosion by Water: A Guide to Conservation Planning With the Revised Universal Soil Loss Equation (RUSLE).* Agricultural Research Service, Agriculture Handbook Number 703. January.

33. U.S. Environmental Protection Agency. 2000. *Draft Methodology for Deriving Ambient Water Quality Criteria for Protection of Human Health. Technical Support Document Volume III: Bioaccumulation Part 1 - Development of National Bioaccumulation Factors.* Office of Science and Technology in the Office of Water.

34. U.S. Environmental Protection Agency. 1993a. *Addendum to the Methodology for Assessing Heath Risks Associated with Indirect Exposure to Combustor Emissions*. Working Group Recommendations. Office of Solid Waste. ORD. Washington D.C. September 24.

35. Cook, P.M., D.W. Duehl, M.K. Walker, and R.E. Peterson. 1991. *Bioaccumulation and Toxicity of Tetrachlorodibenzo-p-dioxin (TCDD) and Related Compounds in Aquatic Ecosystems*. In Gallo, M.A., R.J. Schuplein, and K.A. Van Der Heijden (eds). *Banbury Report 35: Biological Basis for Risk Assessment of Dioxins and Related Compounds*. Cold Spring Harbor Laboratory Press. 0-87969-235-9/91.