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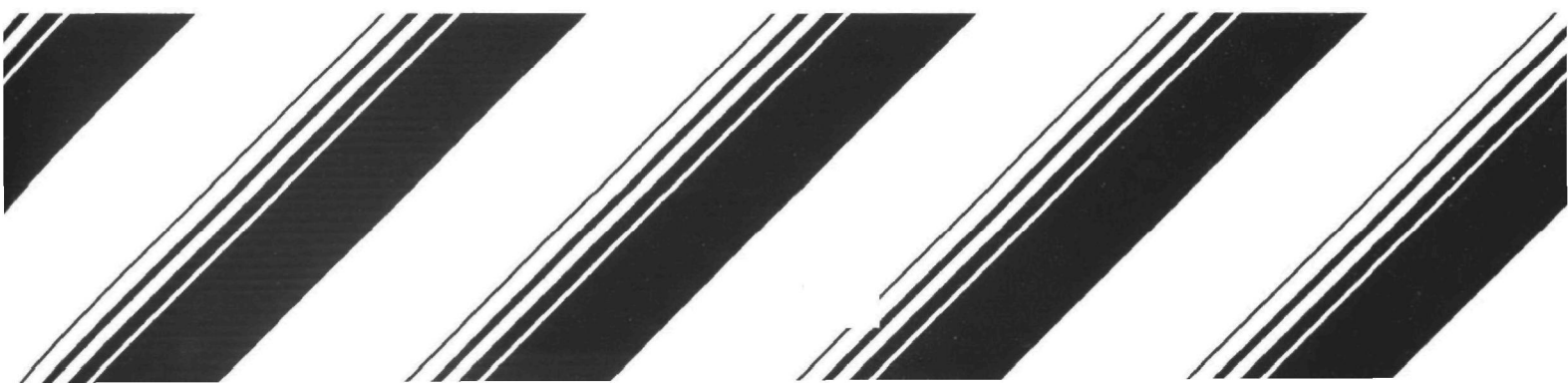
Background Document to the Integrated Risk Assessment for Dioxins and Furans from Chlorine Bleaching in Pulp and Paper Mills



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BACKGROUND DOCUMENT TO THE INTEGRATED RISK ASSESSMENT FOR DIOXINS AND FURANS FROM CHLORINE BLEACHING IN PULP AND PAPER MILLS

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TABLE OF CONTENTS

		Page No.
1.	INTRODUCTION	1-1
1.1	Background	1-1
1.2	Purpose and Scope	1-7
1.3	Industry Profile	1-16
1.3.1	Overview of the Industry	1-16
1.3.2	Processes/Operation	1-16
1.3.3	Potential Source of Dioxin/Furans and Their Concentrations in Pulp and Paper	1-18
1.3.4	End-Uses of Products from Pulp and Paper	1-18
1.4	Organization of This Report	1-20
1.5	References	1-21
2.	CHEMISTRY AND FATE OF DIOXINS AND FURANS	2-1
2.1	Chemical Identity of PCDDs and PCDFs	2-1
2.2	Chemistry and Fate of 2,3,7,8-TCDD	2-5
2.2.1	Chemical Identity	2-5
2.2.2	Chemical and Physical Properties	2-5
2.2.3	Environmental Fate and Transport	2-5
2.3	Chemistry and Fate of 2,3,7,8-TCDF	2-24
2.3.1	Chemical Identity	2-24
2.3.2	Chemical and Physical Properties	2-24
2.3.3	Environmental Fate and Transport	2-25
2.4	References	2-28
3.	DIOXIN AND FURAN HAZARD ASSESSMENT: HUMANS, TERRESTRIAL AND AVIAN WILDLIFE, AND AQUATIC LIFE	3-1
3.1	Introduction	3-1
3.2	Human Hazard Assessment of PCDDs and PCDFs	3-1
3.3	Human Health Hazard of 2,3,7,8-TCDD	3-3
3.3.1	Cancer Effects	3-3
3.3.2	Non-Cancer Effects	3-7
3.3.3	Toxicity Equivalence Factors	3-9

TABLE OF CONTENTS (continued)

		Page No.
3.4	Ecological Hazard of 2,3,7,8-TCDD and 2,3,7,8-TCDF ...	3-15
3.4.1	Aquatic Toxicity of Dioxins	3-16
3.4.2	Aquatic Toxicity of PCDFs	3-26
3.4.3	Conclusions Concerning Aquatic Toxicity	3-26
3.5	Toxicity of PCDDs and PCDFs to Wildlife	3-27
3.5.1	Toxicity Assessment for Birds	3-28
3.5.2	Toxicity Assessment for Bird Eggs	3-28
3.5.3	Toxicity Assessment for Wildlife Mammals	3-31
3.6	Analysis of Uncertainties	3-31
3.7	Conclusions	3-33
3.8	References	3-35
4.	ASSESSMENT OF RISKS TO WORKERS FROM EXPOSURE TO DIOXINS AND FURANS FROM MANUFACTURE, PROCESSING, AND COMMERCIAL USE OF PULP, PAPER, AND PAPER PRODUCTS AND FROM PROCESSING AND COMMERCIAL USE OF PULP AND PAPER MILL SLUDGE	4-1
4.1	Introduction	4-1
4.2	Worker Exposure to Dioxins in the Manufacture, Processing, and Commercial Use of Pulp, Paper, and Paper Products	4-1
4.2.1	Pulp and Paper Industry Workforce Characterization	4-2
4.2.2	Worker Exposure Estimating Methodologies	4-7
4.2.3	Summary of Worker Inhalation and Dermal Exposure, Individual Cancer Risks, and Population Cancer Risks	4-19
4.3	Worker Exposure from Processing and Commercial Use of Pulp and Paper Mill Sludge	4-20
4.3.1	Sludge Formation, Processing, and Disposal Operations	4-20
4.3.2	Dioxins in Pulp and Paper Mill Sludges	4-25
4.3.3	Sludge Handling/Disposal Workforce Characterization	4-26

TABLE OF CONTENTS (continued)

		Page No.
4.3.4	Worker Exposure Estimating Methodologies	4-28
4.3.5	Summary of Worker Exposure, Individual Cancer Risks, and Population Cancer Risks from Processing and Commercial Use of Sludge	4-35
4.4	Analysis of Uncertainties	4-35
4.4.1	Worker Exposure from Manufacture, Processing, and Commercial Use of Pulp, Paper, and Paper Products	4-35
4.4.2	Worker Exposure from Sludge Processing and Commercial Use	4-38
4.5	References	4-39
5.	ASSESSMENT OF RISKS TO THE GENERAL POPULATION FROM EXPOSURE TO DIOXINS AND FURANS DURING USE AND DISPOSAL OF PULP AND PAPER MILL SLUDGE AND DISPOSAL OF PAPER WASTES	5-1
5.1	Introduction	5-1
5.2	Estimates of Exposure and Risks to the General Populations from Disposal and Use of Sludge from the Pulp and Paper Industry and Disposal of Paper Products	5-3
5.2.1	Exposure and Risks from Disposal of Pulp and Paper Sludge in Landfills	5-5
5.2.2	Exposure and Risks from Disposal of Paper Products in Municipal Landfills	5-18
5.2.3	Exposures and Risks from Disposal of Pulp and Paper Sludge in Surface Impoundments	5-26
5.2.4	Exposures and Risks from Land Application of Pulp and Paper Mill Sludge	5-39
5.2.5	Exposures and Risks from Distribution and Marketing of Pulp and Paper Sludge	5-74
5.3	Analysis of Uncertainty	5-96
5.4	Conclusions	5-99
5.5	References	5-101
6.	ASSESSMENT OF RISKS TO THE GENERAL POPULATION FROM THE DISCHARGE OF EFFLUENTS FROM THE PULP AND PAPER INDUSTRY.....	6-1
6.1	Introduction	6-1
6.1.1	Purpose	6-1
6.1.2	Scope	6-1

TABLE OF CONTENTS (continued)

		Page No.
6.2	Exposure and Risk Assessment Methodology Requirements	6-2
6.3	Exposure Assessment Methodology	6-3
6.4	Risk Assessment Methodology	6-8
6.4.1	Bioavailable Dose from Ingestion of Contaminated Fish Tissue and Drinking Water	6-8
6.5	Results of the Assessment	6-10
6.5.1	Exposure Assessment Results	6-11
6.5.2	Risk Assessment Results	6-14
6.6	Discussion of Results	6-23
6.6.1	Assumptions, Limitations, and Uncertainties	6-23
6.6.2	Conclusions	6-29
6.7	References	6-29
7.	ASSESSMENTS OF RISKS TO THE GENERAL POPULATION FROM EXPOSURE TO DIOXINS AND FURANS RESULTING FROM PULP/PAPER WASTEWATER SLUDGE INCINERATION	7-1
7.1	Introduction	7-1
7.2	Methodology	7-1
7.2.1	Unit Risk Estimate	7-1
7.2.2	EPA Human Exposure Model (HEM) (Background)	7-1
7.2.3	Pulp and Paper Mill Source Data	7-2
7.2.4	Risk Calculations	7-8
7.3	Results	7-9
7.4	Analytical Uncertainties	7-9
7.4.1	The Unit Risk Estimate	7-9
7.4.2	Emission Estimates	7-9
7.4.3	Sludge Burning at Municipal Incinerators	7-15
7.4.4	Exposure Assumptions	7-15
7.4.5	Conclusions	7-16
7.5	References	7-17

TABLE OF CONTENTS (continued)

		Page No.
8.	ASSESSMENT OF CANCER RISK FROM EXPOSURE TO PCDDs AND PCDFs IN CONSUMER PRODUCTS	8-1
	8.1 Introduction	8-1
	8.2 Methodology	8-1
	8.2.1 Exposure Assessment	8-1
	8.2.2 Risk Assessment	8-11
	8.3 Results	8-14
	8.3.1 Individual Cancer Risk	8-14
	8.3.2 Population Cancer Risk	8-17
	8.3.3 Non-Cancer Endpoints	8-17
	8.4 Uncertainty Analysis	8-17
	8.4.1 Liquid Mediated Extraction	8-17
	8.4.2 Unmediated Migration	8-19
	8.4.3 Use of *in vitro* Percutaneous Absorption Data	8-19
	8.4.4 Factors Affecting Percutaneous Absorption	8-19
	8.5 References	8-20
9.	ASSESSMENT OF RISKS TO THE GENERAL POPULATION EXPOSURE TO DIOXINS AND FURANS RESULTING FROM THE USE OF PULP-CONTAINING MEDICAL DEVICES	9-1
	9.1 Introduction	9-1
	9.2 Estimates of Exposures and Risks from Dermal Contact with Pulp-Containing Medical Devices	9-1
	9.2.1 Exposure Parameters	9-1
	9.2.2 Exposure/Risk Assessment for Medical Devices	9-8
	9.3 Uncertainty Analysis	9-11
	9.4 Conclusions	9-12
	9.5 References	9-13

TABLE OF CONTENTS (continued)

Page No.

10. ASSESSMENT OF RISKS TO THE GENERAL POPULATION FROM DIOXINS AND FURANS IN FOODS PACKAGED IN OR CONTACTING BLEACHED PAPER PRODUCTS 10-1

10.1 Introduction 10-1
10.2 Dioxin Concentrations in Bleached Wood Pulp and Paper Food-Contact Articles 10-2
10.3 Food-Paper Migration Studies 10-3

10.3.1 Types of Articles Investigated 10-3
10.3.2 Conclusions from the Migration Studies 10-5

10.4 Food Intake Information 10-6
10.5 Estimated Exposures 10-7

10.5.1 Estimated Total Dioxin TEQ Intake: All Foods Contacting Bleached Paper 10-7
10.5.2 Estimated Dioxin TEQ Intake: Food-by-Food Basis for Individual Paper Products 10-10

10.6 Risk Assessment 10-10

10.6.1 Cancer Risks 10-10
10.6.2 Non-Cancer Risks 10-13
10.6.3 Uncertainties 10-13

10.7 References 10-14

11. ASSESSMENT OF RISKS TO THE GENERAL POPULATION FROM DIOXINS AND FURANS IN CELLULOSE DERIVATIVES USED IN FOOD, DRUG, AND COSMETIC FORMULATIONS 11-1

11.1 Introduction 11-1
11.2 Exposure to Dioxins and Furans from Use of Cosmetic Products 11-1

11.2.1 Identity and Use of Cellulose Derivatives 11-1
11.2.2 Dioxin Concentration in Cellulose Derivatives 11-1
11.2.3 Cosmetic Product Use 11-2
11.2.4 Dermal Absorption of Dioxin from Cosmetic Products Applied to the Skin 11-3
11.2.5 Dioxin Exposure from Cosmetic Products Applied to the Skin 11-3

TABLE OF CONTENTS (continued)

Page No.

11.3 Exposure to Dioxins and Furans from Use of Cosmetic Wet Wipes 11-3

11.3.1 Identity and Use of Cosmetic Wet Wipes 11-3
11.3.2 Dioxin Concentration In and Extraction From Wet Wipes 11-5
11.3.3 Wet Wipe Use Information 11-5
11.3.4 Dermal Absorption of Dioxin Congeners 11-6
11.3.5 Dioxin Exposure from Wet Wipes 11-6

11.4 Exposure to Dioxins and Furans in Cellulose Derivatives Used in Foods and Drug Products 11-6

11.4.1 Identity and Use of Cellulose and Cellulose Derivatives 11-6
11.4.2 Dioxin Concentration in Cellulose Derivatives 11-8
11.4.3 Dioxin Exposure Estimates 11-8

11.5 Cancer Risk Estimates 11-14
11.6 Analysis of Uncertainties 11-14
11.7 References 11-17

12. FDA ASSESSMENT OF RISKS FROM EXPOSURE TO DIOXIN AND FURANS IN FISH CONTAMINATED BY BLEACHED KRAFT PULP AND PAPER MILLS 12-1

12.1 Introduction 12-1
12.2 Levels of Dioxin Congeners in Fish 12-1
12.3 Sources of Information on Fish Intake 12-2
12.4 Estimation of Fish Intake by Subsistence and Sports Fishers and Their Families 12-5

12.4.1 Subsistence Fishers 12-5
12.4.2 Sports Fishers 12-6

12.5 Risk Assessment for Cancer 12-7
12.6 Non-Cancer Toxicological Effects of Dioxins 12-9
12.7 Risk of Non-Cancer Toxicological Effects to Subsistence and Sports Fishers 12-10
12.8 References 12-11

TABLE OF CONTENTS (continued)

Page No.

13. ESTIMATES OF RISKS TO TERRESTRIAL AND AVIAN WILDLIFE FROM LAND APPLICATION OF PULP AND PAPER MILL SLUDGE AND TO AQUATIC LIFE FROM DISCHARGE OF EFFLUENTS 13-1

13.1 Introduction 13-1
13.2 Terrestrial and Avian Wildlife Risk Assessment 13-2

13.2.1 Development of Benchmark Doses to Which Terrestrial Wildlife Exposures (Adjusted for Adsorption) Are Compared 13-2
13.2.2 Estimating Exposures to Terrestrial Wildlife . 13-4
13.2.3 Summary of Results: Terrestrial Wildlife 13-21

13.3 Aquatic Life Risk Assessment 13-25

13.3.1 Risk to Fish 13-25
13.3.2 Risks to Aquatic Plants and Herbivores 13-27
13.3.3 Risks to Benthic Organisms 13-28
13.3.4 Risks to Fish-Eating Birds and Mammals 13-29

13.4 References 13-29

APPENDIX A - Bioavailability A-1
APPENDIX B - Summary Information on Chlorinated Chemicals Other than PCDDs and PCDFs (OCOs) Identified in Pulp Mill Effluents, Sludges, and Pulps B-1

LIST OF TABLES

		Page No.
Table 1-1.	Concentration of 2,3,7,8-TCDD and 2,3,7,8-TCDF in Unbleached and Bleached Kraft Pulps from the Five-Mill Study (Cooperative Dioxin Screening Study) (ppt)	1-3
Table 1-2.	Concentration of 2,3,7,8-TCDD and 2,3,7,8-TCDF in Filtrates from Various Stages of Bleaching Process in the Five-Mill Study (Cooperative Dioxin Screening Study) (ppt)	1-5
Table 1-3.	Summary Results of the 104-Mill Study	1-6
Table 1-4.	Concentration (ppt) of 2,3,7,8-TCDD and 2,3,7,8-TCDF Consumer Products	1-8
Table 1-5.	Exposure Routes and Pathways Examined for Each Exposure/Risk Assessment Performed for the Dioxin-in Paper Project	1-10
Table 1-6.	Effect of Defoamer Addition to Brownstock Kraft Pulp on Subsequent Dioxin Formation During the Bleaching Process	1-19
Table 2-1.	Possible Number of Positional PCDD and PCDF Isomers	2-2
Table 2-2.	Summary of Estimated Physical/Chemical Properties of PCDFs and PCDDs	2-4
Table 2-3.	Physical and Chemical Properties of 2,3,7,8-TCDD	2-6
Table 2-4.	Summary of Environmental Fate of Dibenzo-*p*-Dioxins	2-12
Table 2-5.	Physical and Chemical Properties of 2,3,7,8-TCDF	2-20
Table 2-6.	Summary of Environmental Fate of Dibenzofurans	2-26
Table 3-1.	Factors Used by EPA, FDA, and CPSC in Calculating Their Risk Estimates for 2,3,7,8-TCDD Using Multistage Models	3-4
Table 3-2.	Terms Associated with Dose-Response Modeling	3-5
Table 3-3.	I-TEFs/89 and TEFs Developed by Other Groups	3-11

LIST OF TABLES (continued)

		Page No.
Table 3-4.	International Toxicity Equivalency Factors/89 (I-TEFs/89): Comparison of Relative Potency Data for the 2,3,7,8-Substituted CDDs and CDFs	3-13
Table 3-5.	Aquatic Toxicity Data for Chlorinated Dibenzo-p-Dioxins	3-17
Table 3-6.	Aquatic Toxicity Data for Chlorinated Dibenzofurans	3-23
Table 3-7.	Summary of Studies on the Toxicity of 2,3,7,8-TCDD to Wildlife Mammals and Birds	3-29
Table 4-1.	Total Employees and Production Workers in the Entire Paper and Allied Products Industry, 1985 ...	4-3
Table 4-2.	Number of Workers in the Pulp and Pulp Products Job Categories	4-6
Table 4-3.	Summary of Individual and Population Cancer Risks for Workers Involved in the Manufacturing, Processing, and Commercial Usage of Pulp, Paper, and Paper Products	4-21
Table 4-4.	Summary of Outer Bounds of Individual and Population Cancer Risks for Workers Involved in Processing, and Commercial Usage of Pulp, and Paper Mill Sludge ...	4-36
Table 5-1.	Distribution of 2,3,7,8-TCDD and 2,3,7,8-TCDF Sludge Concentrations for All Plants in 104 Mill Study ...	5-2
Table 5-2.	Exposure Pathways Evaluated for Each Pulp and Paper Mill Sludge Disposal or Use Practice	5-4
Table 5-3.	Physical/Chemical Properties and Fate/Transport Assumptions: All Exposure Pathways	5-6
Table 5-4.	Assumptions and Parameter Values - Landfills: All Exposure Pathways	5-7
Table 5-5.	Assumptions and Parameter Values - Landfills: Volatilization Pathway	5-10

LIST OF TABLES (continued)

		Page No.
Table 5-6.	Assumptions and Parameter Values - Landfills: Ground-Water Pathways	5-12
Table 5-7.	Assumptions and Parameter Values - Landfills: Surface Water Pathways	5-16
Table 5-8.	Estimates of Health Risks to the General Public fromLandfill Disposal of Pulp and Paper Sludges Contaminated with 2,3,7,8-TCDD and 2,3,7,8-TCDF	5-19
Table 5-9.	Site and Waste Characteristics for Municipal Landfills Receiving Waste Paper Contaminated with TCDD and TCDF	5-20
Table 5-10.	Assumptions and Parameter Values - Paper Wastes in Municipal Landfills: Ground-Water Pathway	5-24
Table 5-11.	Estimates of Health Risks to the General Population from Landfill Disposal of Paper Contaminated with 2,3,7,8-TCDD and 2,3,7,8-TCDF	5-27
Table 5-12.	Assumptions and Parameter Values - Surface Impoundments: All Exposure Pathways	5-29
Table 5-13.	Assumptions and Parameter Values - Surface Impoundments: Volatilization Pathway	5-30
Table 5-14.	Assumptions and Parameter Values - Surface Impoundments: Ground-Water Pathway	5-33
Table 5-15.	Assumptions and Parameter Values - Surface Impoundments: Surface Water Pathway	5-37
Table 5-16.	Estimates of Health Risks to the General Population from Surface Impoundment of Pulp and Paper Sludges Contaminated with 2,3,7,8-TCDD and 2,3,7,8-TCDF	5-40
Table 5-17.	Assumptions and Parameter Values - Land Application: All Exposure Pathways	5-42
Table 5-18.	Assumptions and Parameter Values - Land Application: Dermal Pathway	5-44

LIST OF TABLES (continued)

		Page No.
Table 5-19.	Assumptions and Parameter Values - Agricultural Application: Dietary Pathway	5-51
Table 5-20.	Assumptions and Parameter Values - Land Application: Soil Ingestion Pathway	5-61
Table 5-21.	Assumptions and Parameter Values - Land Application: Vapor and Particulate Inhalation Pathways	5-63
Table 5-22.	Assumption and Parameter Values - Land Application: Surface Water Pathways	5-70
Table 5-23.	Estimates of Health Risks to the General Population from Land Application of Pulp and Paper Sludges Contaminated with 2,3,7,8-TCDD and 2,3,7,8-TCDF ...	5-75
Table 5-24.	Distribution and Marketing Sludge and Soil Contaminant Concentration	5-78
Table 5-25.	Parameters and Model Inputs Used to Model: Distribution and Marketing	5-79
Table 5-26.	Assumptions and Parameter Values - Distribution and Marketing: Dermal Pathway	5-82
Table 5-27.	Assumptions and Parameter Values - Distribution and Marketing: Dietary Pathway	5-88
Table 5-28.	Assumptions and Parameter Values - Distribution and Marketing: Soil Ingestion Pathway	5-90
Table 5-29.	Assumptions and Parameter Values - Distribution and Marketing: Vapor and Particulate Inhalation Pathways	5-94
Table 5-30.	Estimates of Health Risks to the General Population from Distribution and Marketing of Pulp and Paper Sludges Contaminated with 2,3,7,8-TCDD and 2,3,7,8-TCDF	5-97

LIST OF TABLES (continued)

		Page No.
Table 5-31.	Maximum General Population Health Risks from Exposure to TCDD and TCDF, by Waste Management Method	5-100
Table 7-1.	Locations of Pulp and Paper Mill Wastewater Sludge Incinerators	7-3
Table 7-2.	Pulp and Paper Mill Incinerator Stack Parameters ..	7-4
Table 7-3.	Dioxin TEQ and Furan TEQ Emission Rates Based on Reported Stack Gas Concentrations	7-5
Table 7-4.	Dioxin/Furan Emissions and Stack Flow Rate Data Used to Calculate Emission Rates Based on Stack Gas Concentrations	7-6
Table 7-5.	2,3,7,8-TCDD and 2,3,7,8-TCDF Emission Rates Based on Site-Specific Concentrations in Sludge	7-7
Table 7-6.	Site-Specific Exposure Analysis Based on 2,3,7,8-TCDD Concentrations in Sludge (Maximum Radius = 50 km) .	7-10
Table 7-7.	Site-Specific Exposure Analysis Based on 2,3,7,8-TCDF Concentration in Sludge (Maximum Radius = 50 km) ..	7-11
Table 7-8.	Site-Specific Exposure Analysis Based on Dioxin Concentrations in Stack Gas (Expressed as dioxin TEQs) - Maximum Values (Maximum Radius = 50 km) ...	7-12

LIST OF TABLES (continued)

		Page No.
Table 7-9.	Site-Specific Exposure Analysis Based on Furan Concentrations in Stack Gas (expressed as dioxin TEQs) - Maximum Values (Maximum Radius = 50 km)	7-13
Table 7-10.	Combined Dioxin/Furan Risks and Annual Incidence ..	7-14
Table 8-1.	Concentrations (ppt) of 2,3,7,8-TCDD and 2,3,7,8-TCDF in Consumer Products	8-3
Table 8-2.	Parameters for Estimating Exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF in Disposable Infant Diapers	8-6
Table 8-3.	Parameters for Estimating Exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF in Paper Towels	8-8
Table 8-4.	Parameters for Estimating Exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF in Facial Tissue	8-9
Table 8-5.	Parameters for Estimating Exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF in Toilet Tissue	8-10
Table 8-6.	Parameters for Estimating Exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF in Communications Paper	8-12
Table 8-7.	Parameters for Estimating Exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF in Paper Napkins	8-13
Table 8-8.	Individual Lifetime and Population Cancer Risks from 2,3,7,8-TCDD and 2,3,7,8-TCDF in Consumer Products .	8-15
Table 8-9.	Risks for Non-Cancer Adverse Effects from 2,3,7,8-TCDD and 2,3,7,8-TCDF in Consumer Paper Products...	8-18
Table 9-1.	Medical Devices for Which Exposures and Risks Were Estimated and Their Corresponding Uses	9-2
Table 9-2.	Exposure/Risk Parameters for Medical Devices	9-3
Table 9-3.	Average Concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF in Pulp Calculated Based on Results from the 104-Mill Data Base	9-5

LIST OF TABLES (continued)

		Page No.
Table 9-4.	Concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF in Pulp at Pulp Mills that Produce Pulp Dissolving Cellulose	9-6
Table 9-5.	Estimates of Risks to the General Population from the Use of Pulp-Containing Medical Devices	9-9
Table 10-1.	Representative Dioxin Congener Levels in Pulp and Paper Matrices	10-4
Table 10-2.	Carcinogenic Risk for Consumers Resulting from Total Dioxin TEQ Intake from All Foods Contacting Bleached Paper ("mean consumer - total sample basis")	10-8
Table 10-3.	Industry-Based Per Capita Dioxin TEQ Exposures	10-9
Table 10-4.	Upper Bound Carcinogenic Risk for Consumers of Foods Containing Bleached Paper Contaminated with Dioxin ("eaters only - food-by-food basis")	10-11
Table 11-1.	Dioxin Exposure from Cosmetic Products Applied to the Skin	11-4
Table 11-2.	Dioxin Exposure from Wet Wipes	11-7
Table 11-3.	Total Sample Basis Dioxin TEQ Exposure from Foods	11-11
Table 11-4.	Upper Bound Intake of Dioxin TEQ from Drugs	11-15
Table 11-5.	Upper Bound Carcinogenic Risk for Users of Food, Drug, and Cosmetic Products Containing Cellulose Derivatives	11-16
Table 12-1.	Dioxin TEQ Intake by Subsistence and Sports Fishers	12-3
Table 12-2.	Cancer Risk for Subsistence and Sports Fishers	12-8

LIST OF TABLES (continued)

		Page No.
Table 13-1.	Assumptions and Parameter Values - Land Application: Wildlife Exposures	13-8
Table 13-2.	Characteristics of Land Application Sites and Soil Concentrations Used in Wildlife Analysis	13-13
Table 13-3.	Body Weights and Daily Food Consumption of Animals Affected by Land Application of Sludge	13-14
Table 13-4.	Mix of Food Sources for Birds and Mammals	13-15
Table 13-5.	Estimates of Exposure and Risk to Adult and Hatching Birds from 2,3,7,8-TCDD in Sludge-Treated Land.	13-
Table 13-6.	Estimates of Exposure and Risk to Mammals from 2,3,7,8-TCDD in Sludge-Treated Land	13-
Table 13-7.	Summary of Risks to Birds ("Best Estimate")	13-22
Table 13-8.	Summary of Risks to Bird Eggs ("Best Estimate")....	13-23
Table 13-9.	Summary of Risks to Mammals ("Best Estimate")	13-24
Table 13-10.	Results of Preliminary Search for Endangered and Threatened Species Found in the Counties Where Pulp and Paper Mills Are Located that Apply Dioxin- and Furan-Contaminated Pulp and Paper Mill Sludge to Land	13-26
Table 13-11.	Distribution of Dioxin Concentrations in Whole Fish Sampled in the National Bioaccumulation Study	13-30

LIST OF FIGURES

		Page No.
Figure 2-1.	Destruction Curves of 2,3,7,8-TCDD	2-14
Figure 6-1.	Distribution of the number of mills for which discharges would result in a given range of water column contaminant concentrations as estimated by the simple dilution method	6-12
Figure 6-2.	Distribution of the number of mills for which discharges would result in a given range of water column contaminant concentrations as estimated by the EXAMS II water column method	6-13
Figure 6-3.	Distribution of the number of mills for which discharges would result in a given range of lifetime cancer risk due to the consumption of contaminated fish tissue as estimated by the simple dilution method (6.5 g/day consumption rate and BCF of 5,000 for 2,3,7,8-TCDD)	6-16
Figure 6-4.	Distribution of the number of mills for which discharges would result in a given range of lifetime cancer risk due to the consumption of contaminated fish tissue as estimated by the simple dilution method (30 and 140 g/day consumption rates and BCF of 50,000 for 2,3,7,8-TCDD)	6-17
Figure 6-5.	Distribution of the number of mills for which discharges would result in a given range of lifetime cancer risk due to the consumption of contaminated fish tissue as estimated by the EXAMS II method (6.5 g/day consumption rate and BCF of 5,000 for 2,3,7,8-TCDD)	6-18
Figure 6-6.	Distribution of the number of mills for which discharges would result in a given range of lifetime cancer risk due to the consumption of contaminated fish tissues as estimated by the EXAMS II method (30 and 140 g/day consumption rates and BCF of 50,000 for 2,3,7,8-TCDD)	6-19

LIST OF FIGURES (continued)

Page No.

Figure 6-7. Distribution of the number of mills for which discharges would result in a given range of lifetime cancer risk due to the ingestion of contaminated drinking water as estimated by the simple dilution method 6-21

Figure 6-8. Distribution of the number of mills for which discharges would result in a given range of lifetime cancer risk due to the ingestion of contaminated drinking water as estimated by the EXAMS II method . 6-22

Figure 6-9. Distribution of the number of mills for which discharges would result in a given range of human doses from a one-time exposure to contaminated fish tissue as estimated by the simple dilution method .. 6-24

Figure 6-10. Distribution of the number of mills for which discharges would result in a given range of human doses from a one-time exposure to contaminated fish tissue as estimated by the EXAMS II method 6-25

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1. INTRODUCTION

1.1 Background

Various isomers of polychlorinated dibenzodioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) have been found to be formed during bleaching of wood pulp with chlorine or chlorine-based bleaching chemicals. Quantitative studies conducted by the U.S. Environmental Protection Agency (EPA) and the paper industry have shown that PCDDs and PCDFs may be retained in low levels in bleached pulp, crude paper products (e.g., newsprint, paperboard, fibers), and finished commercial and consumer grade pulp/paper-based products. Furthermore, PCDDs and PCDFs may also be retained in wastewater and sludge generated during the manufacture of these products.

On July 27, 1988, the EPA entered into a Consent Agreement with the Environmental Defense Fund and National Wildlife Federation concerning the regulation of PCDDs and PCDFs formed in pulp and paper processing. In response to the Consent Decree, EPA is conducting risk assessments for PCDDs and PCDFs from pulp and paper mill products and wastes. The Office of Toxic Substances (OTS) has the responsibility to fulfill the commitments as required by the Consent Decree. The assessment is being performed as an inter-Agency, inter-Office effort. Other Agencies involved are the Food and Drug Administration (FDA) and the Consumer Product Safety Commission (CPSC). Exposure and risk resulting from the direct use of bleached pulp and paper in food, drugs, cosmetics, and medical devices is being assessed by FDA. CPSC is evaluating exposure and risk from body contact papers that do not fall into the medical devices category and from other related consumer products. EPA is responsible for the assessment of risks to humans and wildlife resulting from the discharge of effluents from pulp and paper mills and the disposal of wastewater sludge generated at pulp and paper mills. EPA is also assessing risks to workers in the pulp and paper industry as well as processors and commercial users of pulp and paper products.

Upon completion of a multiple exposure pathway risk assessment, EPA must publish a proposed schedule for the regulation of dioxin (2,3,7,8-TCDD) from bleached chemical pulp mills, or publish the Agency's decision not to regulate. The Agency is under a legal consent agreement to consider risks to humans from disposal of sludges and water effluents from these mills as well as those associated with the use and disposal of products made from bleached paper pulp. These risks are to be considered from an occupational and non-occupational exposure perspective. Risks posed to aquatic organisms and wildlife are also to be evaluated.

In an attempt to quantify concentrations of dioxins produced during pulp and paper manufacturing operations, several studies were undertaken cooperatively by the EPA and the paper industry. These studies include the Five-Mill Study (USEPA 1988) and the 104-Mill Study (USEPA 1989).

As a result of finding 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) in native fish collected downstream from a number of pulp and paper mills in the EPA National Dioxin Study (USEPA 1987a) and subsequent findings of 2,3,7,8-TCDD in bleached kraft pulp and paper mill wastewater sludges, the EPA planned a detailed process evaluation at one mill. Through subsequent discussions with the paper industry, EPA and the industry agreed in June 1986 to conduct a cooperative screening study of five bleached kraft pulp and paper mills on a shared-resource basis. Three mills were selected on the basis of known 2,3,7,8-TCDD levels in sludges and two mills were volunteered by their parent companies to attain the geographical diversity desired for the study. The selection of the five mills, which represent about six percent of the bleached kraft mills in the United States, was not intended to characterize the entire industry. The principal objectives of the study, commonly referred to as the "Five-Mill Study," were to:

- Determine, if present, the source or sources of 2,3,7,8-TCDD and other polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) at five bleached kraft pulp and paper mills; and

- Quantify the untreated wastewater discharge loadings, final effluent discharge loadings, sludge concentrations, and wastewater treatment system efficiency for 2,3,7,8-TCDD and other PCDDs and PCDFs.

The Five-Mill Study was conducted during the period June 1986-January 1987 through the combined efforts of four EPA regional offices, five state environmental control agencies, the National Council of the Paper Industry for Air and Stream Improvement, Inc. (NCASI), and the participating paper companies. Samples of wastewater, sludge, and pulp were analyzed for PCDDs and PCDFs. The results uniformly showed that 2,3,7,8-TCDD and 2,3,7,8-TCDF were the principal PCDDs and PCDFs found. This was particularly evident when the data were considered in light of EPA's 2,3,7,8-TCDD toxicity equivalency factor (TEF) method of risk assessment for complex mixtures of PCDDs and PCDFs. (These findings were more recently confirmed in the 104-Mill Study--see below.)

The analysis further showed that 2,3,7,8-TCDD and 2,3,7,8-TCDF are formed during the bleaching of kraft hardwood and softwood pulps with chlorine and chlorine derivatives at the mills in the study. Table 1-1 presents concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF in unbleached and bleached Kraft pulps at the five mills. Although no 2,3,7,8-TCDD was detected in unbleached pulp, three of the seven samples tested above detection levels for 2,3,7,8-TCDF. The positive analysis for 2,3,7,8-TCDF in unbleached pulp may be due to reuse of dioxin-contaminated paper machine wastewater for brown stock pulping or dilution at the mill where the samples were taken. Most of the bleached pulp samples tested

Table 1-1. Concentration of 2,3,7,8-TCDD and 2,3,7,8-TCDF in Unbleached and Bleached Kraft Pulps from the Five-Mill Study (Cooperative Dioxin Screening Study) (ppt)

Pulp	2,3,7,8-TCDD				2,3,7,8-TCDF			
	No. of samples	Range	Mean	Median	No. of samples	Range	Mean	Median
Unbleached	7	ND (0.3-1)	ND	ND	7	<0.16-2.3	1.6	ND
Bleached	9	<0.6-51	13	5	9	<1.2-330	93	50

Source: USEPA (1988).

positively for 2,3,7,8-TCDD and 2,3,7,8-TCDF, which led to the conclusion that their formation was caused by the bleaching of Kraft pulp by chlorination.

Table 1-2 presents concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF in untreated filtrates from the various stages of the bleaching processes used at the five mills. All of the samples tested above detection levels for 2,3,7,8-TCDF. In five samples, 2,3,7,8-TCDD was not detected. Table 1-2 shows that filtrate wastewater from the caustic extraction stage generally contained the highest concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF, followed by the hypochlorite, chlorination, and chlorine dioxide stages. The bleach plant filtrate data do not clearly define the point of dioxin formation but do indicate formation in the chlorination (C) stage and possibly in the extraction (E) stage. It is not possible from the data to determine whether 2,3,7,8-TCDD and 2,3,7,8-TCDF are formed in the C stage and extracted in the E stage or if there is additional formation in the E stage. The data also suggest formation of these compounds in subsequent bleaching stages.

The EPA/Paper Industry Cooperative Dioxin Study, commonly referred to as the "104-Mill Study," was conducted from April 1988 to August 1989 at 104 domestic mills manufacturing chemical pulp. In this study, concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF were measured at 87 kraft (sulfate) and 17 sulfite pulp mills that use chlorine-based bleaching processes (chlorine, chlorine dioxide, or hypochlorite). Samples were taken of the following:

- Bleached pulp after the final stage of bleaching,
- Wastewater sludge, and
- Treated wastewater effluent.

The study also collected data on waste treatment operations, waste discharge characteristics, and sludge disposal information.

The 2,3,7,8-TCDD concentrations in sulfite pulp were much lower than those for the kraft pulp concentrations, ranging from 1 ppt to 15 ppt with a median of no detection corresponding to a detection level of 1 ppt. This observation confirmed the Five-Mill Study which showed the highest level of dioxin concentrations to be associated with the kraft process. The analytical results of the 104-Mill Study are presented in Table 1-3. The results indicate that the concentrations of 2,3,7,8-TCDF measured in the 104-Mill Study are greater than those of 2,3,7,8-TCDD, almost by an order of magnitude. Also, the results of this study confirm the finding of the Five-Mill Study that 2,3,7,8-TCDD and 2,3,7,8-TCDF are the principal PCDDs and PCDFs present. In this study, pulp, effluent, and sludge samples from 9 mills were analyzed for total non-2,3,7,8-substituted isomers and for 18 specific 2,3,7,8-substituted isomers.

Table 1-2. Concentration of 2,3,7,8-TCDD and 2,3,7,8-TCDF in Filtrates from Various Stages of Bleaching Process in the Five-Mill Study (Cooperative Dioxin Screening Study) (ppt)

Pulp	2,3,7,8-TCDD				2,3,7,8-TCDF			
	No. of samples	Range	Mean	Median	No. of samples	Range	Mean	Median
C Stages (C, C_D,C/d)[a]	8	0.02-0.24	0.07	0.05	8	0.068-3.8	0.65	0.24
E Stages (E,E_D)[b]	8	0.045-3.6	1.1	0.26	8	0.056-14	3.3	0.51
H Stages (H,H/D)[c]	10	0.025-1.9	0.40	0.19	10	0.086-9.2	2.3	0.59
D Stages (D)[d]	2	ND[e] (0.003)-0.03	0.015	NA[f]	2	0.014-0.13	0.072	NA[f]

[a]C Stage represents chlorination stage of the mills. C represents use of chlorine only. C_D represents use of a mixture of chlorine dioxide and chlorine, which is predominantly chlorine. C/D represents use of a mixture of chlorine and chlorine dioxide. 2,3,7,8-TCDD was not detected in one sample (C_D) with a detection level of 0.006 ppt.

[b]E Stage represents caustic extraction stage following the bleaching stages at the mills. E represents use of sodium hydroxide only. E_D represents use of a mixture of sodium hydroxide and oxygen, which is predominantly sodium hydroxide. 2,3,7,8-TCDD was not detected in two samples (both E_D) with detection levels of 0.011 and 0.033 ppt.

[c]H Stage represents hypochlorite stage of the mills. H represents use of sodium or calcium hypochlorite only. H/D represents use of a mixture of sodium or calcium hypochlorite and chlorine dioxide. 2,3,7,8-TCDD was not detected in one sample (H) with a detection limit of 0.017 ppt.

[d]D Stage represents chlorine dioxide stage at the mills. D represents use of chlorine dioxide only. 2,3,7,8-TCDD was not detected in one sample with a detection level of 0.003 ppt.

[e]ND = Not detected.

[f]NA = Not applicable.

Source: USEPA (1988).

Table 1-3. Summary Results of the 104 Mill Study[a]

	Range[b] (ng/kg)	Mean (ng/kg)	Median (ng/kg)	Std. Dev. (ng/kg)	No. of mills with no detected values
Pulp					
2,3,7,8-TCDD	ND-116	8.8	4.9	11.8	21
2,3,7,8-TCDF	ND-2,620	94.9	19.0	283.7	6
Effluent					
2,3,7,8-TCDD	ND-0.640	0.068	0.023	0.106	20
2,3,7,8-TCDF	ND-8.400	1.033	0.094	2,358	7
Sludge					
2,3,7,8-TCDD	ND-3,800	77.5	18.0	163.9	2
2,3,7,8-TCDF	2.4-17,100	749.6	89.0	2,079	0

[a] Based on final results obtained from EPA's Office of Water Regulations and Standards in October 1989.

[b] The analytical objectives for detection limits of both compounds were 0.01 ng/kg (or ppt) for effluents and 1 ppt for pulps and sludges.

Based on the 1987 TEF method (USEPA 1987b), the results show that 2,3,7,8-TCDD and 2,3,7,8-TCDF generally account for more than 90 percent of the dioxin toxic equivalents (TEQ) found in pulp, sludge, and effluent (Helms 1989).

In addition to the EPA studies, the National Council of the Paper Industry for Air and Stream Improvement (NCASI) and others have performed some analyses for 2,3,7,8-TCDD and 2,3,7,8-TCDF in composites of several bleached pulp-based products. Table 1-4 summarizes the results of these analyses.

1.2 Purpose and Scope

The purpose of this background document is to present an integrated, multiple exposure pathway assessment of risks resulting from exposure to PCDDs and PCDFs formed during the production of bleached wood pulp. The assessment evaluates and estimates exposure and risk to humans, aquatic organisms, and wildlife, and is based on existing data that have been presented in reports generated for the EPA, CPSC, and FDA. The individual assessments from which this background document was developed encompass the following exposure and risk categories:

- An analysis of the chemistry and environmental fate of 2,3,7,8-TCDD and 2,3,7,8-TCDF;
- An assessment of the hazard/toxicity of 2,3,7,8-TCDD and 2,3,7,8-TCDF to humans, aquatic organisms, and avian and terrestrial wildlife;
- An assessment of exposures and risks to workers in the pulp and paper industry;
- Assessments of exposures and risks to the general population from:
  - use and disposal of pulp and paper mill wastewater sludge and land disposal of paper waste
  - discharge of effluents from the pulp and paper industry
  - incineration of pulp and paper mill wastewater sludge
  - use of pulp-containing medical devices
  - use of paper consumer products
  - ingestion of foods packaged in or contacting bleached paper products

Table 1-4. Concentrations (ppt) of 2,3,7,8-TCDD and 2,3,7,8-TCDF in Consumer Products

Product	2,3,7,8-TCDD	2,3,7,8-TCDF	Reference
Disposable diapers	ND (2.6)	8.8	NCASI (1987); Blosser (1987)
	ND (2.1)	7.2	NCASI (1987); Blosser (1987)
	ND	3.7	WJLA TV[a]
Paper towels	3.7	32	NCASI (1987)
Bond paper	13	290	NCASI (1987)
	13	240	NCASI (1987)
Facial tissue	1.1	13	Beck et al. (1988)
Scrap paper	0.6	13	Beck et al. (1988)
Newsprint	ND	ND	Beck et al. (1988)
Tissue	1.3	31.1	LeBel et al. (1989)

[a] Assays for WJLA TV were performed by Triangle Laboratories, Research Triangle Park, NC.

ND = Not detected; the number in parentheses is the detection limit.

- use of food, drug, and cosmetic products containing cellulose derivatives;

- An assessment of exposures and risks to avian and terrestrial wildlife from land application of sludge and to aquatic organisms from the discharge of pulp and paper mill effluents; and

- An analysis of information on the properties and toxicity of other chlorinated chemicals (OCOs), those organic chemicals other than PCDDs and PCDFs, that are present in pulp and paper mill effluents, sludges, and pulps.

Each pertinent report(s) is cited as the source(s) for the chapters for which they have been used. The reader should refer to the original source documents for specific citations to the many information sources used in each assessment.

As mentioned above, this assessment addressed the range of exposure routes resulting from the production and use of bleached paper products. In certain cases, many specific exposure scenarios were evaluated within each exposure route. Table 1-5 provides a summary of each exposure scenario addressed within each exposure route.

Although many scenarios using different methodologies were assessed by the various agencies and offices participating in the Dioxin-in-Paper Project, several common assumptions were agreed to by the participants.

1. The Toxicity Equivalency Factor (TEF) method states that 2,3,7,8-TCDF is assumed to have one-tenth the potency of 2,3,7,8-TCDD. EPA and FDA agreed to employ this policy for this assessment. Because CPSC does not place similar emphasis on risks calculated by the TEF method as it does for 2,3,7,8-TCDD, it was agreed that, to the extent possible, CPSC risk estimates for each scenario would be based on the contribution to risk of 2,3,7,8-TCDD alone.

2. The assessments focused on exposures and risks to 2,3,7,8-TCDD and 2,3,7,8-TCDF. Based on the TEF values formally adopted by EPA in 1987, the results of the "5-Mill Study" indicated and the results of the "104-Mill Study" confirmed that these two dioxin congeners generally account for more than 90 percent of the dioxin toxic equivalents (TEQ) found in pulps, sludges, and effluents from the pulp and paper mill samples analyzed.

3. EPA, FDA, and CPSC have each derived an estimated slope factor (q_1^* or q_1) for 2,3,7,8-TCDD based on linear-at-low-dose extrapolation procedures. However, because the Agencies differ with respect to selection of animal data and details of

Table 1-5. Exposure Scenarios and Pathways Examined for Each Exposure/Risk Assessment Performed for the Dioxin-in-Paper Project

Assessment	Scenarios	Pathways/Products
Exposures and risks to workers in the pulp and paper industry	Exposure to bleach operators, pulp testers, and utility operators during manufacture	• Inhalation of volatilized TCDD and TCDF • Dermal contact with pulp
	Exposure to operators during pulp drying	• Inhalation of volatilized TCDD and TCDF • Dermal contact with pulp
	Exposure to wet-end operators during paper manufacture	• Inhalation of volatilized TCDD and TCDF • Dermal contact with paper
	Exposure to dry-end and utility operators during paper manufacture	• Inhalation of volatilized TCDD and TCDF • Inhalation of contaminated dust • Dermal contact with paper
	Exposure to workers during paper converting operations	• Inhalation of contaminated dust • Dermal contact with paper
	Exposure to workers during nonwovens manufacture	• Inhalation of contaminated dust • Dermal contact with paper
	Exposure to commercial users of paper	• Dermal contact with paper
	Exposure to waste treatment plant operators and sludge haulers/front-end loader operators during sludge handling/processing	• Inhalation of volatilized TCDD and TCDF • Inhalation of contaminated particulates • Dermal contact with sludge
	Exposure to equipment operators during land-filling and land applications operations	• Inhalation of volatilized TCDD and TCDF • Inhalation of contaminated particulates • Dermal contact with sludge
	Exposure to equipment operators, compost haulers, and screen operators during composting operations	• Inhalation of volatilized TCDD and TCDF • Inhalation of contaminated particulates • Dermal contact with sludge

Table 1-5. (Continued)

Assessment	Scenarios	Pathways/Products
Exposures and risks resulting from the use and disposal of sludge from the pulp and paper industry and land disposal of paper products	Exposures to individuals residing near or utilizing surface or ground waters near landfills in which pulp and paper mill sludge is disposed	• Inhalation of volatilized TCDD and TCDF • Ingestion of drinking water from ground water sources • Ingestion of drinking water from runoff-contaminated surface water sources • Ingestion of fish from runoff-contaminated surface water
	Exposures to individuals residing near or utilizing ground water near municipal landfills in which paper wastes are disposed	• Inhalation of volatilized TCDD and TCDF • Inhalation of drinking water from contaminated ground water sources
	Exposures to individuals residing near or utilizing surface or ground waters near surface impoundments in which pulp and paper mill sludge is disposed.	• Inhalation of volatilized TCDD and TCDF • Ingestion of drinking water from contaminated ground water sources • Ingestion of drinking water from runoff-contaminated surface water sources • Ingestion of fish from runoff-contaminated surface water
	Exposures to individuals residing near or utilizing surface or ground waters near sites at which sludge is land-applied	• Inhalation of volatilized TCDD And TCDF • Inhalation of contaminated particulates • Ingestion of drinking water from contaminated ground water sources • Ingestion of drinking water from runoff-contaminated surface water sources • Ingestion of fish from runoff-contaminated surface water • Ingestion of contaminated soil • Ingestion of foods produced with contaminated soil • Dermal contact with contaminated soil

Table 1-5. (Continued)

Assessment	Scenarios	Pathways/Products
	Exposures to individuals utilizing commercially distributed pulp and paper mill sludge as a soil amendment	• Inhalation of volatilized TCDD and TCDF • Inhalation of contaminated particulates • Ingestion of contaminated soil • Ingestion of foods produced with contaminated soil • Dermal contact with contaminated soil
Exposures and risks to the general population from the discharge of pulp and paper mill effluents	Exposures to individuals utilizing surface waters downstream from pulp and paper mill effluents	• Ingestion of drinking water • Ingestion of fish
Exposures and risks to aquatic organisms from the discharge of pulp and paper mill effluents	Exposures to aquatic organisms directly downsteam from pulp and paper mill effluent discharge	• Exposure under 7Q10 flow conditions
Exposures and risks to the general population from the incineration of pulp and paper mill sludge	Exposures to individuals residing near incinerators in which pulp and paper mill sludge is disposed	• Inhalation of TCDD and TCDF in incinerator emissions
Exposures and risks to the general population from the use of pulp-containing medical devices	Exposures from direct contact with various medical devices under FDA jurisdiction	• Dermal contact with: - menstrual pads and tampons - alcohol pads - skin preparation wipes - absorbable hemostatic agents - surgical apparel - adult diapers - medical disposable bedding - medical absorbent fiber - absorbent-tipped applicators - examination gowns - opthalmic sponges - hydroxypropymethyl cellulose - cottonoid paddies - electro-conductive media - cutaneous electrodes - isolation gowns

Table 1-5. (Continued)

Assessment	Scenarios	Pathways/Products
Exposures and risks to the general population from the use of pulp and paper products	Exposures from direct contact with a variety of consumer products under CPSC jurisdiction	• Dermal contact with: - disposable infant diapers - paper towels - facial tissue - toilet tissue - communications paper - paper napkins
Exposures and risks to the general population from the use of food and drug products containing cellulose derivatives and from the use of paper food-contact articles	Exposures from ingestion of food products and drugs that contain cellulose derivatives	• Ingestion of foods and drugs containing: - powdered cellulose - microcrystalline cellulose - methylcellulose - carboxymethylcellulose - other cellulose derivatives
	Exposures from ingestion of foods packaged in or contacting bleached paper	• Ingestion of foods in contact with the following articles: - dairy cartons - juice cartons - bakery cartons - ovenable board - paper cups, plates, and trays - coffee filters - tea bags - microwave popcorn bags - butter/margarine wraps
Expoures and risks to the general population from the use of cosmetic products containing cellulose derivatives	Exposures from application of cosmetic products to the skin and use of dentifrice	• Dermal use of the following products: - lotions - shampoo - wet wipes • Ingestion of dentrifice
Exposures and risks to terrestrial and avian wildlife from land application of sludge	Exposures and risks to birds, bird eggs, and mammals	• Ingestion of contaminated soil • Ingestion of contaminated prey items

extrapolation, the risk estimates differ by as much as a factor of 10. The Agencies agreed that this Integrated Assessment would report cancer risk estimates calculated by each Agency.

4. The analytical results of the 104-Mill Study (i.e., 2,3,7,8-TCDD and 2,3,7,8-TCDF concentrations in pulp, effluents, and sludge) were to be used in all assessments unless use of alternate data (e.g., product-specific concentrations) could be justified.

5. With the exception of risks calculated by EPA/OSW, EPA/OW, and FDA/CFSAN, all estimated human cancer risks were calculated by multiplying the estimated lifetime average daily doses (LADD) by the slope factor (q_1 or q_1*) for 2,3,7,8-TCDD and dividing by the fraction of TCDD absorbed (A) during the animal bioassay from which the slope factor was derived:

$$\text{Risk} = (\text{LADD} \times q_1)/A$$

The value of A depends on the fraction of 2,3,7,8-TCDD absorbed by the test animals during the bioassay used to estimate q_1 or q_1*. For the EPA and FDA slope factors, which are based on a dietary bioassay, A is assumed to equal 0.55 since it was estimated that 55 percent of the 2,3,7,8-TCDD was absorbed by the test animals; similarly, for the CPSC slope factor, which is based on a gavage bioassay, A is assumed to equal 0.75. The total or population risk was estimated by multiplying the average lifetime risk by the number of persons exposed and dividing by the average life expectancy.

6. With the exception of the bioavailablity values used by EPA/OSW for each ingestion pathway applicable to sludge disposal and reuse, standard values developed for the bioavailability, or fraction of 2,3,7,8-TCDD and 2,3,7,8-TCDF absorbed, were developed and used for each exposure route and pathway. For the inhalation exposure route, the bioavailability was assumed to be 100 percent for 2,3,7,8-TCDD and 2,3,7,8-TCDF vapors and 100 percent for particulate-bound 2,3,7,8-TCDD and 2,3,7,8-TCDF that reach the alveoli. Standard values for the bioavailability of 2,3,7,8-TCDD and 2,3,7,8-TCDF were assumed to be 100 percent from ingestion of drinking water, 85 to 95 percent for ingestion of fatty or oily foods (e.g., milk, fish, meats), 60 to 70 percent for ingestion of paper dust and sludge, and 45 to 55 percent for ingestion of soil. EPA/OSW, however, assumed 100 percent absorption for each exposure pathway applicable to sludge disposal and use except for dermal exposures. For dermal exposures, a dermal transfer coefficient of 0.012/hour is assumed for 2,3,7,8-TCDD and 2,3,7,8-TCDF that are not bound up within a matrix (e.g., soil or paper products).

A variety of terms were used in the source documents for the Integrated Assessment to describe the exposure case or exposed individual for which risks were estimated. Many of these terms are presented in the Background Document to the Integrated Assessment and in Section 2 of this report as they appear in the source document. These exposure case descriptions include: low, high, average, typical, reasonable worst case, extreme worst case, and maximum exposed individual (MEI). Such exposure case descriptions are used by exposure/risk assessors to describe where in the range of exposures possible for a given scenario, they either know (from a statistical array of exposures or exposure parameter values) or judge the calculated exposure to reside. Historically there has been no generally accepted convention or guidance specifying what defines a given exposure description. Therefore, the use of these terms has not been consistent within this report.

In order to provide some consistency in summarizing the results of the Integrated Assessment, the various exposure case descriptions used in the source documents have been collapsed in this section into the following two classifications: typical and reasonable worst case. The majority of exposure cases developed in the source documents fit into one of these two categories if the following somewhat broad definitions are used:

Typical exposure - exposure parameter values selected are: (a) values conventionally used for certain exposure parameters; or (b) average or most probable values when distribution data for the parameter are available; or (c) values considered "typical" or frequently observed based on best professional judgment.

Examples of the types of exposures mentioned in this report that would be characterized as typical include inhalation of volatilized 2,3,7,8-TCDD/TCDF; inhalation of particulate matter (i.e., paper dust or sludge) containing 2,3,7,8-TCDD/TCDF; dermal contact with pulp, paper, or sludge containing 2,3,7,8-TCDD/TCDF; and ingestion of food, water, and drugs containing 2,3,7,8-TCDD/TCDF.

Reasonable worst case exposure - similar to typical exposure with the exception that values for one or more significant exposure parameters are selected within the upper portion of the range of actual or expected values so that the resulting exposure calculated represents a relatively high but possible exposure.

Examples of the types of exposures mentioned in this report that would be characgerized as reasonable worst-case include those cases in which an individual is exposed to the highest possible concentration (i.e., 90th percentile and above); or instances where an individual is exposed at a frequency or duration higher than what is typically observed.

1.3 Industry Profile

This section provides a brief overview of the pulp and paper industry and its products, the pulping and bleaching processes, and potential sources of dioxins and furans in pulp and paper. This section is compiled, in large part, from:

Clement Associates, Inc. 1989. Dioxin production in the pulp/paper industry. Revised draft report. U.S. Environmental Protection Agency, Office of Toxic Substances. EPA Contract No. 68-D8-0116. September 29, 1989.

1.3.1 Overview of the Industry

There are 391 pulp and paper mill companies in the United States with 802 establishments. An establishment is defined as the location of manufacturing or non-manufacturing activities. An establishment can be a single pulp mill, a single paper mill, an integrated mill which has one or more pulp mills as well as one or more paper mills; or a central headquarters and/or a Research and Development facility when these are located separately from the manufacturing activity. There are 605 mills classified as paper mills (this is most likely the majority of pulp buyers; it is unknown whether textile and pharmaceutical companies that buy pulp are included in this figure) and 358 mills (some may be inactive) that produce pulp which is used in 605 paper mills.

The 358 mills fall into two categories--chemical and mechanical. These categories differ in the method used to produce pulp from the wood. Currently in the industry, 49.7 percent of the pulp mills use chemical pulping methods; 30.4 percent use mechanical methods; and the remaining 19.8 percent involve miscellaneous methods (these include deinking, rag, soda, rope, flax, bagasse, and cotton linters pulp mills). There are three types of chemical pulping methods within the industry--kraft (sulphate), sulphite, and semi-chemical. These methods differ in the chemicals, wood types, and techniques used, as well as in the end products produced. In the United States, 104 chemical pulping mills using chlorine in the bleaching process were identified by the EPA Office of Water. Chlorine has been identified as a necessary precursor to dioxin formation. Some of these mills are involved in more than one pulping process, but the dominant process is chemical pulping.

1.3.2 Processes/Operation

(1) Pulping process. The (wood) raw material that enters the pulping process is composed of cellulose fibers, lignin, semicellulose, and other compounds. Lignin is a complex polymer that is believed to contain dioxin precursors. It is responsible for cementing and strengthening the wood fibers, and for the discoloration of wood. In the

production of paper, some lignin is removed to allow flexibility of the fibers. The amount of lignin remaining determines the length of time the paper remains flexible. The remaining portion, about 10 percent in the kraft pulping process, is bleached to produce a desired whiteness and to deter the discoloration produced with age. The amount of lignin destroyed/bleached is specific to the grade of paper product, and ranges from paper sacks and newsprint to writing paper.

The pulping process utilizes mechanical and chemical techniques to produce the desired pulp type. The mechanical process is used primarily with softwoods that are easier to tear and grind, and the resultant pulp is generally used in the manufacture of newsprint, catalogues, and toweling.

Chemical pulping removes the lignin to enhance fibers flexibility, resulting in a stronger paper product but lower fiber yields (40 to 50 percent). The three chemical pulping categories in increasing order of use are sulphite, semi-chemical, and kraft (sulphate). Sulphite pulp, often blended with mechanical pulps as a strengthener, is used for the production of viscose rayon, acetate fibers and films, plastic fillers, and cellophanes. Semi-chemical pulping is generally used for newsprint, containers, and computer cards.

The kraft pulping dominates the U.S. pulp and paper industry, accounting for about 75 percent of the pulp produced for paper and paperboard (OTA 1989). The advantages of this process are (1) the versatility in the types of wood it can process and (2) the released extracts (e.g., turpentine, tall oil, and resin) that can be separated for sale as commodity chemicals. In addition, process chemicals are recovered and recycled. Within the chemical pulping process, kraft pulping is used at over 70 percent of U.S. mills.

(2) Bleaching process. After the wood goes through the pulping process, the degree of bleaching it receives is dependent upon the whiteness that is desired. The bleaching process for chemical pulp proceeds in two phases--delignification and brightening. They are carried out in alternating acid and alkaline stages. The conventional bleaching sequence involves five-stages, and chlorine is used as the dominant chemical. The length of bleaching time and the chemical used depend on the type/condition of the pulp and the desired characteristics of the final products. Generally, sodium hydroxide is used in the alkaline extraction stage. More recently, oxygen and peroxide have been added to improve the extraction efficiency.

There are a wide variety of sequences used to ensure a high level of delignification and whiteness. In 1989, chlorine was used as the initial bleaching chemical by the vast majority of U.S. chemical pulping mills (USEPA 1989). It is the most effective chemical for the delignification

and produces a high quality range of white paper products. Chlorine removes a number of contaminants from the pulp, and makes a strong board without attacking the cellulose.

1.3.3 Potential Source of Dioxin/Furans in Pulp and Paper

The chemical reactions and conditions under which dioxin is formed in pulp and papermaking operations are not yet completely understood. Dioxin is formed as a byproduct during these manufacturing operations and is considered a contaminant. Chlorination of a dioxin precursors in the pulp during bleaching stages may result in the production of 2,3,7,8-TCDD and 2,3,7,8-TCDF and other isomers.

Possible sources of dioxin precursors include natural constituents in wood, contaminants in plant pipes and machinery, and additives. Dioxin formation may result from condensation of chlorophenols formed by chlorination of naturally occurring phenolic compounds such as lignin, which comprises 25 percent of wood, and other plant constituents. To date, the only substances found to contain precursors that have been studied in depth are the oil-based defoamers.

A recent Canadian study has identified potential dioxin precursors in oil-based defoamers. The oil-based defoamers are additives that improve unbleached pulp washing properties. The study positively identified the presence of one dioxin precursor, dibenzofuran (DBF), in oil-based defoamers. DBF is a contaminant in the oil-based defoamers, and the study revealed the potential for DBF contamination in tap water used for process makeup water, air, and raw wood. The presence of another precursor, dibenzo-p-dioxin (DBP), was suspected in oil-based defoamers; however, it could not be confirmed because of excessive interference from other substances during analysis. Tests revealed that the addition of oil-based defoamers resulted in elevated levels of 2,3,7,8-TCDD and 2,3,7,8-TCDF in the final chlorinated pulp. Table 1-6 presents data on the effect of defoamer addition to laboratory-prepared brown stock Kraft pulp on subsequent dioxin formation during the bleaching process. The data were collected from studies using laboratory-prepared western hemlock Kraft pulp. Oil-based defoamer was added at one percent the weight of the pulp, which is 10 times greater than normal industry practice. This was done so that oil-based defoamers could be identified as being the source of the precursors.

1.3.4 End-Uses of Products from Pulp and Paper

The pulp and paper industry is comprised of those establishments that (1) produce pulp for market only; (2) produce pulp for in-house only; (3) buy pulp for use in paper product manufacture; and (4) produce pulp for in-house use in their paper mill(s) and for sale in the market. A pulp mill is usually a separate facility from the paper mill, even at combined pulp and paper manufacturing plants.

Table 1-6. Effect of Defoamer Addition to Brownstock Kraft Pulp on Subsequent Dioxin Formation During the Bleaching Process[a]

Pulp additive	2,3,7,8-TCDD concentration, ppt	2,3,7,8-TCDF concentration, ppt
None	11	160
1% of oil-based defoamer A (virgin oil base)	110	910
1% of oil-based defoamer B from Canadian kraft mill	81	280
1% of oil-based defoamer C (recycled oil base)	140	1,200
1% of recycled oil base used in defoamer C	170	1,400

[a]Voss (1988).

The majority of bleached pulp produced in the U.S. is converted to paper and paperboard products such as printing/writing paper, tissue paper, packaging paper and paperboard, and non-packaging paperboard. The category, tissue, usually includes commonly used household items such as disposable diapers, facial and toilet tissue, wadding, coffee filters, napkins, toweling, and hygiene pads. Food-related paper products include food wrapping and bags. Food-related packaging paperboard includes milk carton stock and ovenable board, and non-packaging paperboard includes cup and plate stock.

1.4 Organization of This Report

This report is organized into 12 sections and appendices. Following is a brief description of each section:

- Section 2 provides an analysis of the chemistry and fate of dioxins and furans.
- Section 3 describes the human, terrestrial and avian wildlife, and aquatic life hazards of 2,3,7,8-TCDD and 2,3,7,8-TCDF.
- Section 4 provides an assessment of exposures and risks to workers in the pulp and paper industry.
- Section 5 provides an assessment of exposures and risks to humans from use and disposal of pulp and paper mill sludge and land disposal of paper waste. Exposures were evaluated based on sludge disposal in landfills, surface impoundments, land application/landfarming, and from marketing the sludge.
- Section 6 provides an assessment of exposures and risks to humans associated with the discharge of effluents from bleached kraft and sulfite pulp and paper mills.
- Section 7 provides an assessment of exposures and risks to humans resulting from incineration of pulp and paper mill wastewater sludges.
- Section 8 provides an assessment of exposures and risks to consumers from use from pulp and paper products.
- Section 9 provides an assessment of exposures and risks to the general population from dermal contact with pulp-containing medical devices.
- Section 10 provides an assessment of exposures and risks to the general population from use of food packaged in or containing bleached paper products.

- Section 11 provides an assessment of exposures and risks to the general population from the use of food, drug, and cosmetic formulations containing cellulose derivatives.

- Section 12 presents FDA's assessment of human exposures and risks resulting from ingestion of fish from pulp and paper mill receiving waters.

- Section 13 provides an assessment of risks to terrestrial and avian wildlife resulting from land application of sludges and an assessment of risks to aquatic life resulting from discharge of pulp and paper mill effluents.

Appendix A provides PCDD and PCDF bioavailability information. Appendix B describes the environmental effects of other chlorinated organics.

Each exposure section evaluates an exposure pathway, identifies and enumerates exposed populations, estimates risk associated with that exposure and presents an analysis of uncertainty in the assessment.

1.5 References

Beck H, Eckart K, Mathar W, Wittkowski R. 1988. Occurrence of PCDD and PCDF in different kinds of paper. Chemosphere, 17: 51-57.

Blosser RO. 1987. Communication to U.S. Consumer Product Safety Commission (CPSC). September 25, 1987.

Helms J. 1989. U.S. EPA/Paper Industry cooperative dioxin study field congener analyses. Memorandum from J. Helms (USEPA/PWRS) to G. Schweer (USEPA/OTS). August 11, 1989.

LeBel GL, Williams DT, Benoit FM. 1989. Determination of chlorinated dibenzodioxins and dibenzofurans in selected paper products. Ninth international symposium on chlorinated dioxins and related compounds, Toronto Ontario, September 17-22, 1989. Abstract PLP23.

NCASI. 1987. National Council of the Paper Industry for Air and Stream Improvement. Assessment of potential health risks from dermal exposure to dioxin in paper products. Technical Bulletin No. 534. November 1987.

OTA. 1989. Office of Technology Assessment. Technologies for reducing dioxin in the manufacture of bleached wood pulp. OTA-BP-O-54. May 1989.

USEPA. 1987a. U.S. Environmental Protection Agency. National dioxin study. Washington, D.C.: U.S. Environmental Protection Agency. EPA 530/SW-87-025.

USEPA. 1987b. U.S. Environmental Protection Agency. Interim procedures for estimating risks associated with exposures for mixtures of chlorinated dibenzo-p-dioxins and -dibenzofurans (CDDs and CDFs). Risk Assessment Forum. EPA 625/3-87-012. National Technical Information Service, Springfield, VA. PB89-125041.

USEPA. 1988. U.S. Environmental Protection Agency. U.S. EPA/Paper Industry cooperative dioxin screening study. Washington, D.C.: Office of Water Regulations and Standards. EPA 440/1-88-025.

USEPA. 1989. U.S. Environmental Protection Agency. U.S. EPA/Paper Industry cooperative dioxin study. Data submittals during 1988 and 1989 to EPA, Office of Water Regulations and Standards.

Voss RH, et al. 1988. Some new insights into the origins of dioxins formed during chemical pulp bleaching. Pulp and Paper Canada, 89(12): 151-161.

2. CHEMISTRY AND FATE OF DIOXINS AND FURANS

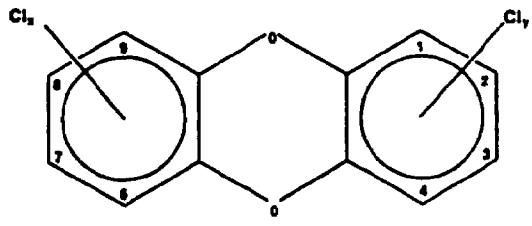
This section reviews the available information regarding the chemistry and fate of the general group of compounds termed polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs), as well as the specific compounds 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) and 2,3,7,8-tetrachlorodibenzofuran (2,3,7,8-TCDF). This section is compiled from:

Versar Inc. 1989. Chemistry and fate of dioxins and furans. Washington, DC: U.S. Environmental Protection Agency, Office of Toxic Substances. Contract No. 68-02-4254, Task No. 231.

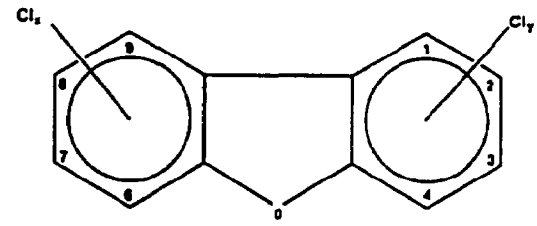
2.1 Chemical Identity of PCDDs and PCDFs

Very little published information is available on the physical and chemical properties of most PCDDs and PCDFs, other than recently reported values for some congeners for such important properties as water solubility and vapor pressure. In general, structure-activity principles hold with higher chlorinated congeners being less soluble and showing lower vapor pressures, thus being less volatile.

PCDDs and PCDFs are tricyclic aromatic compounds having three-ring structures consisting of two benzene rings connected by a third ring. For PCDDs, the connection of the benzene rings is through a pair of oxygen atoms as opposed to one oxygen atom for the PCDFs. In general, both compound classes have similar physical, chemical, biological, and toxicological properties (Rappe et al. 1987; EPRI 1983). The basic structure and numbering of each chemical class is shown below.



Polychlorinated dibenzo-p-dioxins (PCDDs)



Polychlorinated dibenzofurans (PCDFs)

There are 75 possible different positional isomers of PCDDs and 135 different isomers of PCDFs (see Table 2-1). The literature reviewed reports that neither chemical class reacts with weak acids and bases, nor with most redox agents, and that each is only slightly soluble in water and in many organic solvents. The chemical stability increases with increasing halogen content (EPRI 1983).

Table 2-1. Possible Number of Positional PCDD and PCDF Isomers

Chlorine substitution	Number of isomers	
	PCDDs	PCDFs
Mono	2	4
Di	10	16
Tri	14	28
Tetra	22	38
Penta	14	28
Hexa	10	16
Hepta	2	4
Octa	1	1

According to EPRI (1983) the chemistry of these compounds also suggests that PCDFs can form directly from PCBs but the PCDDs cannot. However, both compound classes can be formed from chlorophenols or chlorinated benzenes. In addition, Svenson et al. (1989) reported the formation of PCDDs and PCDFs in an enzyme-catalyzed oxidation of 2,4,5-trichlorophenol. Treatment of the chlorophenol with hydrogen peroxide/peroxidase resulted in formation of small amounts of 2,3,7,8-tetra-substituted congeners as well as other derivatives.

Little, if any, research or analyses have been performed to determine physical and chemical properties of TCDFs and TCDDs, with the exception of the 2,3,7,8-TCDD isomer. Few of the possible isomers are available commercially and preparation and synthesis can be both time consuming and difficult. In addition, these isomers prepared in pure form may not be available in sufficient quantities for testing, and the high toxicity of these compounds necessitates extreme precautions to prevent adverse effects in the workers in the toxicology laboratories (EPRI 1983).

Due to the lack of experimental data, the physical/chemical properties of the various PCDF and PCDD congeners were estimated (Table 2-2). Estimates were derived from the limited experimental data for 2,3,7,8-TCDD and via the methods described below. Water solubilities, soil adsorption coefficients (K_{OC}), and bioconcentration factors (BCF) were estimated from the equations in Lyman et al. (1982). Vapor pressures were estimated via the Meissner method as described in Lyman et al. (1982). The log K_{OW} values were estimated by estimating the value of a fragment constant representing the substitution of a chlorine atom for a hydrogen atom of a PCB. The log K_{OW} values for PCDDs were estimated by starting with the reported log K_{OW} value for 2,3,7,8-TCDD, and for PCDFs by starting with the reported value for dibenzofuran, 4.10 (Leo and Hansch 1979).

The measured bioconcentration factor (BCF) values for PCDDs and PCDFs are much lower than the predicted BCF values using SAR Analysis and the calculated octanol/water partition coefficient (K_{OW}). The differences between predicted versus measured values have been attributed to metabolism. PCDDs were bioconcentrated to greater extent than PCDFs, as predicted by SAR Analysis. There was also a relationship between BCF and exposure concentration for PCDDs and PCDFs, whether exposures were from water or diet. As exposure concentration increased, the BCF decreased.

Table 2-2. Summary of Estimated Physical/Chemical Properties of PCDFs and PCDDs

Congener	Average M_w (g/mol)	Physical State[a] (@25°C)	Boiling Point (°C)	Melting Point (°C)	Log K_{ow}	Sol. in Water (mg/l)	K_{oc}	Vapor Pressure (mm-Hg @ 25°C)	Henry's Constant (A-m^3/mol)	Bioconcentration Factor
PCDFs										
Tetrachloro	306	Solid	340	306	5.78	5.4×10^{-5}	3.3×10^{4}	1.1×10^{-6}	8.2×10^{-3}	1.5×10^{4}
Pentachloro	340	Solid	357	320	6.20	1.2×10^{-5}	5.6×10^{4}	2.4×10^{-7}	8.9×10^{-3}	3.0×10^{4}
Hexachloro	375	Solid	373	335	6.62	2.7×10^{-6}	9.5×10^{4}	5.6×10^{-8}	1.1×10^{-2}	6.3×10^{4}
Heptachloro	409	Solid	389	349	7.04	6.2×10^{-7}	1.6×10^{5}	1.3×10^{-8}	1.1×10^{-2}	1.3×10^{5}
Octachloro	444	Solid	405	363	7.46	1.4×10^{-7}	2.7×10^{5}	3.2×10^{-9}	1.3×10^{-2}	2.8×10^{5}
PCDDs										
Tetrachloro	322	Solid	341	306[b]	6.15[b]	2.0×10^{-4}	5.3×10^{4}	1.7×10^{-6}[b]	3.6×10^{-3}	2.8×10^{4}
Pentachloro	356	Solid	358	321	6.57	4.8×10^{-6}	8.9×10^{4}	2.1×10^{-7}	2.0×10^{-2}	5.8×10^{4}
Hexachloro	391	Solid	374	336	6.99	1.1×10^{-6}	1.5×10^{5}	5.5×10^{-8}	2.6×10^{-2}	1.2×10^{5}
Heptachloro	425	Solid	390	350	7.41	2.4×10^{-7}	2.6×10^{5}	1.1×10^{-8}	2.6×10^{-2}	2.5×10^{5}
Octachloro	460	Solid	406	364	7.83	5.4×10^{-8}	4.3×10^{5}	2.8×10^{-9}	2.7×10^{-2}	5.2×10^{5}

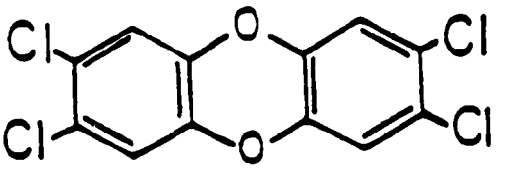
[a] Assumed to be a solid at room temperature (parent compound is a solid).

[b] Measured values for 2,3,7,8-TCDD. Log k_{ow}s for other PCDDs and PCDFs were estimated from measured values of log k_{ow} for 2,3,7,8-TCDD or 2,3,7,8-TCDF and a fragment constant for chlorine substitution on PCBs estimated from measured log k_{ow} values for 27 PCB isomers. Values for all other properties listed were estimated using methods from Lyman et al. (1982).

2.2 Chemistry and Fate of 2,3,7,8-TCDD

2.2.1 Chemical Identity

2,3,7,8-Tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) is a member of the 75 compounds known as polychlorinated dibenzo-p-dioxins (PCDDs). The structure of 2,3,7,8-TCDD is:



2.2.2 Chemical and Physical Properties

The measured properties of 2,3,7,8-TCDD obtained from the literature are listed in Table 2-3. Estimated values might be reported if no measured values are available for an important physical or chemical property. In general, 2,3,7,8-TCDD is very sparingly soluble in water, has a high octanol-water partition coefficient and, therefore, sorbs to organic matter, and has a low vapor pressure but will volatilize into the air under favorable conditions.

2.2.3 Environmental Fate and Transport

2,3,7,8-TCDD is persistent in soils. Upon deposition of 2,3,7,8-TCDD onto surfaces, there is a high initial loss due to photodegradation and perhaps volatilization. Once 2,3,7,8-TCDD moves into soils or sediments, however, it is apparently strongly sorbed unless there are chlorinated organic co-contaminants present. Some recent studies, however, have shown that there may be slow rates of vapor phase transport out of soils, although other recent studies have shown very low mobility.

The only environmentally significant path for destruction of 2,3,7,8-TCDD appears to be photodechlorination. This process, however, requires the presence of another organic material to donate hydrogen atoms. Observations on bioaccumulation indicate that 2,3,7,8-TCDD is readily bioconcentrated in fish, but the data for humans are inconclusive, and the dioxins in soil and sediments are considered to be essentially nonbiodegradable. Erosion and aquatic transport of sediment appear to be the main transport mechanism of sorbed dioxins. Table 2-4 is a summary of the environmental fate of chlorinated dibenzo-p-dioxins.

Table 2-3. Physical and Chemical Properties of 2,3,7,8-TCDD

Property	Value	Comments	References	Analytical Method
Chemical name	2,3,7,8-Tetrachlorodibenzo-p-dioxin			
Synonym	TCDD; 2,3,7,8-TCDD			
Empirical formula	$C_{12}H_4Cl_4O_2$			
CAS number	1746-01-6			
Molecular weight	321.96			
Pure Form	White crystalline solid	25°C		
Melting Point	302-305°C		USEPA (1987b)	
Decomposition temperature	>700°C		USEPA (1987b)	
Critical temperature	661°C		Reid et al. (1977)	
Critical pressure	2,372.829 kPa		Reid et al. (1977)	
Critical volume	763 cm^3/g mole		Reid et al. (1977)	
Water solubility	19.3 ± 3.7* ppt (ng/L)	22°C	Marple et al. (1986a)	Thin film equilibrium, GC/LRMS with ^{63}Ni electron capture detector.
	7.91 ± 2.7 ppt (ng/L)	25°C	Adams and Blaine (1986)	^3H-labelled TCDD, scintillation detector.
	200 ppt	25°C	Kearney, et al. (1973)	
	12.9 ± 1.2 ppt	4.3 ± 0.2°C	Lodge (1989)	Generator column followed by HPLC
	483 ± 94 ppt	17.3 ± 0.1°C	Lodge (1989)	Generator column followed by HPLC

Table 2-3. (continued)

Property	Value	Comments	References	Analytical Method
Solubility in Organic Solvents at 25°C	Solubility (mg/L)		Eposito et al. (1980)	
o-dichlorobenzene	1400			
chlorobenzene	720			
anisole	1730			
xylene	3580			
benzene	570			
chloroform	370			
n-octanol	48			
methanol	10			
acetone	110			
dioxane	380			
Log Octanol-water partition coefficient, (Log K_{ow} or Log P)	6.64* (Average) 6.54 - 6.95 (Range)		Marple et al. (1986b)	Diffusion, GC/LRMS
	7.02 ± 0.5		Burkhard and Kuehl (1986)	Reverse-phase HPLC and LRMS
	6.15 to 7.28 (Range)		USEPA (1984)	
	Apx. 8.5		Sarna et al. (1984)	
Vapor pressure (mmHg) (at 25°C, unless specified)	$(7.4 \pm 0.04) \times 10^{-10}$*		Podoll et al. (1986)	^{14}C-labelled 2,3,4,8 TCDD and gas saturation technique with combustion to $^{14}CO_2$.
30.1°C	$(3.49 \pm 0.05) \times 10^{-9}$		Schroy et al. (1985)	Gas saturation with GC/MS
	1.5×10^{-9}	Values extrapolated	Schroy et al. (1985)	
30°C	3.4×10^{-9}			
50°C	7.2×10^{-8}			

* Recommended values at stated temperature.

Table 2-3. (continued)

Property	Value	Comments	References	Analytical Method
Vapor pressure (continued)				
305°C	49.4			
421°C	760			
	1.5×10^{-9}	Measured	Rordorf (1987)	
	4.65×10^{-9}	Predicted	Rordorf (1987)	
Henry's Law Constant	1.6×10^{-5} atm-m^3/mol*	Calculated from the water solubility and vapor pressure listed above.	Podoll et al. (1986)	
	2.12×10^{-6}		Schroy et al. (1985)	
Volatilization from water	32 days	Half life for ponds and lakes	Podoll et al. (1986)	
	16 days	Half life for rivers	Podoll et al. (1986)	
Soil/sediment adsorption coefficient	7.15-7.34	Experimental	Jackson et al. (1986)	
	6.95	Predicted		
	7.39	Calculated		
Log K_{oc}	6.6 ± 0.7		Walters and Guiseppi-Elie (1988)	
Bioconcentration factor in fish:			Nabholz (1989)	
Exposure from water	20,600	Geometric mean for all fresh water fish, N=7. Most Reliable in concentration range of 0.038 to 107 ppt.		

Table 2-3. (continued)

Property	Value	Comments	References	Analytical Method
Bioconcentration factor in fish (continued)				
Exposure from water (continued)	24,100	Geometric means for cold water species, N=6, Most Reliable in concentration range of 0.038 to 107 ppt.		
	7,900	Warm water species, N=1, concentration was 0.87 ppt.		
Exposure from dietary sources	0.09697	Geometric mean for all fresh water fish, N=7, Most Reliable in concentration range of 39 to 2000 ppt.		
	0.1119	Geometric mean for cold water species, N=4, at a concentration of 494 ppt.		
	0.08016	Geometric mean for warm water species, N=3, Most Reliable in concentration range 39 to 2000 ppt.		
Bioconcentration factors in humans:			Geyer et al. (1987)	
Weight weight basis	104 to 206 (155 average)	Wet weight basis. Calculated assuming estimated daily intakes of 0.0325-0.065 ng of TCDD/day and measured concentrations of TCDD in adipose tissue of humans.		

* Recommended values at stated temperature.

Table 2-3. (continued)

Property	Value	Comments	References	Analytical Method
Bioconcentration factors in humans (continued)				
Lipid basis	115-229 (172 average)	Calculated on a lipid basis.	Geyer et al. (1987)	
Wet weight basis	153	From measured half-life and measured concentrations in body fat at steady state using a linear, one-compartment pharmacokinetic model.		
Lipid basis	170	From measured half-life and measured concentrations in body fat at steady state using a linear, one-compartment pharmacokinetic model.		

Bioconcentration factors in animals: Geyer et al. (1987)

	BCF_{Liver}	BCF_{Fat}	Duration of Feeding	Concentration in diet (ng/kg)
Rats	24.5	24.5	2 years	22
	24.3	8.1	2 years	210
	10.9	3.7	2 years	2200
Beef cattle	0.7	3.5	28 days	24
	-	24.8	499 days	24
Rhesus monkey	-	24 - 40	4 years	25

Table 2-3. (continued)

Property	Value	Comments	References	Analytical Method
Bioconcentration factor:				
Vegetation	0.013	Calculated	Travis and Arms (1988)	
Milk	0.010	Calculated	Travis and Arms (1988)	
Beef	0.05	Calculated	Travis and Arms (1988)	

* Recommended values at stated temperature.

Table 2-4. Summary of Environmental Fate of Dibenzo-p-dioxins

Environmental process	Summary statement	Confidence in data
Photolysis	May be only natural mechanism leading to destruction of dioxins.	High
Oxidation	Dioxins are stable to oxidation.	Low
Hydrolysis	Dioxins are stable to hydrolysis.	High
Volatilization	Possible important mechanism for transport from water. Volatility depressed by presence of organic solids.	Medium
Sorption	Dioxins strongly sorbed by solids, especially with high organic content.	High
Bioaccumulation	Available data indicates process may be important. The data show high degree of confidence for bioconcentration in fish, but low confidence in the limited data concerning bioaccumulation in humans.	Medium
Biodegradation	Considered essentially non-biodegradable.	Medium

(1) Photolysis. Dioxins (i.e., dibenzodioxin and its chlorinated derivatives) absorb electromagnetic radiation above 290 nm, and can be expected, therefore, to be subject to photolysis by sunlight (EPRI 1983). Solar radiation of wavelengths lower than 290 nm is filtered out in the troposphere, so absorption by chemicals of radiation at lower wavelengths is moot in considering environmental photodegradation processes. All chlorinated dioxins can be dechlorinated photolytically in the presence of a suitable hydrogen donor (Crosby et al. 1973, Crosby 1978). Dechlorination is fast in organic media, slow in water, but practically nonexistent on dry inorganic surfaces or in the crystalline state (Crosby 1978).

(a) Solid surfaces. Substantial research on the environmental persistence of 2,3,7,8-TCDD has been done as part of the decontamination of the area around the ICMESA chemical plant in Seveso, Italy, which was contaminated when a trichlorophenol reaction vessel overheated in 1976, blowing out the safety devices and spraying dioxin contaminated material into the environment. The levels of dioxin in the soil decreased substantially during the first six months following the accident, reaching a steady state of 1/5 to 1/11 of the initial levels (DiDomenico et al. 1982). An experiment was conducted at this site to determine the effectiveness of photolysis in decontaminating surface deposits of dioxin on foliage. Test plots were sprayed with olive oil to act as an H-donor, and the levels of dioxin on grass were found to be reduced by over 80% within nine days (Crosby 1981). 2,3,7,8-TCDD in contaminated soil was also found to be photolabile in sunlight when the soil was suspended in an aqueous solution of a surfactant. The destruction of 8 ug/ml of 2,3,7,8-TCDD in 0.02 M hexadecylpyridinium chloride could be accomplished in 4 hours (Botre et al. 1978). Figure 2-1 illustrates the photodegradation of 2,3,7,8-TCDD in the presence of hydrogen donors (Bertoni et al. 1978).

(b) Vapor phase. Photolysis in the gas phase also appears to be a degradative mechanism for 2,3,7,8-TCDD (EPRI 1983). In studies conducted at Stanford Research Institute, the half life of gaseous 2,3,7,8-TCDD in sunlight was found to be 5 to 24 days, and may be the result of indirect photolysis through the attack of hydroxyl radicals on the dioxin. This mode of destruction would be similar to the attack of hydroxyl radicals on PCBs (Leifer et al. 1983). In general, however, it appears that a hydrogen donor is necessary for photodegradation of dioxins (Crosby 1978), although Gebefugi et al. (1977) concluded that in sunlight photodecomposition of TCDD present on soils is likely to occur even in the absence of hydrogen donors (organic solvents).

Orth et al. (1989) conducted photolysis experiments with vapor-phase 2,3,7,8-TCDD under illumination in the UV region from 250 nm to 340 nm. The rate constants in helium and air were very similar, 5.4×10^{-3} sec^{-1} and 5.9×10^{-3} sec^{-1}, respectively, which corresponds to a quantum yield in air of 0.033 ± 0.046. No products could be observed in the mass spectrometer, so Orth et al. (1989) postulated that the product might be sorbing to the surface of the photolysis cell and lost from potential analysis.

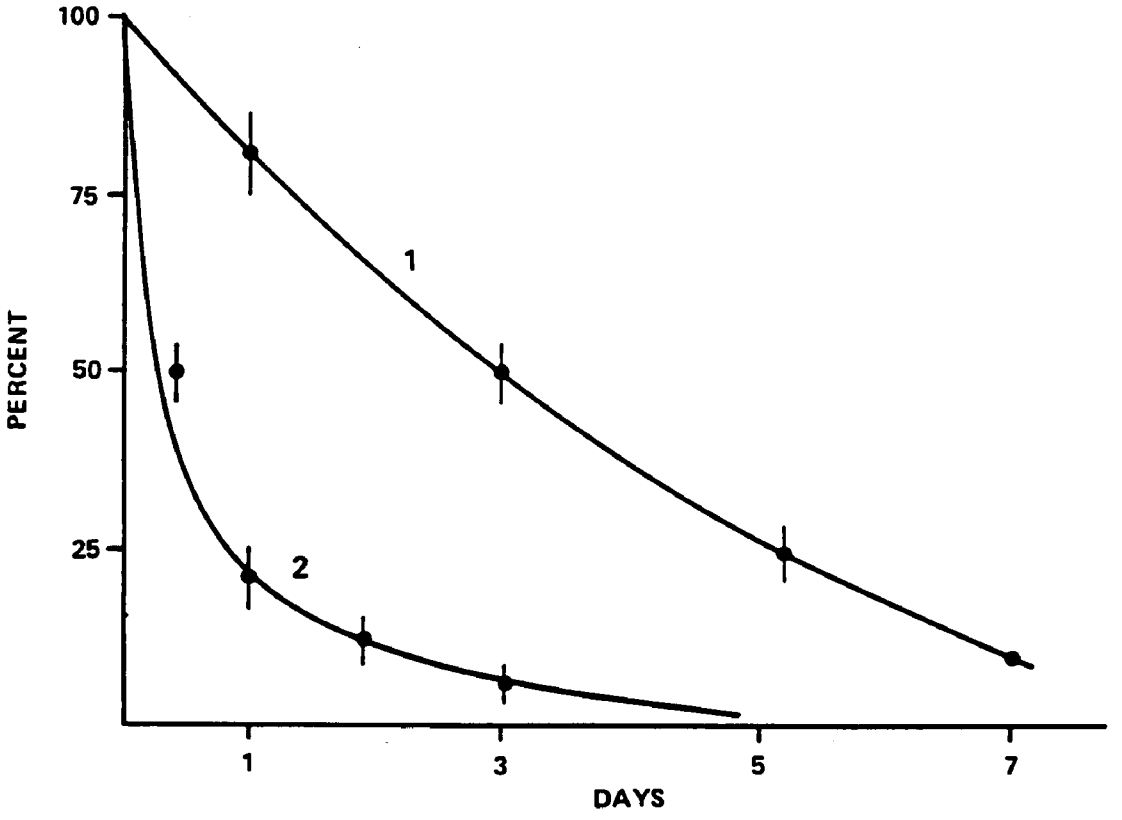


Figure 2-1. Destruction curves of 2,3,7,8-TCDD. (1) Seveso soil spread with mixture of ethyl oleate and xylene exposed to natural solar radiation. (2) TCDD sample placed in a room spread with the ethyl oleate-xylene mixture and irradiated with a Philips MLU 300-W lamp. Irradiation power on the sample: 20μW/cm^2.

Podoll et al. (1986) estimated the photolysis rate of 2,3,7,8-TCDD vapors in the atmosphere based on the quantum yield data for photolysis in hexane. The half-life in summer sunlight at 40° latitude was calculated to be 58 minutes, but Podoll et al. (1986) stated this estimate is an upper limit. The rate constant was also estimated for oxidation of 2,3,7,8-TCDD vapors by OH radical, the dominant transformation pathway in the atmosphere. At an average concentration of OH radicals in the atmosphere of 3×10^{-15}M, the half-life of 2,3,7,8-TCDD vapor via OH radical oxidation was estimated to be 200 hours.

Atkinson (1987) also estimated reaction rate constants and atmospheric half-lives for 2,3,7,8-TCDD and similar haloaromatics. The atmospheric lifetime of 2,3,7,8-TCDD due to the OH radical reaction was calculated to be 7 to 8 hours; based on the room temperature OH radical reaction rate constant and OH radical concentration for the average 12-h daytime, the atmospheric lifetime for 2,3,7,8-TCDD due to this gas-phase reaction was calculated to be an estimated 3 days.

Mill et al. (1987) reported preliminary photolysis experiments with atmospheric 2,3,7,8-TCDD. The half-life for vapor phase 2,3,7,8-TCDD in simulated sun was reported to be several hours. 2,3,7,8-TCDD was also sorbed to fly ash particulates, and the particulate 2,3,7,8-TCDD was suspended in recirculating air. The 2,3,7,8-TCDD sorbed to particulate fly ash showed half-life values of several hundred hours; in fact, for 2,3,7,8-TCDD in particulate form, there was virtually no photolytic reaction after 30 hours of illumination.

(c) Liquid phase. Dulin et al. (1986) studied the photolysis of 2,3,7,8-TCDD in aqueous solutions and reported a quantum yield of 0.05, similar to that reported by Orth et al. (1989) for vapor-phase-2,3,7,8-TCDD. In addition, Dulin et al. (1986) reported that the calculated sunlight photolysis rate constant for 2,3,7,8-TCDD in surface water in summer with sunlight levels found at 40° latitude would yield an estimated half-life value of 4.6 days. The quantum yield for photodegradation of 2,3,7,8-TCDD in water was three times greater under artificial light at 313 nm than under sunlight, and the artificial light photolysis quantum yield in hexane was 20 times greater than in water-acetonitrile as the solvent. Based on their analyses of photodegradation products, Dulin et al. (1986) concluded that cleavage of the C-O bond rather than the C-Cl bond may be the major pathway for 2,3,7,8-TCDD photolytic loss.

Podoll et al. (1986) used the Dulin et al. (1986) quantum yield data for 313 nm, 2.2×10^{-3}, and calculated half-life values for dissolved 2,3,7,8-TCDD under sunlight at 40° latitude in clear near-surface water under clear skies (24-h days). The seasonal values for half-lives were calculated to be 21 hours in summer, 51 hours in fall, 118 hours in winter, and 27 hours in spring. Sorption of 2,3,7,8-TCDD to sediments, even suspended solids, would have the effect of slowing the photolysis rates.

Buser (1988) studied the photolytic decomposition rates of 2,3,7,8-TCDD in a mixture of other chloro- and bromo-dibenzodioxins and dibenzofurans. Studies were performed in dilute hydrocarbon solutions as well as solid phases on quartz surfaces, under both sunlight and artificial laboratory illumination (fluorescent lights). Photochemical decomposition proceeds much faster in hydrocarbon solutions under sunlight compared with decomposition of 2,3,7,8-TCDD dispersed as thin solid films. 2,3,7,8-TCDD in i-octane solutions in quartz vials showed first-order kinetics with an estimated half-life ranging from 14 min to 200 min in two solutions. In tests under ordinary fluorescent laboratory lights, the same solutions were exposed in glass vials (rather than quartz), and half-lives of greater than 28 days were reported. 2,3,7,8-TCDD dispersed as a thin film in quartz vials and exposed to sunlight showed an estimated half-life of 120 hours. Buser (1988) stated the reported half-life values have limited accuracy because of the few data and because of the formation of 2,3,7,8-TCDD during photodecomposition of the higher halogenated forms, so the estimated accuracy was reported to be ±50%. From the data, Buser (1988) concluded that in hydrocarbon solutions, the major photochemical reaction was reductive dehalogenation, leading to lower chlorinated dibenzodioxins which then likely are further degraded to unsubstituted dibenzo-p-dioxin.

(2) Oxidation. Stehl (1973) has suggested that 2,3,7,8-TCDD is probably stable to oxidation in the ambient environment. In general, 2,3,7,8-TCDD is highly persistent in soils below the level that can be reached by sunlight (Miyata and Kashimoto 1979). Although the previously noted gas-phase photolysis of 2,3,7,8-TCDD could mean that dioxins are oxidized by hydroxyl radicals, there is no direct evidence for this environmental process in the absence of sunlight.

(3) Hydrolysis. There is no available evidence indicating that hydrolysis would be an operative environmental process for degradation of dioxins (EPRI 1983).

(4) Volatilization. The vapor pressure values of 2,3,7,8-TCDD at 25°C (in mm Hg) have been reported to range from $(7.4 \pm 0.4) \times 10^{-10}$ by Podoll et al. (1986) to 1.5×10^{-9} by Schroy et al. (1985) to 4.65×10^{-9} by Rordorf (1987). The reported log K_{OC} values are 6.6 ± 0.7 (Walters and Guiseppi-Elie 1988) and 7.15 to 7.34 (Jackson et al. 1986). Based on these values, 2,3,7,8-TCDD should not volatilize rapidly from organic soils and sediments; however, due to its stability and persistence via other transformation and transport pathways, volatilization should not be ignored as a mechanism for loss from soils.

The water solubility of 2,3,7,8-TCDD has recently been reported to be 7.91 ± 2.7 ng/l (ppt) at 25°C by Adams and Blaine (1986), 19.3 ± 3.7 ng/l at 22°C by Marple et al. (1986a), and 483 ± 94 ng/l at 17.3°C by Lodge (1989). This latter value by Lodge is even higher than values from the 1970's, such as 200 ng/l reported by Kearney et al. (1973) with less refined analytical methods. Lodge (1989) also reported a water solubility

value at 4.3°C of 12.9 ± 1.2 ng/l which is more in line with the solubility values at 25°C and 22°C reported by Adams and Blaine (1986) and Marple et al. (1986a), respectively.

Podoll et al. (1986) utilized their reported vapor pressure value and the Marple et al. (1986a) water solubility to compute a Henry's law constant of 1.6×10^{-5} atm•m^3/mol. Estimates of the Henry's law constant lack precision for vapor pressures and water solubility measurements, because of the ranges of reported values, but the values reported do suggest that 2,3,7,8-TCDD may volatilize from water and moist surfaces.

Observations from the Seveso incident indicate that when 2,3,7,8-TCDD is deposited on the soil surface, both volatilization and photodegradation are initially rapid (DiDomenico et al. 1982). Once it has been washed into the soil, however, 2,3,7,8-TCDD is unaffected by either volatilization or photolysis (Plimmer 1978, Kriebel 1981). Podoll et al. (1986) reported that when 2,3,7,8-TCDD has been mixed to a depth of only a few millimeters in dry soil, the volatility will be extremely slow because of the very low unsteady-state diffusion of the vapor in the dry soil air phase. . However, Freeman and Schroy (1985) point out that low volatility chemicals may bind strongly with very dry soil but, once a molecular microlayer of water covers the soil particles, the chemical should become more volatile; in addition, water vaporization may enhance the rate of chemical vaporization from a soil column.

Freeman and Schroy (1985) reported that a simple half-life model is totally inadequate to describe volatilization and environmental persistence, and used two coupled partial differential equations both coupled to a second-order heat transfer equation to describe 2,3,7,8-TCDD transport in soils. At the surface, the apparent half-life for 2,3,7,8-TCDD is measured in weeks, but mixed with soils below a depth of 5cm, the apparent half-life is measured in years and the "half life concept" is invalid. At Times Beach, MO, 2,3,7,8-TCDD volatilized from soils most rapidly in summer and did not volatilize to any appreciable extent during the winter. At the end of the first summer, over 90% of the applied 2,3,7,8-TCDD contained in the top one centimeter of soil was lost, and by the end of the first year, over 95% of the applied 2,3,7,8-TCDD had been lost from the top 1 cm. Further, of the total applied 2,3,7,8-TCDD, over 50% volatilized during the first summer after the initial application at Times Beach in 1972, based on the estimates of losses provided by Freeman and Schroy (1985).

Subsequently, Freeman and Schroy (1989) utilized another mathematical model to evaluate the transport of 2,3,7,8-TCDD in soil via vapor transport and other mechanisms. Results of the simulation predicted that only 0.01 percent of the 2,3,7,8-TCDD present in the top 1 cm of soil should be lost due to vaporization over the 16 months of the experiment, and that 0.1 percent of the 2,3,7,8-TCDD in the top 1 mm should have volatilized. An observed reduction of approximately 50 percent in the 2,3,7,8-TCDD

concentration in the top 3 mm of soil was not explainable by the model results, so another mechanism such as surface photodegradation was postulated. In addition to clarifying that the rate of transport in soil is very slow via vaporization, Freeman and Schroy (1989) reported that analyses indicated that over 99 percent of the 2,3,7,8-TCDD applied to the roads at Times Beach, Missouri is still in the soil.

Eduljee (1987) estimated the volatilization fluxes for 2,3,7,8-TCDD from the Jury et al. mathematical model for transport of chemicals through soil and loss at the soil-air interface. The diffusion of 2,3,7,8-TCDD in soil was estimated to be vapor-dominated up to a volumetric water content of 0.3 m^3/m^3, and then liquid-dominated to saturation; i.e., initially decreasing in transport as pore spaces are filled, then increasing in transport as liquid diffusion predominates. In addition, the model predicted that 2,3,7,8-TCDD is subject to wicking, and Eduljee (1987) pointed out this was contrary to the views of Podoll et al. (1986). Increasing the organic content of the soil retards the movement of 2,3,7,8-TCDD and depresses the volatilization flux.

Palausky et al. (1986) injected 2,3,7,8-TCDD in various organic solvents into soil columns to determine the extent of vapor phase diffusion of 2,3,7,8-TCDD in soil and the effects of carrier medium on the degree of migration. The solvents used included acetone/water, xylene, chloroform, toluene, methanol, iso-octane, and decane. No noticeable changes in 2,3,7,8-TCDD concentration profiles were observed after 30 days incubation at temperatures ranging from 0° to 20°; however, a measurable change was observed at 40°C, indicating volatilization may have been causing transport. Little or no migration was observed with the acetone/water mixture. With organic solvents, the slowest migration was with saturated hydrocarbons, somewhat greater with methanol, and migration was highest with aromatic solvents and chloroform. The extent of migration was related to solubility of 2,3,7,8-TCDD in the solvent, but a direct correlation between solubility and extent of migration was not found. Thus, the data at 40°C reflect not only the possible 2,3,7,8-TCDD vapor transport but also the volatility, viscosity, and interaction with soil organic matter for each of the solvents.

Concerning the volatility of 2,3,7,8-TCDD from water, Podoll et al. (1986) utilized data for the molecular diffusivity in air and in water to calculate volatilization half-lives. The volatilization half-life was about 32 days for ponds and lakes and about 15 days for rivers.

(5) Sorption. If 2,3,7,8-TCDD is washed into a water body, it will become strongly sorbed to bottom sediments (NRC Canada 1981). Its lipophilicity (log $K_{ow} \approx 6.6$ to 7) implies that 2,3,7,8-TCDD can be part of the oily surface scum of a water body as well as part of the sediment. 2,3,7,8-TCDD is not easily washed out of soils, and it is considered to be virtually immobile to aqueous phase transport in all but very sandy soils (Young 1981).

Palausky et al. (1986) conducted studies with 2,3,7,8-TCDD in various organic solvents and found that transport of 2,3,7,8-TCDD does occur. By inference, these data suggest that 2,3,7,8-TCDD may be highly sorptive to soils, especially with high levels of organic matter, but that organic solvents may play a role in the sorption/transport of 2,3,7,8-TCDD by acting as dispersing media within soils. Puri et al. (1989) reported research which indicated that 2,3,7,8-TCDD is highly sorptive, but that organic co-contaminants such as waste oil and surfactants may act to enhance the translocation of 2,3,7,8-TCDD. Also, the distribution coefficient of 2,3,7,8-TCDD in soils is dependent on the total organic matter, with higher sorption at higher soil organic levels, but the degree of sorption is also dependent on the types of organic matter present and their stability and solubility in the presence of the various co-contaminants.

Kapila et al. (1989) also tested the soil migration of 2,3,7,8-TCDD in waste crankcase oil. Field experiments showed that 2,3,7,8-TCDD moved downward in the soil column, and this movement was attributed to application of water to simulate rainfall. The water application resulted in displacement of the waste oil components and 2,3,7,8-TCDD from the macropore spaces in the soil column. The formation of colloidal suspensions and the presence of 2,3,7,8-TCDD in these suspensions may play a significant role in the downward movement of 2,3,7,8-TCDD. Since the amount of 2,3,7,8-TCDD recovered from each column after 12 months was "approximately the same as the amount initially applied," it was inferred by Kapila et al. (1989) that there was little loss of 2,3,7,8-TCDD from the soil surface via volatilization or photolysis during the one-year period.

Walters and Guiseppi-Elie (1988) evaluated the sorption of labeled 2,3,7,8-TCDD to soils from methanol/water mixtures in batch shake testing. The log K_{OC}, (the logarithm of the aqueous phase partition coefficient normalized for the organic content of soils) was reported to be 6.6 ± 0.7, as extrapolated from a linear regression analysis of various data. Data were presented which indicated that methanol/water mixtures of 2,3,7,8-TCDD did not result in solvent-soil interactions to increase 2,3,7,8-TCDD accessibility to soil organic matter to the same degree as that for various other organic compounds which are less hydrophobic. At the methanol concentrations tested, there was no significant increase in 2,3,7,8-TCDD mobility over that expected in the absence of solvents in the liquid phase. This relationship might apply to other water-miscible solvents, but Walters and Guiseppi-Elie (1988) stated it was unclear whether immiscible solvents would affect 2,3,7,8-TCDD mobility.

(6) Bioaccumulation/bioconcentration. Bioconcentration factors (BCF) for exposures from water and sediment are presented for 2,3,7,8-TCDD in Table 2-3. Nabholz (1989) reported that the data base for 2,3,7,8-TCDD was much better than the information for 2,3,7,8-TCDF (Table 2-5) (i.e., there were 14 measured factors for 2,3,7,8-TCDD and only 3 for the TCDF). In addition, the data for freshwater fish from

Table 2-5. Physical and Chemical Properties of 2,3,7,8-TCDF

Property	Value	Comments	References	Analytical Method
Chemical name	2,3,7,8-Tetrachlorodibenzo-furan			
Synonym	TCDF; 2,3,7,8-TCDF			
Empirical formula	$C_{12}H_4Cl_4O$			
CAS number	51207-31-9			
Molecular weight	305.98			
Pure form	White crystalline solid	25°C		
Melting Point	227-228		USEPA (1978)	
Log K_{ow}	5.82		Burkhard and Kuehl (1986)	Reverse-phase HPLC and LRMS detection.
Vapor pressure at 25°C	9.21×10^{-7} torr (mm Hg)		Eitzer and Hites (1988)	Gas saturation and effusion method.
Vapor density (above solid)	3.0×10^{-11} g/cm^3		USEPA (1978)	
Evaporation rate (from absorbed state)	2.1×10^{-12} g/cm^2/s		USEPA (1978)	
Water solubility	4.33 ug/l		Chemest, Versar (1989)	
Henry's law constant	8.6×10^{-5} atm.m^3/mol		Chemest, Versar (1989)	
K_{oc}	3.49×10^4		Chemest, Versar (1989)	

Table 2-5. (continued)

Property	Value	Comments	References	Analytical Method
Volatilization from Water				
	14 hrs.	Half-life for rivers	Versar (1989)	
	12 hrs.	Half-life for lakes & ponds	Versar (1989)	
Bioconcentration factors in fish:			Nabholz (1989)	
Exposure from water only	3,900	Geometric means for fresh water fish, equilibrium time is 7 days, n=2. Most Reliable in concentration range of 0.410 to 3.93 ppt. There is no data on warm water species exposure to 2,3,7,8-TCDF; therefore, the geometric mean for fresh water fish is calculated from cold-water species data.		
Exposure from dietary sources	0.1538	Based on one study on warm water species (N=1), dietary concentration of 182.0 ppt.		

* Recommended values at stated temperature.

water exposures of 2,3,7,8-TCDD were better than the data from exposure from dietary sources (i.e., food, sediment, or fly ash). While there were an equal number of studies (seven for water exposure and seven for dietary exposure), the 2,3,7,8-TCDD concentrations used in the water exposure studies had a much broader range than the range of exposure concentrations used in the studies with contaminated dietary sources.

There was a relationship between BCF and exposure concentration, whether the exposures were from water or dietary sources. As exposure concentration increased, the BCF decreased. This relationship was observed both for cold water species of freshwater fish exposed to 2,3,7,8-TCDD in water and for warm water species of freshwater fish exposed to 2,3,7,8-TCDD in diet.

For freshwater fish exposed to 2,3,7,8-TCDD via water only, the geometric mean BCF is 20,600. This mean BCF was based on 7 measurements. This BCF is most reliable for water concentrations from 0.038 ppt to 107 ppt; outside this range (especially the upper bound), this BCF is less reliable.

Nabholz (1989) reported that for cold water species of freshwater fish, there was a significant correlation between water concentration (in ppt) and fish BCF (on a whole fish, wet weight basis): the lower the concentration of 2,3,7,8-TCDD in the water, the higher the BCF in fish. The regression equation describing the relationship between exposure concentration in water and fish BCF is:

$$\log Y = 4.44397 - 0.27923 (\log X)$$

where Y = fish BCF on a wet weight, whole body basis; X = water concentration in ppt; N = 6; $r^2 = 0.76$; and $r = 0.87$ (which is statistically significant at $p < 0.05$). This regression was calculated based on six measurements. This equation is most reliable for water concentrations from 0.038 to 107 ppt; outside this range (especially the upper bound), this equation is less reliable. The geometric mean fish BCF from this data set is 24,100.

Nabholz (1989) reported that for warm water species of freshwater fish there was only one data point. The water concentration was 0.870 ppt and the fish BCF was 7,900.

There were no data available for bioconcentration factors for primarily water exposures of 2,3,7,8-TCDD in freshwater benthic fish species, marine pelagic fish, or marine benthic species.

When the exposure of 2,3,7,8-TCDD to fish is mostly through dietary sources (i.e., food, sediment or fly ash) rather than water, then a BCF based on dietary exposure must be computed. Bioconcentration factors (BCF) based on dietary exposures are probably more realistic than those

based on water concentrations because of the strong potential for 2,3,7,8-2,3,7,8-TCDD to partition to suspended solids and sediments in the aquatic environment.

Nabholz (1989) reported that for freshwater fish, the geometric mean BCF for 2,3,7,8-TCDD from contaminated dietary sources was 0.09697 and the mean equilibrium time was 84 days. This BCF was based on 7 measurements, and is most reliable for dietary concentrations of 2,3,7,8-TCDD from 39.0 ng/kg (ppt) to 2,000.0 ng/kg (ppt); outside this range (especially the upper bound), this BCF is less reliable.

For cold water fish species exposed primarily via dietary sources, the fish BCF was 0.1119 (geometric mean, N = 4), and the time to equilibrium was 91 days. This BCF was based on 4 fishes, all collected from the same contaminated area, which had a sediment 2,3,7,8-TCDD concentration of 494 ng/kg (ppt).

Nabholz (1989) reported that for warm water species, there was a significant correlation between dietary concentration (ng/kg; ppt) and fish BCF (on a whole fish, wet weight basis): the lower the water concentration, the higher the BCF. The regression equation describing the relationship between exposure concentration in diet and fish BCF is:

$$\log Y = 1.544 - 1.11199 (\log X)$$

where Y = fish BCF on a wet weight, whole body basis; X = concentration of 2,3,7,8-TCDD in dietary sources in ng/kg (ppt); N = 3; $r^2 = 0.66$; and $r = 0.81$. This regression was calculated from 3 measurements. This equation is most reliable for dietary concentrations from 39.0 ng/kg (ppt) to 2,000.0 ng/kg (ppt); outside this range (especially the upper bound), this equation is less reliable. The geometric mean fish BCF from this data set is 0.08016.

There are apparently no data available for fish BCF values primarily via dietary sources for either freshwater benthic species, marine pelagic species, or marine benthic species.

(7) Biotransformation and biodegradation. Investigations on dioxins have focused on the microbial degradation of 2,3,7,8-TCDD. Matsumura and Benezet (1973) tested approximately 100 strains of microorganisms which had previously shown the ability to degrade persistent pesticides, and only five strains showed any ability to degrade 2,3,7,8-TCDD, based on autoradiographs of thin-layer chromatograms. Although it is possible that the less chlorinated dioxins are more susceptible to biodegradation, microbial action on 2,3,7,8-TCDD is very slow under optimum conditions (Hutter and Philippi 1982). Long-term incubations of radiolabeled 2,3,7,8-TCDD yielded no radioactivity in carbon dioxide traps after one year, and analyses of the cultures showed that at most, 1 to 2 percent of a potential metabolite (assumed to be an hydroxylated deriva-

tive of 2,3,7,8-TCDD) could be detected. Camoni et al. (1982) added organic compost to soil in an attempt to enrich the soil and enhance the 2,3,7,8-TCDD biodegradation rate, but the soil amendment with compost was shown to have had no clear effect on degradation of 2,3,7,8-TCDD. It has been suggested that losses of dioxins from soil are due to processes other than microbial degradation.

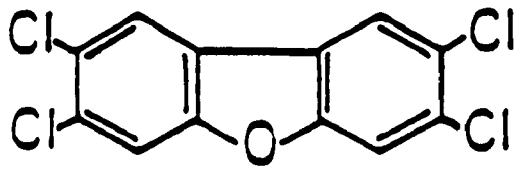
Bumpus et al. (1985) tested the white rot fungus, Phanerochaete chrysosporium, which secretes a unique H_2O_2-dependent extracellular lignin-degrading enzyme system capable of generating carbon-centered free radicals. Radiolabeled 2,3,7,8-TCDD was oxidized to labeled CO_2 by nitrogen-deficient, ligninolytic cultures of P. chrysosporium. Since the label was restricted to the ring, it was concluded that the strain was able to degrade halogenated aromatic rings. In 10 ml cultures containing 1,250 pmol of substrate, 27.9 pmol of 2,3,7,8-TCDD were converted to labeled-CO_2 during the 30-day incubation period, thus only about 2 percent of the starting material was converted.

Arthur and Frea (1988) studied the microbial activity in Times Beach, Missouri soils contaminated at levels from 0.008 to 2.4 ug/g (±10 percent) 2,3,7,8-TCDD. Although biodegradation of 2,3,7,8-TCDD per se was not tested, there were no adverse effects on numbers of microorganisms (aerobic eutrophic or oligotrophic bacteria, fungi, or actinomycetes), on enzymatic assays (including dehydrogenase, arylsulfatase, rhodanase, and acid and alkaline phosphatase), or on soil respiration (CO_2 evolution). The data suggest a lack of observed toxicity to microorganisms, and may indicate that 2,3,7,8-TCDD is not bioavailable to microorganisms because of soil binding or other factors causing low mobility.

2.3 Chemistry and Fate of 2,3,7,8-TCDF

2.3.1 Chemical Identity

2,3,7,8-Tetrachlorodibenzofuran (2,3,7,8-TCDF) is a member of the 135 compounds known as polychlorinated dibenzofurans (PCDFs). The structure of 2,3,7,8-TCDF is:



2.3.2 Chemical and Physical Properties

The few measured properties of 2,3,7,8-TCDF obtained from the literature are listed in Table 2-5. In general, fewer measured properties are available than for 2,3,7,8-TCDD, and the chemicals show somewhat similar

values for those properties where data are available or have been estimated.

2.3.3 Environmental Fate and Transport

Polychlorinated dibenzofurans can be photodechlorinated by sunlight in the presence of organic substances that can serve as donors of hydrogen atoms. This process of photodechlorination is similar to what occurs in the degradation of dioxins, and it is probably the only degradative fate pathway for dibenzofurans in the environment.

Since there is little or no information on dibenzofurans for other environmentally relevant processes, fate and transport pathways must be derived from behavior of structurally similar dioxins. Dibenzofurans can be expected then to be sorbed strongly to soils and sediments, to be bioconcentrated in fish, and to be essentially nonbiodegradable in the environment. Erosion and aquatic transport of sediment will be the main transport pathway of the sorbed dibenzofurans. Table 2-6 is a summary of the environmental fate of dibenzofurans.

(1) Photolysis. Polychlorinated dibenzofurans (PCDFs) absorb electromagnetic radiation at wavelengths above 290 nm and should be capable, therefore, of undergoing photolysis when subjected to sunlight (EPRI 1983). Crosby et al. (1973) report that polychlorinated dibenzofurans undergo photolytic dechlorinate in the presence of a hydrogen donor, with more highly chlorinated congeners being more stable. In contrast, Hutzinger (1973) and Buser (1976) report that the more highly chlorinated congeners undergo photodegradation more rapidly. Hutzinger (1973) found that both 2,8-dichloro- and octachlorodibenzofuran photodechlorinate rapidly in methanol or hexane. The potential for photodegradation of dibenzofurans in the environment appears, therefore, to be similar to the photodegradation potential of dioxins; in the presence of a hydrogen donor and sunlight, polychlorinated dibenzofurans will dechlorinate.

Buser (1988) studied the photolytic decomposition rates of 2,3,7,8-TCDF in a mixture of other chloro- and bromo-dibenzofurans and dioxins. Studies were performed in dilute hydrocarbon solutions and as solid phases on quartz surfaces under sunlight and artificial laboratory illumination (fluorescent lights). 2,3,7,8-TCDF undergoes rapid photolytic decomposition in dilute hydrocarbon solutions of i-octane. When the solutions were illuminated with sunlight in quartz vials, the estimated half-life was 220 min and 90 min in each of the two solutions. For the same solutions illuminated with artificial light in glass vials, the half lives were greater than 28 days. When 2,3,7,8-TCDF was dispersed as a thin film in quartz vials and exposed to sunlight, the estimated half-life was reported to be 35 hours. While these data do indicate that photolytic decomposition of 2,3,7,8-TCDF does occur, Buser (1988) stated the half-life values have limited accuracy because of the few data and because of the formation

Table 2-6. Summary of Environmental Fate of Dibenzofurans

Environmental process	Summary statement	Confidence in data
Photolysis	May be only natural mechanism leading to destruction of dibenzofurans.	Medium
Oxidation	No information found.	Low
Hydrolysis	Dibenzofurans are stable to hydrolysis.	High
Volatilization	No information found.	Low
Sorption	Dibenzofurans strongly sorbed by solids, especially with high organic content.	Medium
Bioaccumulation	No specific information found; potential is inconclusive.	Low
Biodegradation	Probably nondegradable in the environment.	Low

of 2,3,7,8-TCDF during photodegradation of higher halogenated forms, so the estimated accuracy of all the half-life data was reported to be ±50%. From the data, Buser (1988) concluded that the major photochemical reaction in hydrocarbon solution was reductive dehalogenation, leading to lower chlorinated dibenzofurans which would then likely be further degraded to unsubstituted dibenzofuran.

(2) Oxidation. There is no information available on the oxidation of dibenzofurans under environmentally relevant conditions (EPRI 1983).

(3) Hydrolysis. No information is available indicating that hydrolysis would be an operative environmental process for degradation of polychlorinated dibenzofurans (EPRI 1983).

(4) Volatilization. The vapor pressure of 2,3,7,8-TCDF is 9.21 x 10^{-7} mm Hg at 25°C (Eitzer and Hites 1988). The reported log Kow value is 5.82 (Burkhard and Kuehl 1986), suggesting the soil-sorption coefficient would be low. Based on these data, TCDF should not volatilize rapidly from organic soils or sediments; however, due to the stability and persistence of TCDF via other transformation and transport pathways, volatilization should not be ignored as a mechanism of loss from soils.

Experimental values have not yet been reported for the water solubility or Henry's law constant for 2,3,7,8-TCDF, but similarities to 2,3,7,8-TCDD suggest that TCDF might volatilize rapidly from water or moist surfaces. Estimations from Chemest (Versar 1989) indicate volatilization half-lives from water bodies on the order of 12 to 14 hours.

(5) Sorption. Sorption of PCDFs has not been studied (EPRI 1983). However, the log K_{ow} value of 5.82 (Burkhard and Kuehl 1986) and the structural similarity of PCDFs to PCDDs suggest that after TCDF has been washed into a soil, it will readily sorb to organic materials and other components of soils.

(6) Bioaccumulation/bioconcentration. The data available for bioconcentration factors (BCF) of 2,3,7,8-TCDF (Table 2-5) are more limited than the data for 2,3,7,8-TCDD (Table 2-3). The data suggest that BCF values for 2,3,7,8-TCDF are lower than BCF values for 2,3,7,8-TCDD.

Nabholz (1989) reported that for freshwater fish exposed to 2,3,7,8-TCDF via water only (not dietary sources), the geometric mean BCF value was 3,900 and the time to equilibrium is 7 days. This mean BCF was based on 2 measurements. This BCF is most reliable for water concentrations from 0.410 to 3.93 ppt; outside this range (especially the upper bound) this BCF is less reliable.

Nabholz (1989) also reported that for cold water species of freshwater fish, there was a significant correlation between water concentration (in ppt) and fish BCF (on a whole fish, wet weight basis): the lower the

water concentration, the higher the BCF. The regression equation describing the relationship between exposure concentration in water and fish BCF is:

$$\log Y = 3.6272 - 0.39897 (\log X)$$

where Y = fish BCF on a wet weight, whole body basis; X = water concentration in ppt; N = 2; r^2 = 1.0; and r = 1.0. This regression was calculated based on 2 measurements. This equation is most reliable for water concentrations from 0.410 to 3.93 ppt; outside this range (especially the upper bound), this equation is less reliable. The geometric mean fish BCF from this data set is 3,900.

There are apparently no data available for BCF values for 2,3,7,8-TCDF for warm water pelagic or benthic freshwater species or for marine fishes.

Nabholz (1989) reported that there was only one study available which measured the bioconcentration of 2,3,7,8-TCDF from contaminated dietary sources, and that was for a warm water species of freshwater fish. The BCF was 0.1538 on a whole body, wet or fresh weight basis, based on a dietary concentration of 182.0 ng/kg (ppt). There were apparently no data available for cold water species of freshwater pelagic fish, or for freshwater benthic species, or for marine fishes.

(7) Biotransformation and biodegradation. No information was found relating to the biodegradation of chlorinated dibenzofurans. Structurally similar chlorinated dioxins are considered to be essentially nonbiodegradable in the environment (EPRI 1983), and it is, therefore, possible that chlorinated dibenzofurans will behave in a like manner and remain persistent to attack by microorganisms and other types of biotransformation processes.

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3. DIOXIN AND FURAN HAZARD ASSESSMENT: HUMANS, TERRESTRIAL AND AVIAN WILDLIFE, AND AQUATIC LIFE

3.1 Introduction

The human health section of this chapter was compiled from:

Lee CC. 1989. Human health hazard assessment of dioxins/furans. U.S. EPA, Office of Toxic Substances. Memorandum to L. Dicker, EPA, Office of Toxic Substances, October 31, 1989.

The ecological effects section was compiled from:

Rabert WS. 1989. Update of aquatic toxicity and bioavailability data of polychlorinated dioxins and polychlorinated difurans. U.S. EPA, Office of Toxic Substances. Memorandum to S. Kroner, EPA, Office of Water Regulations and Standards, July 28, 1989.

The wildlife effects section was compiled from:

USEPA. 1990. United States Environmental Protection Agency. Assessment of risks from exposure of humans, terrestrial and avian wildlife, and aquatic life to dioxins and furans from disposal and use of sludge from bleached kraft and sulfite pulp and paper mills. Washington, DC: Office of Toxic Substances and Office of Solid Waste. EPA 560/5-90-013.

3.2 Human Hazard Assessment of PCDDs and PCDFs

The USEPA (1984a, 1985), the Ontario Ministry of the Environment (MOE) (1984), and other organizations have compiled and evaluated the existing toxicological data on PCDDs and PCDFs. Although there is extensive literature on a few of these isomers, the toxicological information on these families of more than 200 compounds (75 PCDD and 135 PCDF isomers) is far from complete. Nevertheless, a growing body of information on mechanisms of action and structure-activity relationships (SARs) within these families of compounds makes it possible, with reasonable confidence, to infer information where data are missing.

Among the 210 congeners of PCDDs and PCDFs, the compound that appears to be the most toxic and has generally raised the greatest health concerns is 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD). Experimental studies with 2,3,7,8-TCDD in animal systems have demonstrated a variety of toxic effects resulting from exposure to this compound (USEPA 1985). These effects include carcinogenesis, cancer promotion, reduced fertility and postnatal survival, teratogenic effects, immunotoxic effects, thymic atrophy, liver damage, effects on the thyroid, and chloracne and other effects on the skin. Acute exposures of sensitive species of animals to 2,3,7,8-TCDD result in a characteristic

"wasting syndrome," followed by death. Extensive experimental studies have revealed marked variations among species in both the array of effects caused by 2,3,7,8-TCDD and the dose levels at which these effects are elicited (USEPA 1985, Pitot et al. 1986). Limited toxicological testing of other PCDDs and PCDFs has demonstrated that several of these compounds cause similar toxicological effects, but that higher doses of these compounds are generally required to cause effects of comparable magnitude to those induced by 2,3,7,8-TCDD.

The nature and extent of effects in humans exposed to 2,3,7,8-TCDD are not nearly so well defined (Ontario MOE 1984, USEPA 1985, Pitot et al. 1986). There is a consensus that exposure of humans to 2,3,7,8-TCDD can result in a skin condition known as chloracne, an acne-like lesion which, while not life-threatening, can be disfiguring, persistent, and refractory to treatment.

The evidence for an association of 2,3,7,8-TCDD with human cancer is equivocal. Several studies of human populations exposed to chemical mixtures containing 2,3,7,8-TCDD have suggested increased frequencies of certain cancers (Hardell and Sandstrom 1979, Hardell et al. 1981, Thiess et al. 1982, MDPH 1983b), but inconsistencies among the studies and incomplete characterization of exposure make the evidence inconclusive (USEPA 1985, Blair 1986). Evidence for reproductive impairment in humans exposed to 2,3,7,8-TCDD, including one study conducted in Midland County, Michigan (MDPH 1983a), is inconclusive for similar reasons (USEPA 1985, Kimbrough 1986). Other effects in humans that have been more clearly associated with exposure to mixtures containing 2,3,7,8-TCDD include disturbances in lipid metabolism (Moses et al. 1984, Suskind and Hertzberg 1984) and increased frequency of gastric ulcers (Bond et al. 1983, Suskind and Hertzberg 1984).

For the PCDFs, compared with the PCDDs, there is more specific and quantitative information available on toxic effects in humans as a result of two large-scale poisoning incidents in Japan and Taiwan (Kuratsune and Shapiro 1984). Over periods of weeks to months, the affected persons ingested food contaminated with a mixture of PCDFs, polychlorinated biphenyls (PCBs) and polychlorinated quaterphenyls (PCQs). Comparative toxicological studies have indicated that PCDFs were the primary toxic agents in these poisoning incidents and that 2,3,4,7,8-PCDF was probably the most important single compound (Bandiera et al. 1982, Masuda and Yoshimura 1984, Kunita et al. 1984, 1985, Masuda et al. 1985, Chen et al. 1985, Miyata et al. 1985). The most prominent toxic signs were skin eruptions similar to those of chloracne, along with skin pigmentation and eye abnormalities (Lu and Wong 1984, Urabe and Asahi 1985). Other effects reported include impairments in lipid metabolism and immune function (Okumura et al. 1974, Chang et al. 1982a,b) and persistent respiratory symptoms (Nakanishi et al. 1985). A preliminary report by Kuratsune et al. (1987) indicated a significant excess frequency of liver cancer deaths (9 observed vs. 1.6 expected) and possibly lung cancer

among male victims within 15 years after exposure. However, the excess incidence of liver cancer deaths had an uneven geographic distribution, and four of the deaths occurred relatively soon after the poisoning incident. In addition, potent confounding variables, such as alcohol consumption, were not assessed. Reported effects on reproduction include menstrual disturbances (Kusuda 1971), skin hyperpigmentation in infants (Yamashita and Hayashi 1985, Hsu et al. 1985), and perinatal mortality (Hsu et al. 1985). These effects observed in humans are qualitatively similar to those reported in animals exposed to PCDFs and PCDDs (McNulty 1985); this provides support for the use of animal data as the basis for hazard assessment for other members of these families of compounds.

3.3 Human Health Hazard of 2,3,7,8-TCDD

3.3.1 Cancer Effects

2,3,7,8-TCDD is classified by the EPA as a "probable human carcinogen" (Category B2, according to the EPA (1986) classification scheme), based on "sufficient" evidence in animals but "inadequate" evidence in humans (USEPA 1988a). 2,3,7,8-TCDD is the most potent animal carcinogen which has been evaluated by EPA (USEPA 1985). Very low oral doses of 2,3,7,8-TCDD have increased the incidence of tumors at a variety of sites in rats, mice, and hamsters (USEPA 1985, Rao et al. 1988), principally and most consistently in the liver. As discussed below, the tumor data for the female rat (Kociba et al. 1978) and the male mouse (NTP 1982) were used in estimating the increased cancer risk from exposure to 2,3,7,8-TCDD. In a dietary study (Kociba et al. 1978), 2,3,7,8-TCDD caused significantly increased incidences of tumors in the liver (neoplastic nodules/heptaocellular carcinoma), lung (keratinizing cell carcinoma), and nasal turbinates/hard palate (squamous cell carcinoma) in female Sprague-Dawley rats. In a National Toxicology Program (NTP 1982) gavage study, the incidence of hepatocellular carcinoma was significantly increased in male B6C3F1 mice.

USEPA, U.S. Food and Drug Administration (FDA) and U.S. Consumer Product Safety Commission (CPSC) each derived a cancer risk estimate for 2,3,7,8-TCDD (USEPA 1985, 1988b, FDA 1983, Babich 1988). The risk estimates differ by as much as a factor of 10 (EPA vs. FDA). This range reflects the current lack of consensus among the agencies regarding approaches to carcinogen risk assessment and dose-response modeling. All three agencies based their estimates on a multistage model with linear-at-low-dose extrapolation procedures, but differed with respect to selection of animal data and details of extrapolation. Table 3-1 shows the factors used by each agency and the resulting risk estimates. The dose-response modeling terminology used in the following discussion of the cancer risk estimates is defined in Table 3-2.

EPA employed the linearized multistage (LMS) procedure to estimate a plausible upper bound slope factor, designated as q_1^* for carcinogens.

Table 3-1. Factors Used by EPA, FDA, and CPSC in Calculating Their Risk Estimates for 2,3,7,8-TCDD Using Multistage Models

Factor	EPA	FDA	CPSC	Effect of difference on upper-bound unit risk estimates (EPA vs FDA only)
Animal study used	Kociba female rat feeding study	Kociba female rat feeding study	NTP male mouse gavage study	
Pathologist (Kociba or Squire)	Both	Kociba	NA	Less than 10%
Adjustment for early mortality	Yes	No	NA	Adjustment changes estimates by a factor of +1.7 or 1/2.6[a]
Selection of tumor types	Liver, lung, hard palate/ nasal turbinates	Liver only	Liver only	Less than 10%
Animal-to-man dose equivalence	Dose/surface area	Dose/body weight	Dose/surface area	Dose/surface area increases estimate by a factor of 5.38
Dose used for curve fit	Administered	Administered	Administered	
Extrapolation curve	Upper-bound	Upper-bound	Maximum likelihood estimate	
Slope factor[b] $(pg/kg\text{-}day)^{-1}$	1.6×10^{-4}	1.8×10^{-5}	6.7×10^{-5}	
Dose at risk of 10^{-6}; units of pg/kg/day	0.006	0.060	0.015	

[a] Adjustment increases by a factor of about 1.7 compared with unadjusted Kociba anlaysis; adjustment decreases estimate by a factor of 2.6 compared with unadjusted Squire analysis where high dose is excluded due to poor fit.

[b] The slope factors should not be used if the exposure (in pg/kg/day) exceeds 63 for EPA (556, FDA; 149, CPSC), since above these exposures the slope factor may differ from that stated. These exposures are associated with a risk of 1 in 100 or greater.

Source: Adapted from USEPA (1988b).

Table 3-2. Terms Associated with Dose-Response Modeling

Modeling	An estimation of the incremental or extra cancer risk to humans arrived at by fitting a mathematical function to animal response data.
Coefficient (or parameter)	A constant in the model, associated either with the control response, or the time or dose variable inputs. For example, in the Crump linearized multistage procedure, the coefficient associated with the variable that is linear in dose is denoted as q_1.
Maximum likelihood estimate (MLE)	The estimate from a statistical procedure by which the coefficients of the model are estimated to fit the observed data. The MLE has many properties, in a statistical sense, which allow it to be referred to as a "statistical average" or "best" estimate.
q_1	In the Crump multistage procedures, q_1 is the coefficient associated with the variable that is linear in dose. q_1 can be defined as the increase in cancer risk associated with an incremental increase per unit of dose. For this reason, q_1 is expressed in units of reciprocal dose such as $(ng/kg\text{-}day)^{-1}$.
q_1^*	In the Crump multistage procedure q_1^* is the upper bound estimate associated with the linear term. q_1^* is expressed in units of reciprocal dose such as $(ng/kg/day)^{-1}$. The upper bound increased lifetime cancer risk associated with a given exposure can be estimated by multiplying the q_1^* $(ng/kg/day)^{-1}$ times the exposure (ng/kg/day).
Risk-specific dose (RsD)	A dose (loosely, an exposure) associated with a specified cancer risk. For example, suppose a linearized multistage procedure is used with some data and the coefficient estimates are $q_1 = 3.0 \times 10^{-3}$ $(ng/kg\text{-}day)^{-1}$ and $q_1^* = 7.5 \times 10^{-3}$ $(ng/kg\text{-}day)^{-1}$. Then for an extra risk of 1 in 1,000,000, the dose (d) would be the solution to $10^{-6} = 1\text{-}\exp(-7.5 \times 10^{-3} d)$, and $RsD = 1.3 \times 10^{-4}$ ng/kg-day would be called its risk-specific dose. If the MLE is used instead of the upper bound (as by CPSC), then q_1 is used instead of q_1^* and the solution here would be $RsD = 3.3 \times 10^{-4}$ ng/kg-day.
Slope factor	The slope of the upper bound dose extrapolation model at doses approaching zero. Also called q_1^*.
Unit risk	The incremental upper bound lifetime risk estimated to result from lifetime exposure to an agent if it is in the air at a concentration of 1 microgram per cubic meter or in the water at a concentration of 1 microgram per liter.

Table 3-2. (continued)

Upper-confidence bounds (UB)	The estimates resulting from a statistical procedure in which the upper-limit values of the coefficients still consistent with the data are estimated. In the linearized multistage procedure (Crump), the upper limit associated with the linear term is designated q_1^*. It is selected to be the 95% upper-limit estimate, in a statistical sense, of the linear term associated with the fitting of a multistage model to the animal data. In making cross-species extrapolations to humans, however, the "95%" label is dropped, since the uncertainty associated with cross-species extrapolations is considered far greater than the statistical uncertainty associated with the model-fitting procedure.

FDA used a similar extrapolation procedure to estimate the upper bound slope factor. CPSC used the maximum likelihood estimate from the multistage model, since it, as well as the upper bound, is linear at low dose for the data set used. CPSC also differed with EPA and FDA in the selection of animal data. CPSC used the liver tumor data for male mice from the NTP (1982) gavage study, whereas EPA and FDA used the tumor data for female rats from a dietary study (Kociba et al. 1978). EPA and FDA differed in their selection of tumor types in female rats. EPA combined all tumor types of significantly increased incidence, whereas FDA considered only liver tumor incidence. EPA made two adjustments in the data not made by FDA. EPA adjusted for high early mortality in high-dose animals by excluding animals dying during the first year. EPA also incorporated two pathologists' reviews of the liver slides by using the geometric mean of the slope factors derived from each pathologist's analysis, while FDA considered only the original pathologist's review of the slides. While these differences in data selection account for some of the difference in the EPA and FDA risk estimates (Table 3-1), the major contributing factor was choice of interspecies dose scaling factor. EPA used the surface area correction, whereas FDA used dose per body weight. When extrapolating from rat to man, the use of dose per surface area versus dose per body weight increases the risk estimate by a factor of 5.4. CPSC, which used the male mouse data, applied the surface area correction, but the CPSC selection of extrapolation procedures cannot be compared quantitatively with that of the other agencies because of the use of different data sets.

USEPA estimated a plausible upper bound slope factor (q_1^*) of 1.6×10^{-4} $(pg/kg\text{-}day)^{-1}$ for 2,3,7,8-TCDD. This q_1^* gives an exposure (also called risk-specific dose, see Table 3-2) of 0.006 pg/kg/day at an upper bound increased lifetime cancer risk of one in a million (USEPA 1985). The FDA derived a q_1^* of 1.8×10^{-5} $(pg/kg\text{-}day)^{-1}$, which gives an exposure of 0.06 pg/kg/day for an increased upper bound lifetime cancer risk of one in a million (USEPA 1988a). CPSC calls for the use of the maximum likelihood estimate (MLE) of the unit risk; for humans this is 6.7×10^{-5} $(pg/kg\text{-}day)^{-1}$. Thus, the exposure (here also an MLE) at which the extra lifetime cancer risk is one in a million was estimated by CPSC to be 0.015 pg/kg/day (Babich 1988).

3.3.2 Non-Cancer Effects

In assessing the risk of non-cancer effects, USEPA is concerned about two aspects of exposure, the dose level and the duration. Adverse effects could result from long-term, low-level exposure, or from a relatively brief exposure to a high dose. For evaluating long-term exposures, USEPA has adopted the concept of the Reference Dose (RfD) (USEPA 1987a). The RfD is defined as an estimate of the lifetime daily exposure to the human population likely to be without any appreciable risk of a deleterious effect. For assessing brief exposures to high levels, USEPA has adopted procedures to establish exposure levels for

issuing "health advisories" (HA) (USEPA 1987b). Thus, the HA dose level is appropriate for comparison with single dose or single-day intakes, or short-term exposure, whereas the RfD is appropriate for comparison with long-term or lifetime exposures. Both values are operationally derived from the "No Observed Adverse Effect Level (NOAEL)" determined in the critical toxicological study, divided by an "Uncertainty Factor (UF)" selected on the basis of specific attributes of the study.

In a previous risk assessment, EPA has determined that reproductive effects and developmental toxicity in animals are the most critical or sensitive noncarcinogenic effects to consider for risk assessment of 2,3,7,8-TCDD (USEPA 1988a). A number of studies have been conducted to assess the possible adverse effects of chronic 2,3,7,8-TCDD exposure on reproductive capacity. These studies have shown that exposure to 2,3,7,8-TCDD prior to mating, as well as during pregnancy, results in reduced fertility, fetotoxicity, and/or reduced survival of the offspring. A three-generation rat reproductive study (Murray et al. 1979) was selected as the "critical" study for determining the RfD for low-level, long-term exposure to 2,3,7,8-TCDD. A combined UF of 1000 was selected, which includes three subfactors of 10: one subfactor of 10 because the lowest administered dose of 1 ng/kg/day was not a NOAEL, the second subfactor of 10 to account for possible interspecies differences in sensitivity, and the third subfactor to account for possible intraspecies differences in sensitivity. Thus, for purposes of this risk assessment an estimated RfD of 1 pg/kg/day was calculated by dividing the lowest dose administered (1 ng/kg/day) by the UF of 1000.

A number of animal studies have been conducted to assess the potential developmental toxicity of short-term 2,3,7,8-TCDD exposure during pregnancy (USEPA 1985). These studies clearly demonstrated that exposure to 2,3,7,8-TCDD during pregnancy is teratogenic and/or fetotoxic in all animal species tested. The most common malformations observed in mice after 2,3,7,8-TCDD exposure are cleft palate and kidney anomalies; while edema, hemorrhage, and kidney anomalies are most commonly observed in rats. At higher doses, 2,3,7,8-TCDD has marked fetotoxic effects in both rats and mice, as measured by decreased fetal body weight and increased fetal death. Adverse effects have also been demonstrated in nonhuman primates (Bowman et al. 1989a, b).

For purposes of this risk assessment, a rat teratology study (Sparschu et al. 1971) was selected as the "critical" study for estimating a HA. The NOAEL in this study was 30 ng/kg/day and an UF of 100 was used, which includes two subfactors of 10, one each to account for possible interspecies and intraspecies differences in sensitivity; these data yield an estimated HA of 300 pg/kg/day.

Since the estimated RfD and HA for noncarcinogenic effects are derived from reproductive and teratology studies, some uncertainty arises as to whether these values are applicable to people of nonreproductive age (e.g., children or post-menopausal women), or people who are not reproducing for other reasons. The RfD is probably applicable to the

general population since 2,3,7,8-TCDD is stored for periods of years in the body fat, and hence may exert effects long after the exposure occurred. To derive the HA which could be applied directly to any population except pregnant women, the data selected were from an acute toxicity study in which hepatic effects were the sensitive endpoint (Turner and Collins 1983). The LOAEL for hepatic lesions in this study was 100 ng/kg/day. For purposes of this risk assessment, a one-day HA of 100 pg/kg/day was calculated, using procedures for issuing HA (USEPA 1987b), by dividing the LOAEL by the UF of 1,000. A 10-day HA of 10 pg/kg/day was calculated by dividing the LOAEL by an additional UF of 10 (a total UF of 10,000).

3.3.3 Toxicity Equivalence Factors

The toxicity equivalency factor (TEF) method is an interim procedure for assessing the risks associated with exposures to complex mixtures of chlorinated dibenzo-p-dioxins and dibenzofurans (USEPA 1989). The method relates the toxicity of the 210 structurally related chemicals and is based on a limited data base of *in vivo* and *in vitro* toxicity testing. By relating the toxicity of the 209 PCDDs and PCDFs to the highly-studied 2,3,7,8-TCDD, the approach simplifies the assessment of risks involving exposures to mixtures of PCDDs and PCDFs.

Little is known about the toxicity of the various PCDDs or PCDFs. Of the limited number of these congeners tested thus far, other than 2,3,7,8-TCDD, only a mixture of 1,2,3,6,7,8- and 1,2,3,7,8,9-HxCDD has been tested for carcinogenicity and liver tumors in rats and mice when administered at low doses for a lifetime (NCI 1980). A larger body of data is available on short-term *in vivo* studies and a variety of *in vitro* studies for a number of PCDDs and PCDFs (NATO-CCMS 1988b). These experiments cover a wide variety of end points (e.g., LD_{50}, body weight loss, thymic atrophy and dysfunction of the immune system, cell transformation, the enzyme aryl hydrocarbon hydroxylase (AHH) induction, and reproductive and/or teratogenic effects). While the doses necessary to elicit the toxic responses differ in each case, the relative potencies of the different compounds (compared to 2,3,7,8-TCDD) are generally consistent from one end point to another.

This toxicity information, developed by researchers in several labs around the world, reveals a strong structure-activity relationship between the chemical structure of a particular PCDD or PCDF congener and its ability to elicit a biological/toxic response in various *in vivo* and *in vitro* test systems (Bandiera et al. 1984, Bellin and Barnes 1987, NATO-CCMS 1988a,b, Olson et al. 1989). Research has also revealed a mechanistic basis for these observations. That is, a necessary (but not sufficient) condition for expression of much of the toxicity of a given PCDD or PCDF congener is its ability to bind with great specificity to a particular protein receptor located in the cytoplasm of the cell. This congener-receptor complex then migrates to the nucleus of the cell, where

it initiates reactions leading to the expression of toxic effects (Poland and Knutson 1982).

To assess the risks of mixtures of PCDDs and PCDFs, the TEF approach, first suggested in 1977 (Grant 1977), was pursued by scientists and jurisdictions during the early- and mid-1980s (Ontario MOE 1982, Swiss Government 1982, Commoner et al. 1984, Eadon et al. 1986, California Air Resources Board 1986). As a result, numerous slightly different TEFs existed, thus complicating the communication among scientists and agencies in addressing the toxicological significance of complex mixtures of PCDDs and PCDFs.

An international effort was conducted under the auspices of the North Atlantic Treaty Organization's Committee on Challenges of Modern Society (NATO-CCMS) with participation by scientists from United States and other countries. The International group adopted a common set of TEFs, the International TEFs/89 (I-TEFs/89) (NATO-CCMS 1988a,b) to promote consistency in addressing contamination involving PCDDs and PCDFs. USEPA has adopted the I-TEFs/89 as a revision to the EPA-TEFs/87 currently in use for assessing risks of PCDDs and PCDFs other than 2,3,7,8-TCDD (USEPA 1989). The I-TEF is used to estimate carcinogenic and noncarcinogenic effects of PCDD/PCDF mixtures in terms of an equivalent amount of 2,3,7,8-TCDD, as if they were concentrations of 2,3,7,8-TCDD itself. The I-TEFs and TEFs used by other groups are presented in Table 3-3. Relative potency data for the PCDD and PCDF isomers substituted in the 2,3,7,8-positions are shown in Table 3-4.

The International Report by NATO-CCMS (1988a) identified limitations to the TEF approach, e.g., the extrapolation from short-term to long-term effects and the possible differences in metabolic effects among species. For example, many of the short-term results seen in the metabolic systems of mice have not been observed in similar systems in rats. Also, the connection between the enzyme induction response (which supports several of the TEF values) and several of the toxic end points manifested by PCDDs/PCDFs is unclear. Other mechanisms of action (e.g., effect on vitamin A synthesis and estrogen-like activity) have been suggested as playing an important role in the toxicity of PCDDs/PCDFs. Thus, the TEF approach remains interim in nature and will be revised as new data are developed.

It should also be noted that the accuracy of an assessment of the risks associated with exposure to a mixture of PCDDs and PCDFs depends upon a number of factors, of which the uncertainty of the TEF approach is only one and perhaps not the largest. The uncertainties relating to estimated intakes, bioavailability, interspecies extrapolation, safety factors or mathematical models, and possible antagonistic or synergistic interactions are likely to carry as much or more uncertainty than the TEF values themselves.

Table 3-3. T-TEFs/89 and TEFs Developed by Other Groups

Basis compound	Swiss Government[a]	Grant[b] Olie[c] Commoner[d]	New York State[e]	Ontario[f]	FDA[g]	CA[h]	EPA 1981[i]	EPA current recommendation	I-TEFs/89
(Basis)	Enzyme		LD_{50}	Various effects	Various effects			Various effects	Various effects
Mono thru di CDDS	0	0	0	0	0	0	0	0	0
Tri CDDS	0	0	0	1		0	0	0	0
2,3,7,8-TCDD	1	1	1	1	1	1	1	1	1
Other TCDDS	0.01	1	0	0.01	0	0	1	0.01	0
2,3,7,8-PeCDDs	0.1	0.1	1	1	0	1	0	0.5	0.5
Other PeCDDs	0.1	0.1	0	0.01	0	0	0	0.005	0
2,3,7,8-HxCDDs	0.1	0.1	0.03	1	0.02	1	0	0.04	0.1
Other HxCDDs	0.1	0.1	0	0.01	0.02	0	0	0.0004	0
2,3,7,8-HpCDDs	0.01	0.1	0	1	0.005	1	0	0.001	0.01
Other HpCDDs	0.01	0.1	0	0.01	0.005	0	0	0.00001	0
OCDD	0	0	0	0	<0.00001	1	0	0	0.001
2,3,7,8-TCDFs	0.1	0.1	0.33	0.02	0	1	0	0.1	0.1
Other TCDFs	0.1	0.1	0	0.0002	0	0	0	0.001	0
2,3,7,8-PeCDFs	0.1	0.1	0.33	0.02	0	1	0	0.1	0.05,0.5[j]
Other PeCDFs	0.1	0.1	0	0.0002	0	0	0	0.001	0
2,3,7,8-HxCDFs	0.1	0.1	0.01	0.02	0	1	0	0.01	0.1
Other HxCDFs	0.1	0.1	0	0.0002	0	0	0	0.002	0

Table 3-3. (continued)

Basis compound	Swiss Government[a]	Grant[b] Olie[c] Commoner[d]	New York State[e]	Ontario[f]	FDA[g]	CA[h]	EPA 1981[i]	EPA current recommendation	I-TEFs/89
(Basis)	Enzyme		LD_{50}	Various effects	Various effects			Various effects	Various effects
2,3,7,8-HpCDFs	0.1	0.1	0	0.12	0	1	0	0.001	0.01
Other HpCDFs	0	0.1	0	0.002	0	0	0	0.00001	0
OCDF	0	0	0	0	0	0	0	0	0.001

[a]Swiss Government 1982.
[b]Grant 1977.
[c]Olie et al. 1983.
[d]Commoner et al. 1984.
[e]Eadon et al. 1986.
[f]Ontario MOE 1982.
[g]U.S. DHHS 1983.
[h]Gravitz et al. 1983.
[i]U.S. EPA 1981.
[j] 0.05 for 1,2,3,7,8-PeCDF, 0.5 for 2,3,4,7,8-PeCDF

Table 3-4. International Toxicity Equivalency Factors/89 (I-TEFs/89): Comparison of Relative Potency Data for the 2,3,7,8-Substituted CDDs and CDFs

Congener	I-TEFs/89	Observed TEF ranges (In vivo toxicities)	Aryl hydrocarbon hydroxylase (AHH) induction (in vivo)	Aryl hydrocarbon hydroxylase (AHH) induction (in vitro)
2,3,7,8-TCDD	1	1	1	1
1,2,3,7,8-PCDD	0.5	Range = 0.053 - 0.59 Data = (0.59[ml], 0.42[gl], 0.081[r], 0.053[r])	0.13 (0.13[r])	Range = 0.0065 - 0.011 Data = (0.011/0.0065[h])
1,2,3,4,7,8-HxCDD	0.1	Range = 0.018 - 0.24 Data = (0.24[ml], 0.084[r], 0.018[gl], 0.13[r])	0.13 (0.13[r])	Range = 0.034 - 0.046 Data = (0.034/0.046[h])
1,2,3,6,7,8-HxCDD	0.1	Range = 0.015 - 0.16 Data = (0.16[ml], 0.015[gl])	-	0.012 (0.012[hB])
1,2,3,7,8,9-HxCDD	0.1	Range = 0.016 - 0.14 Data = (0.016[gl], 0.14[ml])	-	0.008 (0.008[hB])
1,2,3,4,6,7,8-HpCDD	0.01	-	-	0.003 (0.003[hB])
OCDD	0.001	-	0.0002	0.0006 (0.0006[hB])
2,3,7,8-TCDF	0.1	Range = 0.016 - 0.17 Data = (0.017[gl], 0.17[ml], 0.05[ml], 0.025[4], 0.016[r])	0.006 (0.006[r])	Range = 0.018 - 0.09 Data = (0.018/0.09[h])
2,3,4,7,8-PCDF	0.5	Range = 0.048 - 0.80 Data = (0.8[ml], 0.47[g], 0.43[r], 0.13[gl], 0.12[mt], 0.048[r])	0.11 - 0.23 (0.23[g], 0.11[r])	Range = 0.28 - 1.41 Data = (0.28/1.41[h])
1,2,3,7,8-PCDF	0.05	Range = 0.019 - 0.95 Data = (0.95[g], 0.05[r], 0.031[mt], 0.019[r])	0.003 - 0.047 (0.047[g], 0.003[r])	Range = 0.028 - 0.06 Data = (0.06/0.028[h])

Table 3-4. (continued)

Congener	I-TEFs/89	Observed TEF ranges (In vivo toxicities)	Aryl hydrocarbon hydroxylase (AHH) induction (in vivo)	Aryl hydrocarbon hydroxylase (AHH) induction (in vitro)
1,2,3,4,7,8-HxCDF	0.1	Range = 0.013 - 0.18 Data = (0.18[r], 0.038[r], 0.013[mt])	0.014 (0.014[r])	Range = 0.20 - 0.50 Data = (0.20/0.50[h])
1,2,3,6,7,8-HxCDF	0.1	Range = 0.016 - 0.097 Data = (0.097[r], 0.016[r])	0.012 (0.012[r])	Range = 0.049 - 0.153 Data = (0.049/0.153[h])
1,2,3,7,8,9-HxCDF	0.1	-	-	-
2,3,4,6,7,8-HxCDF	0.1	Range = 0.011 - 0.097 Data = (0.097[r], 0.018[r], 0.011[g])	0.015 (0.015[r])	Range = 0.11 - 0.33 Data = (0.11/0.33[h])
1,2,3,4,6,7,8-HpCDF	0.1	-	-	-
1,2,3,4,7,8,9-HpCDF	0.01	-	-	-
OCDF	0.001	-	-	-

[g] guinea pig data
[r] rat data
[gl] guinea pig lethalities
[ml] mouse lethalities
[mt] mouse teratogenicity
[mi] mouse immunotoxicity
[h] rat hepatoma data (ratio of activity of aryl hydrocarbon hydroxylase (AHH) to activity of ethoxyresorufin O-deethylase)
[h0] rat hepatoma data (activity of AHH)

Source: NATO-CCMS (1988b).

3.4 Ecological Hazard of 2,3,7,8-TCDD and 2,3,7,8-TCDF

Based on the available data, the most toxic congener of PCDDs appears to be 2,3,7,8-TCDD. Many aquatic tests have been conducted with 2,3,7,8-TCDD, but most of these studies were either bioavailability tests or short-term exposure studies with long post-exposure observation periods. The available test data on 2,3,7,8-TCDD indicate that none of the studies are adequate to define an acceptable, no-observed-effect concentration (NOEC). Only one study exposed a fish to 2,3,7,8-TCDD in water for a reasonable duration, 28 days. Even then, the exposure duration in the rainbow trout study was only about half of the 48 days estimated to be required to achieve 90 percent of the steady-state BCF level in fish. The NOEC was less than 0.038 ng/l, a concentration which produced 45 percent mortality in rainbow trout fry.

The mode of action of 2,3,7,8-TCDD is unknown, but it appears related to neural toxicity and may affect the immune system, like many other organochlorine chemicals. The pattern of TCDD-induced mortality typically occurs between 30 to 80 days after the initial exposure, even for exposure as short as 6 hours. Mortality in fish appears to be a function of both exposure duration and the test concentration. None of the bioconcentration tests were of sufficient duration to achieve a steady-state bioconcentration factor (BCF). All BCF values have been estimated using the uptake rate (k_1) and depuration rate (k_2) values.

Many sublethal effects have been reported for polychlorinated dioxins and furans. 2,3,7,8-TCDD elicits a broad range of toxic effects which include: reduced growth; weight loss; abnormal hatching; cellular alterations; numerous types of lesions in a broad spectra of organs; and behavioral effects on swimming, reduced feeding, and loss of response to external stimuli.

In a risk assessment, the normal procedure is to identify the NOEC for the most sensitive toxicological endpoint and apply a safety margin (e.g., 100) to that NOEC. Unfortunately, the lowest tested concentration (0.038 ng/l) among all known 2,3,7,8-TCDD studies produced 45 percent mortality. Consequently, a definitive NOEC for 2,3,7,8-TCDD has not yet been determined. All studies that have been completed are of insufficient duration to determine the full extent of TCDD effects. For purposes of this assessment, it was determined that the margin of safety for TCDD should be 1000. This factor is justified for use in this assessment because of the high level of lethality found at 0.038 ng/l, because of the exposure duration which was too short for steady-state equilibrium, and because of the absence of exposure to pre-spawning adults and TCDD-deposition in egg yolk, which generally yields the most sensitivity life stage for chlorinated chemicals.

3.4.1 Aquatic Toxicity of PCDDs

The available studies on PCDDs and PCDFs published after 1984 were collected and reviewed in this document to update the aquatic toxicity literature published subsequent to the comprehensive EPA reviews in the ambient water quality criteria document (USEPA 1984a) and the health and environmental effects profile (USEPA 1984b). Thus, the information contained herein does not include a detailed review of earlier aquatic toxicity data. Test concentrations and effects are summarized for PCDDs in Table 3-5 and for PCDFs in Table 3-6.

It appears that the most toxic PCDD is 2,3,7,8-TCDD. While several methods for calculation of toxic equivalence have been developed for 2,3,7,8-TCDD and other dioxin congeners, the methods have substantially different toxicity factors and therefore have considerably different outcomes (Helder and Seinen 1985).

Moreover, PCDDs appear to be more toxic to fish than to other aquatic species. However, aquatic invertebrates also show toxic effects, including oligochaetes, pulmonate snails, and mosquito larvae exposed to 2,3,7,8-TCDD at 0.2 ppb for 55, 36, and 36 days, respectively (Miller et al. 1973).

Aquatic studies with 2,3,7,8-TCDD have generally been conducted either as continuous long-term exposures or short-term exposures (6 to 96 hours) with long-term observation periods. The long observation periods are necessary to monitor the delayed mortality which is characteristic of 2,3,7,8-TCDD. Most of the lethal effects after short-term exposures are reported to be delayed (e.g., for from 30 to 50 or more days following exposure). The occurrence of delayed effects has some serious implications for both hazard evaluations and risk assessments. For example, the reported results of typical acute toxicity tests are suspect because of the short observation periods. In risk scenarios, sporadic acute releases may cause serious delayed effects. Of most concern is that there are insufficient data to assess the effects from the many complex combinations of exposure concentrations and the various durations of the exposure that occur in nature.

Delayed mortality found in dioxin studies indicates that the most appropriate method of reporting the effects of dioxin is not a simple 96-hour LC_{50} value or a maximum acceptable toxicant concentration (MATC) range. The toxicity of 2,3,7,8-TCDD is a function of the duration of the exposure, as well as the dose level. At any given test concentration, continuous exposures appear to produce toxic effects in a shorter time than do short-term exposures. Table 3-5 summarizes dioxin effects from various routes of exposure, including water, sediment, diet, and intraperitoneal injection. Based on the data in Table 3-5, exposure via water appears to be the most toxic, single route of exposure; however, it is likely that the degree of an effect could be increased by

Chemical form	Test species	Duration (days) Exposure	/ Observation	Concentration (ng/l)	Toxic effects	Reference
Monochloro-	No data					
Dichloro-	No data					
Trichloro-	No data					
Tetrachloro-						
1,2,3,7-TCDD	Rainbow trout (fry - 0.2 g)	5 (water)	/ + 24 days	134 54	No deaths No deaths	Muir et al. (1985)
	Fathead minnow (0.2 g)	5 (water)	/ + 24 days	28 23	No deaths No deaths	Muir et al. (1985)
	Rainbow trout (0.5 - 1.0g)	30 (diet)	/ + 30 days	110 ng/g	No significant (P = 0.05) adverse effects	Muir et al. (1988)
	Fathead minnor (1.0 g)	30 (diet)	/ + 30 days	110 ng/g	No significant (P = 0.05) adverse effects	Muir et al. (1988)
1,3,6,8-TCDD	Rainbow trout (0.1 - 0.5 g)	5 (water)	/ + 48 days + 48 days + 48 days	211 74 4	No deaths No deaths No deaths	Muir et al. (1986)
	Fathead minnow (1.5 - 2.5 g)	5 (water)	/ + 29 days + 48 days	41 10	No deaths No deaths	Muir et al. (1986)
2,3,7,8-TCDD	Fathead minnow	20 (water)	/ + 20 days	1.0	3 out of 30 dead	Adams et al. (1986)
	Fathead minnow	1,2,3,4 (water)	/ + 60 days	0.0 0.12	10% dead <5% dead	Adams et al. (1986)

Table 3-5. (continued)

Chemical form	Test species	Duration (days) Exposure	/ Observation	Concentration (ng/l)	Toxic effects	Reference
2,3,7,8-TCDD (continued)	Fathead minnow (28-day LC_{50} 1.7 ng/l)	28 (water)	/ + 20 days	1.7 6.7 63	53% dead (28 days) 100% dead (22 days) 100% dead (12 days)	Adams et al. (1986)
	Coho salmon	1 (water)	/	0.056	Deaths after several weeks	Miller et al. (1973)
	Rainbow trout (35 g)	0.25 (water)	/ +139 days	107	4 deaths, one each on days 78, 136, 137, 139 and decreased growth	Branson et al. (1985)
	Rainbow trout (yolk sac fry)	4 (water)	/ + 16 days	54.6 33.1 20.1 7.4 4.5 2.7 1.6	100% dead (13 days) 100% dead (15 days) 100% dead (17 days) 95% dead (20 days) 63% dead (20 days) 20% dead (20 days) 4% dead (20 days)	Helder and Seinen (1985)
	Rainbow trout (fry - 0.38 g)	28 (water)	/ + 28 days	0.789 0.382 0.176 0.079 0.038 0.0011 (control)	85% dead (28 days) 73% dead (28 days) 50% dead (28 days) 95% dead (56 days) 18% dead (28 days) 83% dead (56 days) 6% dead (28 days) 45% dead (56 days) 5% dead (28 days) 7% dead (56 days)	Mehrle et al. (1988)
	Daphnia magna (48-hr EC_{50} >1,030 ng/l)	2 (water)	/ + 7 days	0.2 - 1,030	No mortality	Adams et al. (1986)

Table 3-5. (continued)

Chemical form	Test species	Duration (days) Exposure / Observation	Concentration (ng/l)	Toxic effects	Reference
2,3,7,8-TCDD (continued)	Rainbow trout	91 (diet) / + 91 days	494	No overt effects	Kleeman et al. (1986a)
	Yellow perch	91 (diet) / + 91 days	494	No overt effects	Kleeman et al. (1986b)
	Rainbow trout	1 dose intraperitoneal injection (i.p.) / + 14 days	10 ng/kg 1.0 0.1 0.01	Hypophagic, etc. Normal Normal Normal	Spitsberg (1986)
	Rainbow trout (LC50 - 10 ug/kg)	1 dose (i.p.) / + 80 days	125 25 5 1	95% dead (80 days) 90% dead (80 days) 20% dead (80 days) 0% dead (80 days)	Spitsberg et al. (1988a)
	Rainbow trout (LD50 - 3 ug/kg)	1 dose (i.p.) / + 80 days	125 25 5 1	95% dead (22 days) 100% dead (40 days) 80% dead (75 days) 8% dead (80 days)	Kleeman et al. (1986a)
	Carp (LD50 - 3 ug/kg)	1 dose (i.p.) / + 80 days	125 25 5 1	100% dead (45 days) 100% dead (55 days) 90% dead (75 days) 5% dead (80 days)	Kleeman et al. (1988)
	Bullhead (LD50 - 5 ug/kg)	1 dose (i.p.) / + 80 days	125 25 5 1	100% dead (22 days) 100% dead (22 days) 50% dead (65 days) 3% dead (80 days)	Kleeman et al. (1988)

Table 3-5. (continued)

Chemical form	Test species	Duration (days) Exposure	/ Observation	Concentration (ng/l)	Toxic effects	Reference
2,3,7,8-TCDD (continued)	Largemouth bass (LD_{50} - 11 ug/kg)	1 dose (i.p.)	/ + 80 days	125 25 5 1	100% dead (22 days) 95% dead (80 days) 5% dead (80 days) 5% dead (80 days)	Kleeman et al. (1988)
	Bluegill (LD_{50} - 16 ug/kg)	1 dose (i.p.)	/ + 80 days	125 25 5 1	100% dead (23 days) 80% dead (76 days) 2% dead (80 days) 5% dead (80 days)	Kleeman et al. (1988)
	Yellow perch (LD_{50} - 10 ug/kg)	1 dose (i.p.)	/ + 80 days	125 25 5 1	100% dead (35 days) 90% dead (40 days) 20% dead (60 days) 5% dead (80 days)	Kleeman et al. (1986b)
	Yellow perch (LC_{50} - 3 ug/kg)	1 dose (i.p.)	/ + 80 days	125 25 5 1	<95% dead (28 days) <95% dead (28 days) 80% dead (80 days) 0% dead (80 days)	Spitsberg et al. (1988b)
Pentachloro-						
1,2,3,4,7-PCDD	Rainbow trout (fry - 0.2 g)	5 (water)	/ + 24 days	16	No deaths	Muir et al. (1985)
	Fathead minnow (0.2 g)	5 (water)	/ + 24 days + 24 days	19 11	No deaths No deaths	Muir et al. (1985)
	Rainbow trout (0.5 - 1.0 g)	30 (diet)	/ + 30 days	105 ng/g	No significant (P = 0.05) adverse effects	Muir et al. (1988)
	Fathead minnow (1.0g)	30 (diet)	/ + 30 days	105 ng/g	No significant (P = 0.05) adverse effects	Muir et al. (1988)

Table 3-5. (continued)

Chemical form	Test species	Duration (days) Exposure	/ Observation	Concentration (ng/l)	Toxic effects	Reference
Hexachloro-						
1,2,3,4,7,8-HxCDD	Rainbow trout (fry - 0.2 g)	5 (water)	/ + 24 days	47	26% dead (12 days) and weight loss	Muir et al. (1985)
		5 (water)	/ + 48 days	10	No effects	
	Fathead minnow (0.2 g)	5 (water)	/ + 24 days	18	No deaths	Muir et al. (1985)
			/ + 48 days	7	No deaths	
	Rainbow trout (0.5 - 1.0 g)	30 (diet)	/ + 30 days	109 ng/g	Significant (P = 0.05) growth rate effects	Muir et al. (1988)
	Fathead minnow (1.0 g)	30 (diet)	/ + 30 days	109 ng/g	No significant (P = 0.05) adverse effects	Muir et al. (1988)
1,2,3,7,8,9-HxCDD	Rainbow trout (yolk sac fry)	4 (water)	/ + 10 days	Less than 10^{-5} times as toxic as 2,3,7,8-TCDD		Helder and Seinen (1986)
Heptachloro-						
1,2,3,4,6,7,8-HpCDD	Rainbow trout (fry - 0.2 g)	5 (water)	/ + 24 days	55	No deaths	Muir et al. (1985)
			/ + 48 days	11	No deaths	
	Fathead minnow (0.2 g)	5 (water)	/ + 24 days	39	No deaths	Muir et al. (1985)
			/ + 48 days	8	No deaths	
	Rainbow trout (0.5 - 1.0 g)	30 (diet)	/ + 30 days	109 ng/g	No significant (P = 0.05) adverse effects	Muir et al. (1988)
	Fathead minnow (1.0 g)	30 (diet)	/ + 30 days	109 ng/g	No significant (P = 0.05) adverse effects	Muir et al. (1988)

Table 3-5. (continued)

Chemical form	Test species	Duration (days) Exposure	/ Observation	Concentration (ng/l)	Toxic effects	Reference
Octachloro-	Rainbow trout (0.1 - 0.5 g)	5 (water)	/ + 18 days	415	No deaths	Muir et al. (1986)
			+ 32 days	20	No deaths	
	Fathead minnow (1.5 - 2.5 g)	5 (water)	/ + 48 days	9	No deaths	Muir et al. (1986)

Table 3-6. Aquatic Toxicity Data for Chlorinated Dibenzofurans

Chemical form	Test species	Duration (days) Exposure / Observation	Concentration (ng/l)	Toxic effects	Reference
Monochloro-	No data				
Dichloro-	No data				
Trichloro-	No data				
Tetrachloro-					
1,3,7,8-TCDF	Rainbow trout (yolk sac fry)	4 (water) / + 10 days	5.7×10^{-4} times as toxic as 2,3,7,8-TCDD		Helder and Seinen (1986)
2,3,6,8-TCDF	Rainbow trout (yolk sac fry)	4 (water) / + 10 days	9.5×10^{-5} times as toxic as 2,3,7,8-TCDD		
2,3,7,8-TCDF	Rainbow trout (fry - 0.38 g)	28 (water) / + 28 days	8.78	28% dead (28 days) 46% dead (56 days)	Mehrle et al. (1988)
			3.93	18% dead (28 days) 22% dead (56 days)	
			1.79	3% dead (28 days) 3% dead (56 days)	
			0.90	6% dead (28 days) 6% dead (56 days)	
			0.41	2% dead (28 days) 2% dead (56 days)	
			Controls	0% dead (56 days)	
	Rainbow trout	/ days (same effects at same conc. as TCDD)		Lethal edema	Helder (1980)
	Rainbow trout (yolk sac fry)	4 / + 10 days	8.9×10^{-2} times as toxic as 2,3,7,8-TCDD		Helder and Seinen (1986)

a combination of exposure routes, such as would be found in a natural ecosystems, especially since the low water solubility and high octanol/water partition coefficient of 2,3,7,8-TCDD suggest most of that which would be found in the aquatic ecosystem would be sorbed to or bioaccumulated in dietary sources.

The duration of an exposure is important when evaluating the inherent chronic toxicity of 2,3,7,8-TCDD. Toxic effects were reported even for the shortest 2,3,7,8-TCDD exposure period in water, 6 hours for rainbow trout, following which rainbow trout mortalities occurred between 78 and 139 days after the exposure (Branson et al. 1985). Adams et al. (1986) estimated that 90 percent of steady-state uptake of 2,3,7,8-TCDD in rainbow trout would take longer than 48 days. Consequently, an adequate chronic study would require that an exposure period be extended until the maximum level of 2,3,7,8-TCDD has been reached, plus sufficient additional time for effects to be produced. Review of the studies in Table 3-5 indicates that none of the studies with water exposures are of sufficient duration for steady-state concentration of 2,3,7,8-TCDD to have been achieved in fish tissues.

The longest exposure to 2,3,7,8-TCDD via water was reported to be 28 days, with an additional 28-days post-treatment for observation for rainbow trout fry (Mehrle et al. 1988). In that study, a NOEC was not determined. At the lowest test concentration (0.038 ng/l), 45 percent mortality occurred before the test ended.

The 28-day trout study (Mehrle et al. 1988) did not include deposition of 2,3,7,8-TCDD in the eggs by the female. Some evidence is available which suggests that for trout the most sensitive life stage for chlorinated chemicals occurs when the toxicant stored in the yolk is sorbed into the developing embryo at the end of the yolk sac stage. Similarly, Helder (1980) reported that following a 96-hour exposure to 2,3,7,8-TCDD, the highest mortality in freshly fertilized pike eggs occurred during the resorption of the yolk. Mortality reached almost 100 percent at 2,3,7,8-TCDD exposure concentration of 10 ng/l. The NOEC was reported as less than the lowest test concentration (0.1 ng/l).

Sublethal effects of PCDDs have been studied by several researchers, but it appears that none of the studies have been conducted for sufficient duration to determine the inherent toxicity of PCDDs. The effects of 2,3,7,8-TCDD on the development of medaka embryos have been studied by Wisk and Cooper (1986). It was found that 20 to 40 ug/l caused tube hearts, hemostasis, and liver necrosis. In another study (Helder and Seinen 1986), 2,3,7,8-TCDD and 2,3,7,8-TCDF produced lethal edematous conditions in rainbow trout. Similar effects of 2,3,7,8-TCDD were reported by Helder (1980) during early life stages of the pike (Esox lucius), in which the sublethal effects included: reduced size at hatch,

tail-first-hatching, alteration of blood vessel walls, pericardial edema, enlarged nuclei and degradation in hepatocytes, and severe generalized edemas preceeding death.

Sublethal effects on rainbow trout have been studied by Spitsberg et al. (1988a) following single intraperitoneal injections of either 1, 5, 25, or 125 ug 2,3,7,8-TCDD/kg. At both 25 and 125 ug/kg, mortality occurred before body weight loss could be detected. At 5 ug/kg, effects included reductions in activity, feeding, and growth. Moderate to marked leukopenia and thrombocytopenia were also found. The 2,3,7,8-TCDD-treated trout also suffered higher stress from handling than controls. Numerous types of lesions were reported in lymphomyeliod tissues of the thymus, spleen, and kidneys, as well as epithelial lesions of the stomach mucosa, liver, pancreas, gill, and skin. The types of lesions included the following: thymic involution; splenic lymphoid depletion; decreased kidney hematopoiesis; multifocal necrosis, atrophy, and hyperplasia of the stomach mucosa; mild to severe hepatocyte vacuolation and ballooning degeneration of the liver and hyperplasia of the bile-duct; vacuolation of pancreatic exocrine cells; mild fusion of gill lamellae; and necrosis of fin margins. Histologic changes in epithelial organs resembled the lesions found in early stage studies of rainbow trout exposed to water-borne 2,3,7,8-TCDD.

Sublethal effects in yellow perch following single intraperitoneal injections of either 1, 5, 25, or 125 ug 2,3,7,8-TCDD/kg have also been reported (Spitsberg et al. 1988b). Many effects were, in general, similar to those reported in rainbow trout. Yellow perch did not, however, show lethality resulting from handling as seen in rainbow trout. Lesions in the thymus, spleen, kidney, stomach, and skin are similar to the effects observed in rainbow trout. Cardiac lesions not seen in rainbow trout were found, including: necrosis of myocytes subjacent to the epicardial surface of the ventricle; fibrinous pericarditis; and hypertrophy and hyperplasis of the pericardial mesothelium. Many of the histologic lesions, including fibrinous pericarditis, have also been found in mammals, chickens, or turkeys treated with 2,3,7,8-TCDD.

Spitsberg et al. (1986) measured immune responses in rainbow trout at 14 to 30 days after single intraperitoneal injections of either 0.1, 1.0, or 10 ug/kg of 2,3,7,8-TCDD. Trout injected with 2,3,7,8-TCDD at either 0.1 or 1.0 ug/kg remained clinically normal. Trout treated with 10 ug/kg of 2,3,7,8-TCDD became hypophagic and exhibited fin necrosis, ascites, and suppression of hematopoiesis. Concanavalin A-induced blastogenesis of thymic and splenic lymphocytes were not significantly changed, however, suppression of the pokeweed mitogen-induced response of splenic lymphocytes occurred. No statistically significant alterations occurred in humoral immune responses, and phagocytic activity of peritoneal macrophages was not decreased. In rainbow trout, immunosuppression was

evident only at 2,3,7,8-TCDD doses approaching 20 ug/kg, which is the 80-day, single-dose, parenteral LD_{50} value.

Spitsberg et al. (1988c) found no statistically significant effects on rainbow trout mortality or mean time-until-death following a challenge with infectious haematopoietic necrosis virus (IHNV). Rainbow trout were injected intraperitoneally with single doses of either 0.01, 0.1, 1.0, or 10 ug/kg 2,3,7,8-TCDD. In virus-free trout, TCDD-induced effects first appeared four to five weeks after injection of 10 ug/kg. The effects included fin necrosis, as well as reduction in activity and food consumption. No deaths occurred in the virus-free rainbow trout during the five weeks after treatment.

Behavioral effects induced by 2,3,7,8-TCDD in rainbow trout have also been reported by Merhle et al. (1988). Rainbow trout were severely stressed at all test concentrations (i.e., 0.038 to 0.789 ng/l of 2,3,7,8-TCDD). Behavioral impairments increased over time and with increasing test concentration. The behavioral changes included: lethargic swimming, abnormal head-up swimming posture, feeding inhibition, and lack of response to external stimuli. All of these behaviors would increase the likelihood of predation in a natural ecosystem.

3.4.2 Aquatic Toxicity of PCDFs

The longest exposure to 2,3,7,8-TCDF via water was 28 days, with an additional 28-days post-treatment for observation of the rainbow trout fry (Table 3-6) (Merhle et al. 1988). The NOEC for 2,3,7,8-TCDF was reported to be 0.41 ng/l. It appears that 2,3,7,8-TCDF levels in the trout may have reached equilibrium at the higher concentration (3.93 ng/l), but it is not evident that equilibrium was achieved in 28 days at the lower concentration (0.41 ng/l). Consequently, it is uncertain whether the only study available concerning 2,3,7,8-TCDF was of sufficient duration to produce maximum toxic effects.

The behavioral effects induced by 2,3,7,8-TCDF were similar to those observed in 2,3,7,8-TCDD exposures; however, the observed responses were of lesser magnitude (Merhle et al. 1988).

3.4.3 Conclusions Concerning Aquatic Toxicity

A definitive NOEC has not been reported for 2,3,7,8-TCDD. Even the lowest test concentration (0.038 ng/l) produced 45 percent mortality in rainbow trout exposed to 2,3,7,8-TCDD for 28 days. The reported NOEC value for 2,3,7,8-TCDF is 0.41 ng/l, but this is also uncertain because of the limited duration of observation. The toxicity data available on 2,3,7,8-TCDD, and possibly 2,3,7,8-TCDF, do not adequately define the inherent toxicity of these substances for two reasons: (1) the exposure periods are of insufficient duration for a steady-state equilibrium to be

reached; and (2) the studies do not address toxic effects on developing embryos resulting from deposition of either 2,3,7,8-TCDD or 2,3,7,8-TCDF in the eggs by the female.

The most sensitive stage in the life of a rainbow trout for chlorinated chemicals appears to occur during resorption of the yolk sac in developing embryos. During this stage, the lipophilic substances (e.g., PCDDs and PCDFs) deposited in yolk by the female become more concentrated in the yolk on a per-unit-weight basis and they are resorbed. This occurs as the fry metabolizes and incorporates nutrients contained in the yolk. As the toxic substance is released during metabolism of the yolk, the amount of substance entering the blood stream reaches a higher concentration than would otherwise normally be found. It is at this stage of development of organisms that the greatest sensitivity has been observed for substances such as DDE in both birds and fish.

While the adult-to-fry test results will not be available for some time, an estimate is needed at this time to evaluate the aquatic toxicity risks. Because the duration of the available studies are too short and NOEC values have not been found, it is suggested that a larger-than-normal margin of safety should be used, but how large a margin of safety is uncertain. Margins of safety usually are 100 in the USEPA Offices of Water, Pesticide Programs, and Toxic Substances, and the Office of Water has used margins of safety as large as 1000. For the purpose of this assessment, it was determined that the margin of safety for 2,3,7,8-TCDD should be 1000, based on the level of lethality found at 0.038 ng/l and the short exposure period tested. Based on a margin of safety of 1000 and an NOEC for TCDD that is <0.038 ng/l, it was assumed for the purpose of this assessment that concentrations of 2,3,7,8-TCDD in water greater than 0.038 pg/l will exhibit toxic effects to some aquatic species. Applying this same approach to 2,3,7,8-TCDF, it was assumed that concentrations of 2,3,7,8-TCDF greater than 0.41 pg/l will exhibit toxic effects to some aquatic species.

3.5 Toxicity of PCDDs and PCDFs to Wildlife

The adverse effects to individual wildlife species from 2,3,7,8-TCDD have been documented in laboratory studies. Using the results of these studies to estimate effects on wild populations has some limitations. The route and medium of adminstration and the duration of exposure to 2,3,7,8-TCDD for laboratory animals usually will differ from that of wild animals. Using these studies to assess effects on wild species assumes that the wild species are as sensitive or more sensitive to 2,3,7,8-TCDD than the laboratory species. The methodologies for predicting the effects of chemicals on terrestrial wildlife populations and ecosystems, however, are still in development. Thus, in the absence of sophisticated predictive methods, measures of the effects of chemicals on reproduction are currently the most useful indicators of possible effects on the populations of the species in the wild.

The estimated exposures to each animal listed in Table 3-7 are compared to benchmark doses that have been identified as causing adverse effects in laboratory species. Exposure levels approaching or exceeding the selected benchmark suggest that the exposed animal is at risk for experiencing adverse effects. Where possible, doses observed to cause adverse reproductive effects were selected as benchmarks; the exposure of a number of individual members of a species to a dose exceeding such a reproductive effect benchmark may lead to adverse overall population effects.

For birds, the estimated daily dose to adult birds is compared to the dose that had no observable adverse effects level (the NOAEL) in laboratory experiments. The concentration in bird eggs is compared to the lowest observed adverse effects level (the LOAEL) in laboratory studies. For mammals, the dose is compared to the lowest observable dose to cause reproductive effects in laboratory animals. The studies upon which the LOAELS or NOAELS were based are described below. The discussion that follows also describes the adjustments made to these doses in order to compare them to estimated wildlife exposures.

3.5.1 Toxicity Assessment for Birds

2,3,7,8-TCDD was administered at 100 ng/kg body weight/day in a corn oil/acetone vehicle to 3-day old white leghorn chickens (Schwetz et al. 1973). This dose was administered for 21 days and produced no adverse effects. It was assumed that the 2,3,7,8-TCDD was 100 percent absorbed from the corn oil/acetone vehicle (see Appendix A.2). However, it is possible that the absorption of 2,3,7,8-TCDD from laboratory feed or food sources for wild animals would not be the same as the assumed 100 percent absorption of 2,3,7,8-TCDD from a corn oil/acetone vehicle. Accordingly, the estimated dose to wildlife species from the ingestion of prey items is adjusted by the percent of 2,3,7,8-TCDD assumed to be absorbed from the diet. Values for this percentage are found in a recent review of the literature performed by Boyer (see Appendix A.2). In addition, the laboratory dose must be converted to an equivalent dose over the length of time that wild species of birds are exposed to 2,3,7,8-TCDD.

3.5.2 Toxicity Assessment for Bird Eggs

Bird eggs can contain 2,3,7,8-TCDD transferred from the mother's body burden of 2,3,7,8-TCDD. Eggs are an important model to consider in determining a toxicity endpoint because of the sensitivity of eggs to 2,3,7,8-TCDD. Sullivan et al. (1987) concluded that the LOAEL for chicken embryos is 65 pg/g in the egg (65 ppt), based on a study that found a 2-fold increase in cardiovascular malformations in chicken embryos at an estimated egg concentration of 65 pg/g. Although effects were found at lower concentrations of 2,3,7,8-TCDD, the study concluded

Table 3-7. Summary of Studies on the Toxicity of 2,3,7,8-TCDD to Wildlife Mammals and Birds

Species	Exposure level	Symptoms/effects	Reference
Chickens	1 mg/kg/day	Positive chick edema (lesions)	Schwetz et al. (1973)
Eastern Bluebird	10,000 ppt	LOAEL for eggs.	Thiel et al. (188)
Chickens	10^{-10} M concentration of TCDD	60% reduction in tymphoid cell numbers.	Nikolaidis et al. (1988)
White Leghorn Chickens	30--65 ppt	LOAEL for eggs	Sullivan et al. (1987)
Northern Bobwhite	15 ug/kg body weight single oral dose	LD_{50}	Eisler (1986)
Ringed Turtle-Dove	810 ug/kg body weight single oral dose	LD_{50}	Eisler (1986)
Mallards	108 mg/kg body weight single oral dose	LD_{50}	Eisler (1986)
Rhesus Monkeys	.0017 ug/kg body weight daily (7-29 months)	Abortion and weight loss.	Eisler (1986)
Monkeys	.011 ug/kg/day (9.3 months)	Generalized toxicity: anemia, hair loss, death. 1 of 2 pregnancies aborted.	Kociba and Schwetz (1982)
	.0017 ug/kg/day (7-20 months)	Slight loss of weight and hair. 4 of 7 pregnancies aborted.	
	.24 ug/kg three times weekly for 3 weeks of early gestation	Severe toxicity (death). 2/2 pregnancies aborted.	Kociba and Schwetz (1982)
	.048 ug/kg three times weekly for 3 weeks of early gestation	Slight toxicity. 3/4 pregnancies aborted.	
	.0095 ug/kg three times weekly for 3 weeks of early gestation	No toxicity. 1/4 pregnancies aborted.	
	Control	3/11 pregnancies aborted.	

Table 3-7. (continued)

Species	Exposure level	Symptoms/effects	Reference
Bobwhite	167 ppt oral exposure in a feeding test for 5 days with total observation period of 8 day totals	LC_{50}	Kenaga and Norris (1983)
	3 ppt 18 wk/18 wk test 0.3 ppt 18 wk/18 wk test	No effect on reproduction.	
Mallard	278 ppt oral exposure in a feeding test for 5 days with total observation period of 8 days	LC_{10}	Kenaga and Norris (1983)
Turkey	>259 ppt 11 day/11 day test	"EC_0" (no effect level)	Kenaga and Norris (1983)
Chicken	>500 ng/kg/dy 21 day/21 day test >200 ng/kg/dy 21 day/21 day test >100 ng/kg/dy 21 day/21 day test	LC_{90} Reduced feeding and growth, "LC_0" (no mortality). "LC_0" (no mortality)	Kenaga and Norris (1983)

that the evidence for effects at these lower levels was inconclusive; thus, the 65 ppt value was used for comparison with predicted egg concentrations for wild species.

3.5.3 Toxicity Assessment for Wildlife Mammals

This analysis compares small mammals (i.e., mammals less than 1 kg) to the lowest observed adverse reproductive effect level in laboratory rats. Murray et al. (1979) administered rats at 100, 10, and 1 ng/kg/day through the diet, and studied the effects on subsequent generations. At the 10 ng/kg/day level, Murray et al. (1979) found decreased fertility in the f_1 and f_2 generations.

For larger mammals, the expected dose for wild species is compared to the lowest dose observed to produce adverse reproductive effects in rhesus monkeys. Schwetz et al. (1973) reported that rhesus monkeys were given 1.7 ng/kg body weight of 2,3,7,8-TCDD in the diet. Of the seven pregnancies which occurred, it was observed that four had been terminated because of chemical-induced abortions.

In these laboratory studies, it was assumed that absorption from a laboratory diet is similar to the absorption from a wild diet, and that these doses are directly comparable to the daily dose to wild species from the ingestion of prey items.

3.6 Analysis of Uncertainties

The data base available concerning toxicological information on 2,3,7,8-TCDD is not complete, less information is available on 2,3,7,8-TCDF, and there is practically no toxicological information on the other congeners. Thus, SARs must be relied on to evaluate the potential toxic effects and to develop I-TEFs. However, different animal species exhibit different types of adverse effects and at different levels of exposure to 2,3,7,8-TCDD, so predicting the exact type of adverse effect at given exposure level for another congener has a great deal of uncertainty.

Human health effects following exposure to 2,3,7,8-TCDD are not nearly so well defined as effects on laboratory animals. There is consensus concerning chloracne, but evidence regarding the association of 2,3,7,8-TCDD with human cancer is equivocal, as is the evidence concerning reproductive effects.

Information concerning adverse human health effects of PCDFs is available, but little specific information is available concerning 2,3,7,8-TCDF. More studies are needed on past poisoning incidents concerning the specific exposure chemicals and the follow-up health histories to develop epidemiological information concerning both 2,3,7,8-TCDF and 2,3,7,8-TCDD.

The cancer risk estimates developed by three U.S. regulatory agencies (USEPA, FDA, and CPSC) differ by as much as a factor of 10, because of differences in the models, details of extrapolation, and the selection of animal data utilized. The approaches are technically valid, but the fact the three agencies have each developed a different value may be the cause of some confusion among the users of this information.

Based on the available data, the most sensitive noncarcinogenic effects of 2,3,7,8-TCDD in animals are reproductive effects and developmental toxicity. There is uncertainty as to whether these endpoints are applicable to people of nonreproductive age or who are not reproducing for other reasons. In addition, the noncancer effects data for the other congeners are more limited, so sensitive endpoints and Reference Doses are apparently not available for the other PCDDs and PCDFs.

The TEF approach is used to estimate carcinogenic and noncarcinogenic effects of PCDD and PCDF mixtures in terms of an equivalent amount of 2,3,7,8-TCDD. Although a common set of TEF values was adopted in 1989 (I-TEFs/89), the approach remains interim in nature and will be revised as new data are developed or until replaced (i.e., should a more direct biological assay appear to be feasible in the future). In addition to an accurate assessment of the congeners present, the uncertainties relating to estimated intakes, bioavailability, interspecies extrapolation, safety factors or mathematical models, and possible antagonistic or synergistic interactions are likely to carry as much or more uncertainty than the TEF values themselves.

Studies are available concerning the effects of 2,3,7,8-TCDD on aquatic organisms, but most of the studies involved bioavailability tests or short-term exposure studies with long post-exposure observation periods. The available test data on 2,3,7,8-TCDD indicate that none of the studies are adequate to define an acceptable, no-observed effect concentration (NOEC). Only one study exposed a fish to TCDD in water for a reasonable duration, 28 days. Even then, the exposure duration was only about half of the 48 days estimated to be required to achieve 90 percent of the steady-state BCF level in that species of fish.

The reported results of typical acute toxicity tests are suspect because of the short observation periods. In addition, intermittent, sporadic releases may cause serious delayed effects.

In a risk assessment, the normal procedure is to identify the NOEC for the most sensitive toxicological endpoint and apply a safety margin (e.g., 100) to that NOEC. Unfortunately, the lowest tested concentration (0.038 ng/l) among all known 2,3,7,8-TCDD studies produced 45 percent mortality. Consequently, a definitive NOEC for 2,3,7,8-TCDD has not yet been determined. All studies which have been completed are of insufficient duration to determine the full extent of TCDD effects. For

purposes of this assessment, it was determined that the margin of safety for TCDD should be 1000. This factor is justified for use in this assessment because of the high level of lethality (found to be 45 percent at 0.038 ng/l, the lowest concentration tested), because of the exposure duration (which was too short for steady-state equilibrium), and because of the absence of exposure to pre-spawning adults and TCDD-deposition in egg yolk (which generally yields the most sensitivity life stage for chlorinated chemicals).

Sublethal effects have been documented for 2,3,7,8-TCDD, but none of the studies conducted have been of sufficient duration to determine the inherent toxicity of 2,3,7,8-TCDD. The most sensitive life stage appears to be during yolk-sac resorption in developing embryos, but there are no adult-to-fry test results available at present.

There are insufficient data on mixtures of PCDD/PCDFs to assess the effects from the many complex combinations of exposure concentrations and the various durations of exposure which occur in nature.

Laboratory studies of 2,3,7,8-TCDD to individual wildlife species are well-documented, but there are uncertainties in extrapolating these results to wild populations. The exposure route, medium of administration, and duration of exposure in the laboratory will be different from these experienced by wild animals.

Effects have been observed at very low levels, and the no observed effects levels reported are only for the specific endpoints evaluated in each study. In addition, the absorption from laboratory diet may be different from the absorption from natural diets, such as ingestion of prey items.

3.7 Conclusions

The most toxic congener among the PCDDs and PCDFs is 2,3,7,8-TCDD. Among the carcinogens that have been evaluated by EPA, it is the most potent animal carcinogen. Its reported effects in animal systems include carcinogenesis, cancer promotion, reduced fertility and postnatal survival, teratogenic effects, immunotoxic effects, thymic atrophy, liver damage, effects on the thyroid, and chloracne and other effects on the skin. There are marked variations among species in both the types of observed effects and the dose levels of 2,3,7,8-TCDD causing these effects. Higher doses of the congeners are required to elicit the same type of adverse effect.

There is consensus that 2,3,7,8-TCDD causes chloracne in humans, but the evidence concerning cancer and reproductive effects is equivocal. There is stronger evidence that 2,3,7,8-TCDD causes disturbances in lipid metabolism and increased frequency of gastric ulcers.

PCDF exposure in humans has been shown to cause skin eruptions similar to chloracne, skin pigmentation, and eye abnormalities, as well as some evidence concerning persistent respiratory symptoms and impairments of lipid metabolism, and immune function. Further analyses are needed for some preliminary reports of liver cancer deaths and lung cancers among males following PCDF exposure. Some of these effects are qualitatively similar to those observed in animals, supporting the use of animal data as a basis for hazard assessment for other members of these families of compounds.

The cancer risk estimate is represented as the upper bound slope factor, q_1^* (EPA and FDA) or as the maximum likelihood estimate of the extra risk, q_1 (CPSC); these factors have been estimated by the three U.S. regulatory agencies using somewhat different methods, with estimated values for 2,3,7,8-TCDD of 1.6×10^{-4} $(pg/kg/day)^{-1}$ for EPA, 6.7×10^{-5} $(pg/kg/day)^{-1}$ for CPSC, and 1.8×10^{-5} $(pg/kg/day)^{-1}$ for FDA. Based on these values, the estimated exposure (also called risk-specific dose), giving an upper bound increased lifetime cancer risk of one in a million, ranges from 0.006 pg/kg/day (EPA) to 0.06 pg/kg/day (FDA).

Concerning noncancer effects of 2,3,7,8-TCDD, EPA has calculated, for the purpose of this assessment, lifetime daily exposure to human populations likely to be without appreciable risk of deleterious effect, called a Reference Dose (RfD). Based on observed reproductive effects in animal testing studies and the lack of a NOAEL from this testing, and based on Uncertainty Factors to account for interspecies and intraspecies differences, an RfD of 1 pg/kg/day was calculated for 2,3,7,8-TCDD. Reproductive effects and developmental toxicity in animals are the most sensitive noncarcinogenic effects for 2,3,7,8-TCDD.

International Toxicity Equivalency Factors (I-TEFs) have been developed for 2,3,7,8-TCDD and the other PCDD and PCDF congeners. The doses necessary to elicit the toxic responses differ in each case, but the relative potencies of the different compounds (compared to 2,3,7,8-TCDD) are generally consistent from one endpoint to another. The I-TEFs are based on *in vivo* and *in vitro* test systems. Various systems had been developed by various scientists and regulatory agencies including international efforts. In 1989, a common set of TEFs were adopted (I-TEFs/89) to assess the risks of PCDDs and PCDFs, including mixtures of congeners, relative to an equivalent amount of 2,3,7,8-TCDD, as if the components of the mixtures were concentrations of 2,3,7,8-TCDD itself. There remain uncertainties in the TEF approach, however.

In aquatic toxicity studies, the most toxic congener of polychlorinated dibenzo-p-dioxins and dibenzofurans appears to be 2,3,7,8-TCDD, probably due to its tendency to be taken up more readily than other congeners. Dioxins appear to be more toxic to fish than to other aquatic species.

The mode of action of 2,3,7,8-TCDD is unknown, but it appears related to neural toxicity and may affect the immune system, like many other organochlorine chemicals. The pattern of TCDD-induced mortality typically occurs between 30 to 80 days after the initial exposure, even for exposure durations as short as 6 hours. Mortality in fish appears to be a function of both exposure duration and the test concentration.

None of the bioconcentration tests were of sufficient duration to achieve a steady-state bioconcentration factor (BCF). Thus, all the reported BCF values have been estimated using the uptake rate (k_1) and depuration rate (k_2) values.

Many sublethal effects have been reported for polychlorinated dioxins and furans. 2,3,7,8-TCDD elicits a broad range of toxic effects which include: reduced growth; weight loss; abnormal hatching; cellular alterations; numerous types of lesions in a broad spectra of organs; and behavioral effects on swimming, reduced feeding, and loss of response to external stimuli.

Continuous exposures to fish appear to produce toxic effects more rapidly than short-term exposure to the same test concentration. Based on the available data, the most sensitive fish life stage at present appears to be during yolk sac resorption in the developing embryo.

For wildlife species, the lowest NOEL for 2,3,7,8-TCDD exposure to birds is 100 ng/kg/day in white leghorn chickens over a 21-day exposure period. The LOAEL for chicken embryos is 65 pg/g (ppt) in the egg, but a NOEL value is apparently not available. For wildlife mammals, a NOEL of 10 ng/kg/day in the rat has been reported in a multi-generation effects study.

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4. ASSESSMENT OF RISKS TO WORKERS FROM EXPOSURE TO DIOXINS AND FURANS FROM MANUFACTURE, PROCESSING, AND COMMERCIAL USE OF PULP, PAPER, AND PAPER PRODUCTS AND FROM PROCESSING AND COMMERCIAL USE OF PULP AND PAPER MILL SLUDGE

4.1 Introduction

Workers may be exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF at various stages of the paper production process. Exposures may occur during the bleaching of wood pulp, and from contact with raw paper products (e.g., newsprint, paperboard, fibers), finished commercial paper products, and wastewater and sludge generated during the manufacture of paper products. Additional exposure may occur from the commercial use of pulp and paper mill sludge.

This chapter summarizes data presented in the following two reports prepared for EPA:

PEI (1990a). PEI Associates, Inc. Estimated worker exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF in the manufacture, processing, and commercial use of pulp, paper, and paper products. Washington, D.C.: USEPA, Office of Toxic Substances. Contract No. 68-D8-0112. March 1990.

PEI (1990b). PEI Associates, Inc. Estimated worker exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF from processing and commercial use of pulp and paper mill sludge. Washington, D.C.: USEPA, Office of Toxic Substances. Contract No. 68-D8-0112. April 12, 1990.

Section 4.2 summarizes data from the PEI (1990a) report, Section 4.3 summarizes the PEI (1990b) report, and Section 4.4 addresses the issue of uncertainties.

4.2 Worker Exposure to Dioxins in the Manufacture, Processing, and Commercial Use of Pulp, Paper, and Paper Products

This section provides an overview of worker exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF during the manufacture, processing, and commercial use of pulp, paper, and paper products. Section 4.2.1 characterizes the worker population for different operations involving the manufacture, processing, and commercial use of pulp, paper, and paper products. Section 4.2.2 discusses the potential for exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF in pulp and paper processes/operations; it also includes a description of the methodology used to estimate the levels of inhalation and dermal exposure for workers involved in different types of processes. Section 4.2.3 summarizes the estimated exposure, individual cancer risks, and population cancer risks.

4.2.1 Pulp and Paper Industry Workforce Characterization

In 1986, the paper and allied products industry employed over 674,000 people, of which approximately three-fourths were directly involved in pulp and paper production (OTA 1989). Table 4-1 summarizes the number of total employees and production workers in 1985 in the various paper and allied product categories. Both integrated and non-integrated paper mills are included in Table 4-1.

As Table 4-1 shows, over 75 percent of the employees in the industry are production workers. Not all of these production workers, however, are exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF; the exposure varies for different job categories. It is important to note that job categories and descriptions vary considerably throughout the industry depending on equipment layout, automation, and complexity. Pulp and papermaking mills generally operate 24 hours per day, 3 shifts per day, 7 days per week. Paper converting operations usually shut down at night and on weekends (Soklow 1984).

The workforce characterization in kraft pulp and paper mills has not been fully studied. A limited amount of information was obtained from such sources as EPA literature, NIOSH data bases, industry data, and various industrial contacts. Additional assumptions were needed to calculate the number of workers in each job category. It was assumed that the number of workers in kraft pulp mills was proportional to the ratio of the amount of bleached pulp to the total amount of pulp produced. Other assumptions are stated in the respective sections.

(1) Brown stock washing. In brown stock washing operations, the potential for 2,3,7,8-TCDD and 2,3,7,8-TCDF exposure exists only if paper machine white waters, which have been used in the processing of bleached pulp, are recycled. However, the recycling of white waters is a rare practice throughout the pulp and paper industry. The job categories which could have a potential for exposures for 2,3,7,8-TCDD and 2,3,7,8-TCDF are the wash and screen room operators, pulp testers, and utility employees.

The wash and screen room operators monitor and control operations of washer lines, screens, filtrates, and high-density storage tanks (Soklow 1984). The number of wash and screen operators in a plant may range from 0 to 4, averaging 1 to 2 per shift (NIOSH 1983).

Pulp testers are responsible for retrieving and analyzing production area samples. Samples are usually analyzed in laboratory areas located away from the production area such as a wash/screen control room (Soklow 1984). The number of testers per shift may range from 0 to 3, but is usually 1 (NIOSH 1983).

Table 4-1. Total Employees and Production Workers in the Entire Paper and Allied Products Industry, 1985[a]

Industry group	Total number of employees	Production workers
Pulp mills	16,000	12,000
Paper mills, except building paper	132,000	102,000
Paperboard mills	54,000	41,000
Miscellaneous converted paper products	211,000	161,000
Paperboard containers and boxes	188,000	143,000
Building paper and board mills	4,000	3,000
TOTAL	604,000	462,000

[a]USDOC (1987)

(2) Bleaching operations. In the bleaching operations, 2,3,7,8-TCDD and 2,3,7,8-TCDF are formed through the use of chlorine or chlorine derivatives, and thus are expected to be at its highest concentrations. Four major job activities in such operations include: (1) process monitoring and control; (2) manual operation, and adjustment of equipment, or inspection; (3) process quality control sampling and testing; and (4) housekeeping and spill cleanup. The total number of production workers per shift ranges from 2 to 5 (NIOSH 1983). The bleach plant population potentially exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF is expected to be 1,300 workers total, based on the assumption that there are three employees on each of the four shifts within the 104 mills.

(3) Papermaking and finishing. In this stage, additives such as coatings, colorings, or sizing agents can be mixed with the pulp stock. Job categories potentially exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF include wet-end additives/stock preparation operators, coating preparation operators, dry-end paper machine operators, off-machine coating operators, assistants, and utility employees.

Wet-end additives/stock preparation operators are responsible for operating and monitoring the equipment during the following types of activities: dry pulp dissolving; beating, refining, thickening, cleaning, and blending of pulps; preparing additives such as sizes, fillers, and covering agents; and mixing and blending of ingredients. Operators in the coating preparation category are responsible for the preparation of the coatings, unloading the raw materials, and monitoring the system (Soklow 1984).

Dry-end paper machine operators are responsible for web drying, on-machine coating and drying, reeling, on-machine calendering, slitting/rewinding, quality control testing, and packaging and shipping. Drying operations utilize hot air circulated under a hooded ventilation system for the final drying step (Soklow 1984). Off-machine coating operations include application of the coating formulation, paper drying, and rewinding of the paper into rolls. Operators are responsible for monitoring the process, checking paper quality, assisting the utility personnel with loading or unloading rolls of coated paper, and cleaning of the dryer during upsets (Soklow 1984).

The number of workers per shift in the papermaking operations will vary depending on (1) number and location of paper machines, coaters, and slitters/rewinders; (2) the type and complexity of wet-end operations; (3) the complexity, speed, and automation of papermaking equipment; and (4) the type, number, and complexity of the dry-end operations (NIOSH 1983).

The paper mill population potentially exposed to PCDDs and PCDFs is estimated to be 32,000 workers total. This is based on the assumption that there are two machines per facility, with 18 workers per each of the

four shifts working at each machine in the 221 facilities processing bleached kraft and sulfite pulp. The number of facilities processing bleached pulp was estimated to be proportional to the quantity of bleached pulp production.

(4) Pulp drying. Operators in the drying stage of pulp production are responsible for monitoring the equipment, weighing the pulp sheets, off-loading and stacking them for shipment. The utility operators clean up any spills and assist the operators in keeping the production area clean (McCubbin 1989). The number of pulp drying operators potentially exposed to PCDDs and PCDFs was estimated to be 240 people assuming four workers in each shift of the 15 mills that use pulp drying (USDOC 1988).

(5) Converting operations. Converting operations transform paper into end products such as paper towels, cardboard boxes, and typing paper. One hundred twenty-nine thousand paper converting workers are estimated to be potentially exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF based on Bureau of Census data (USDOC 1988) and the assumption that the number of workers is proportional to the amount of bleached paper produced.

(6) Nonwoven operations. Operators in the nonwoven industry are responsible for machine operation and monitoring the system, raw material handling, quality control testing, and general housekeeping. Typically, there are two to four operators per shift for each finished-product machine (Nonwovens 1989). No precise information was available on the total number of workers in the nonwoven industry; however, the number of workers has been estimated to be more than 15,000 workers (Cunningham 1990).

(7) Commercial users. Commercial users of paper products include almost all workers. Some occupational classifications where workers are exposed for large portions of their work day include lawyers, computer programmers, secretaries, accountants, librarians, teachers, architects, postal workers, printers, and other government workers. According to information from the Bureau of Census, over 50 million workers in the United States have occupations which involve handling bleached paper products.

Medical workers who use nonwoven products containing bleached pulp include doctors, nurses, dentists, and other workers involved in health maintenance and diagnosing. These workers may wear nonwoven garments or breathe through nonwoven face masks for several hours each day.

Table 4-2 summarizes the overall number of workers (by industry segment and job category) exposed to bleached pulp and paper products. The following section presents the levels of exposure associated with these various activities.

Table 4-2 Number of Workers in the Pulp and Pulp Products Job Categories

Job category	No. of workers
Pulp mill	
Pulp manufacture	1,300
- Bleach operators	434
- Pulp testers	433
- Utility operators	433
Pulp drying	240
- Operators	160
- Utility workers	80
Paper mill	
Paper and paperboard manufacture	32,000
- Wet-end operator	10,667
- Dry-end operator	12,445
- Utility operator	8,888
Converting operations	
Paper converting operations	68,000
- General worker	
Paperboard converting operations	61,000
Nonwovens production (pulp converting)	15,000
- General worker	
Commercial users	
Paper and paperboard	48,671,000
Nonwovens	2,895,000

4.2.2 Worker Exposure Estimating Methodologies

Although considerable data have been collected on concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF for pulp, sludge, and wastewaters in the pulp and paper industry, no inhalation or dermal exposure data for 2,3,7,8-TCDD and 2,3,7,8-TCDF are currently available. Furthermore, little information is available on the effectiveness of engineering controls or personal protective equipment in protecting workers in this industry. Therefore, modeling techniques were used to estimate worker exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF.

Pulp and paper mill workers may be exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF either through dermal contact with wet or dry pulp, paper, or bleaching filtrates, or through inhalation of volatilized 2,3,7,8-TCDD/2,3,7,8-TCDF or particulate containing these chemicals. The source of particulates is paper dust generated from paper cutting, rolling, or packaging operations.

The estimates of inhalation exposures due to volatilized 2,3,7,8-TCDD/2,3,7,8-TCDF are relatively low because of the low vapor pressures of these chemicals. In addition, TCDDs/TCDFs have a tendency to preferentially bind with organic matter. No attempt was made to compensate for 2,3,7,8-TCDD/2,3,7,8-TCDF affinity for organic materials.

(1) Pulp manufacturing. In pulp manufacturing operations, the potential for exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF exists primarily in the bleaching operations. In brownstock washing operations, exposure can occur if water used for these operations is recycled paper machine white water containing 2,3,7,8-TCDD and 2,3,7,8-TCDF formed during bleach operations. The most common wash method, however, is effluent from the filters and fresh hot water which are not likely to contain PCDDs, PCDFs, or their precursors. Worker activities in brownstock operations include servicing of the brownstock washers and screens, sample collection and testing, and general plant maintenance, which usually involves cleanup of spills. Brownstock washers are well ventilated, with exhausts through canopy hoods or full enclosures.

Most kraft pulping processes are highly automated and, consequently, the operators spend considerable portions of each shift inside control rooms (NIOSH 1983). In brownstock washing operations with no control room in the washer area, operators may spend essentially the entire shift in the production area, usually at operating consoles (Soklow 1984). Average times spent in the control rooms for workers involved in brownstocking were 65 percent, 75 percent, and 33 percent for the operators, testers, and utility employees, respectively (NIOSH 1983).

The number of bleach plant workers potentially exposed to dioxin and their job descriptions depend upon the process size, degree of automation, plant layout and equipment, and integration of bleach

chemical handling and preparation operations. Various bleaching process characteristics may inadvertently reduce the potential for dioxin exposure. Ventilation of chlorine and chlorine dioxide towers, washers, and washer filtrate tanks, with or without gas scrubbing, in bleaching operations may inadvertently reduce worker exposure and thereby control worker inhalation exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF. Chlorine alarms are installed in the pulp bleaching building and usually set to go off at 1 ppm, the current OSHA Short-Term Exposure Limit (STEL) (15 minutes maximum for chlorine). The revised OSHA PEL for chlorine is 0.5 ppm (8-hr TWA). Rinsing operations often occur in open washers. Workers can often reach into the wash stream to gather samples of bleached or partially bleached wet pulp. Sampling of the wet bleached pulp or spill cleanup provides potential for worker dermal exposure unless proper personal protective equipment is worn.

The pulp manufacturing population exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF is estimated to be 1,300 workers total. The following sections discuss the various modeling techniques and assumptions used to estimate dermal and inhalation exposures.

(a) **Inhalation exposure.** In the pulp mill, there are three job categories of workers (bleach plant operators, pulp testers, and utility operators) who are potentially exposed to PCDDs and PCDFs through inhalation. These workers spend approximately 75, 25, and 20 percent of their shifts in the control room.

In the manufacture of pulp, only vapors generated from volatilization of the PCDDs and PCDFs constitute a potential route for inhalation exposure. No pulp dust is generated at this point in the production process. Pulp dust may be generated during pulp drying operations, which are discussed in the next subsection. There are no existing data available to determine inhalation exposure to 2,3,7,8-TCDD/2,3,7,8-TCDF vapors during pulp manufacturing. In the absence of exposure monitoring data, worker exposure to 2,3,7,8-TCDD/2,3,7,8-TCDF was estimated using two different approaches (based on the nature of the worker activities). The first approach utilizes a mass balance model to estimate worker exposure for specific activities (e.g., for pulp testers during sampling). The second approach is applicable for workers in a general area (e.g., bleach plant operators, utility operators) and is based on estimating the maximum 2,3,7,8-TCDD/2,3,7,8-TCDF air concentration available for inhalation based on their partial pressures. These partial pressures are calculated by assuming that TCDD, TCDF, and pulp water solutions behave as ideal mixtures. Specific details of the approaches are discussed under each scenario for which worker exposures are estimated.

There are many mass balance models available for estimating worker exposure. The following equation, derived using one of the mass balance models (Clement 1982) in combination with an equation describing the

generation rate (Thibodeaux 1979), was used to estimate inhalation exposure concentrations to 2,3,7,8-TCDD/2,3,7,8-TCDF for pulp testers:

$$Cv = \frac{6.3 \times 10^{-5} \; K \; A \; Pa}{k \; Q \; T} \qquad (4\text{-}1)$$

where:

Cv = concentration of 2,3,7,8-TCDD/2,3,7,8-TCDF in the vapor, ppm
K = gas mass transfer coefficient, cm/sec
A = surface area, cm^2
Pa = partial pressure of a component, atm
k = mixing factor, unitless
T = temperature, K
Q = ventilation rate, ft^3/min

The partial pressures in Equation 4-1 are calculated based on Raoult's law. The mass transfer coefficients for 2,3,7,8-TCDD/2,3,7,8-TCDF are not available in the literature and are estimated by an equation presented in Clement (1982).

The approach for estimating worker inhalation exposure concentrations to 2,3,7,8-TCDD/2,3,7,8-TCDF around closed systems in the bleaching area was based on comparison with the OSHA PEL for another chemical (chlorine) handled in the same process area and a knowledge of the vapor pressures of chlorine and 2,3,7,8-TCDD/2,3,7,8-TCDF. The airborne concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF were estimated based on a comparison with the PEL for chlorine, since there are alarms in the bleach processing area for chlorine to limit workers' exposure to this chemical below its PEL. The use of a PEL to estimate inhalation exposures gives a maximum or reasonable worst case workplace concentration. The exposures for the bleach plant operator and utility operator were assumed to be comparable since their job duties require them to be in the bleaching area of the pulping process for a portion of the shift. Therefore, the same approach was used to estimate inhalation exposure for these job categories; and the empirical equation for this approach is presented in Equation 4-2:

$$Cv = Cvc \times \frac{Po}{Pc} \qquad (4\text{-}2)$$

where:

Cv = hourly concentration of 2,3,7,8-TCDD/2,3,7,8-TCDF, ppm
Cvc = 8-hour PEL for chlorine, ppm (0.5 ppm)
Po = vapor pressure of the pure component at 25°C, atm
Pc = vapor pressure of chlorine at 25°C, atm (7.9 atm).

The inhalation exposure from volatilization for all job categories is converted from ppm to mg/m^3 using the equation presented in Equation 4-3. This value is substituted into Equation 4-4 resulting in a daily inhalation exposure (Iv) in mg/day.

It is pointed out that all the calculated inhalation exposure levels from volatilization are biased high because no consideration is given to the 2,3,7,8-TCDD/2,3,7,8-TCDF binding with organic matter and the presence of other chemicals in the matrices that could interfere with the volatilization of the 2,3,7,8-TCDD/2,3,7,8-TCDF. No estimates could be either found in the literature or provided by contacts in the field which would allow for quantifying the impact of these interferences on volatilization.

$$Cm = Cv\ M/Vm \tag{4-3}$$

$$Iv = Cm \times 1.25\ m^3/h \times ED \tag{4-4}$$

where:

Cm = concentration of 2,3,7,8-TCDD/2,3,7,8-TCDF in the vapor, mg/m^3
M = molecular weight, g/mole
Vm = molar volume, liter/mole (24.45 liter/mole at T = 25°C and P = 760 mm Hg)
Iv = daily inhalation exposure from volatilization, mg/day
ED = exposure duration, h/day

The relative toxicity of 2,3,7,8-TCDF with respect to 2,3,7,8-TCDD can be determined by calculating toxicity equivalents (TEQ) for the daily exposure. In addition, the percent exposure due to 2,3,7,8-TCDD can also be calculated. Equation 4-5 presents the equation for calculating TEQ, while Equation 4-6 is used for calculating the percent of the exposure due to 2,3,7,8-TCDD. These equations are found in USEPA (1989b).

$$DTEQv = Iv_{TCDD} + 0.1\ Iv_{TCDF} \tag{4-5}$$

$$\%TCDDv = \frac{Iv_{TCDD}}{Iv_{TCDD} + Iv_{TCDF}} \times 100 \tag{4-6}$$

where:

DTEQv = daily toxicity equivalents from volatilization of 2,3,7,8-TCDD and 2,3,7,8-TCDF
IvTCDD = daily exposure to 2,3,7,8-TCDD from volatilization, mg/day
IvTCDF = daily exposure to 2,3,7,8-TCDF from volatilization, mg/day
%TCDDv = percent of the exposure from volatilization due to 2,3,7,8-TCDD, %

In addition to daily TEQ, the lifetime average daily TEQ (LTEQv) for workers was also calculated. This is presented in Equation 4-7.

$$LTEQv = DTEQv \times DY \times LF/(BW \times LE) \quad (4\text{-}7)$$

where:

LTEQv = lifetime average daily TEQ from volatilization, mg/day-kg
DTEQv = daily TEQ from volatilization, mg/day
DY = number of days per year exposed, day/year
LF = number of years of exposure per lifetime, years/lifetime
BW = average body weight for a worker, kg
LE = lifetime expectancy, days/lifetime

The number of years of exposure per lifetime (LF) was assumed to be 40 years and the lifetime expectancy (LE) was assumed to be 25,550 days (i.e., 70 years). The average body weight for male workers (BW) is 70 kg and a female worker is 58 kg (NCASI 1988a). It was assumed that the worker would be in the plant for 250 days per year.

(b) Dermal exposure. Dermal exposure levels to 2,3,7,8-TCDD and 2,3,7,8-TCDF were computed based on the assumption that workers do not wear any type of gloves that effectively limit exposure to PCDDs and PCDFs. However, in actual practice, some operators may wear chemical-resistant gloves during the handling of the pulp. Since the type of glove used, the extent of glove use, and the frequency of glove replacement could not be determined, consideration in the estimation of dermal exposure could not be given to the degree of protection provided by personal protective equipment.

There are a few different approaches available for estimating dermal exposure. The approach used was that agreed upon by EPA, the Food and Drug Administration, and the Consumer Product Safety Commission (Babich et al. 1989) for use in this project. This approach considers the partitioning of PCDD/PCDF from the appropriate matrix (e.g., soil, sludges, paper) to a liquid (i.e., water, skin oils, urine, blood) and percutaneous absorption of PCDDs and PCDFs from the liquid. In this reference, common assumptions for the assessment of dermal exposure are presented; however, equations for estimating dermal exposures were not provided. CPSC supplied three equations for estimating dermal exposure (CPSC 1989); these equations are for estimating dermal exposure to pulp, paper, and sludge/soil. The equation for handling wet pulp was selected and is presented in Equation 4-8.

$$DEW = DC\ (ppt) \times \rho\ (mg/cm^3) \times FT\ (cm) \times \frac{1}{K} \times AD\ (h^{-1}) \times S\ (cm^2) \times ED\ (h/day) \quad (4\text{-}8)$$

where:

DEW = dermal exposure from handling wet pulp, mg/day
DC = 2,3,7,8-TCDD.2,3,7,8-TCDF concentration in the wet pulp, ppt
ρ = density of the wet pulp, 1,000 mg/cm^3
FT = liquid film thickness, 0.025 cm
K = liquid equilibrium partition coefficient of 2,3,7,8- TCDD and 2,3,7,8-TCDF from water, unitless, 13,000 and 29,000, respectively
AD = absorption coefficient of TCDD/TCDF through the skin, 0.012 hr^{-1}
S = skin surface area, cm^2
ED = exposure duration, h/day.

Most of the variables in Equation 4-8 are constants except the skin surface area and exposure duration. Assumptions were made for these variables depending on the individual job category.

The concentrations reported in the 104 Mill Study for 2,3,7,8-TCDD and 2,3,7,8-TCDF were based on dry weight fraction; however, a wet weight fraction for 2,3,7,8-TCDD and 2,3,7,8-TCDF was needed for Equation 4-8. Based on an average pulp composition of 11 weight percent pulp (NIOSH 1983) and the assumption that the aqueous phase contains 2,3,7,8-TCDD/2,3,7,8-TCDF and water, the weight fraction in the aqueous phase is 0.12 times the weight fraction on a dry basis.

(2) Pulp drying.

(a) Inhalation exposure. Inhalation is one route of exposure to PCDDs and PCDFs in pulp-drying operations. During drying of the pulp, vapors are released into the air. It is estimated that the pulp-drying and utility operators may be exposed to PCDDs and PCDFs by inhalation for two hours during their shift because their job activities are conducted away from the drying machines for a majority of the shift. The method for estimating inhalation exposure from volatilization for this open operation is based on the maximum partial pressure of 2,3,7,8-TCDD and 2,3,7,8-TCDF computed using the ideal gas law. This is a worst-case approach. In this approach, it is assumed that 2,3,7,8-TCDD, 2,3,7,8-TCDF, pulp and water mixtures have two phases--an aqueous phase and a solid or pulp phase. Furthermore, TCDDs and TCDFs are assumed to reside only in the aqueous portion of the mixture. The aqueous phase is assumed to consist only of TCDDs, TCDFs, and water and is assumed to behave as an ideal solution and obey Raoult's law. It was assumed that TCDDs/TCDFs and water are removed from the pulp at rates that ensure that relative concentrations of these components in the ideal mixture remain constant throughout the drying phase. The ideal gas law equation is presented in Equation 4-9. This equation transforms into Equation 4-10 by solving the right-hand side of the equation for

moles per volume (m^3) and then multiplying both sides of the equation by the molecular weight to obtain the inhalation exposure concentration of 2,3,7,8-TCDD/2,3,7,8-TCDF.

$$P \times V = n \times R \times T \qquad (4\text{-}9)$$

$$Cm = M \times \frac{n}{V} = M \times \frac{P}{RT} \times 1 \times 10^6 \qquad (4\text{-}10)$$

where:

P = partial pressure of 2,3,7,8-TCDD/2,3,7,8-TCDF, atm
V = volume of the gas, m^3
n = number of moles, moles
R = ideal gas constant, atm. liter/mole•K (0.0821)
T = temperature of the gas, K
Cm = concentration of 2,3,7,8-TCDD/2,3,7,8-TCDF in the vapor, mg/m^3
M = molecular weight, g/mole.

The inhalation exposure concentration in mg/m^3 based on the ideal gas law is then substituted into Equation 4-4 resulting in a daily inhalation exposure (Iv) in mg/day.

The relative toxicity of 2,3,7,8-TCDF with respect to 2,3,7,8-TCDD can be determined by calculating TEQ values. The percent exposure due to 2,3,7,8-TCDD can also be calculated. Using Equations 4-5 and 4-6, these two variables are calculated.

(b) **Dermal exposure.** Workers in the pulp-drying operations are potentially exposed to PCDDs and PCDFs through dermal contact with the wet or dry pulp. Dermal exposure may occur during pulp sheet weighing when sheets are added or removed by hand to achieve a predetermined weight. Fork-truck drivers usually do not handle the dry pulp. Dermal exposure to the wet or dry pulp is possible for the utility workers if personal protective equipment is not worn.

Dermal exposures to PCDDs and PCDFs were based on the worst-case assumption that pulp-drying workers do not wear any type of gloves that effectively limits exposure to PCDDs and PCDFs. Pulp-drying operators are potentially exposed to PCDDs and PCDFs while handling the dry pulp sheets during weighting operations. This is similar to the handling of dry paper; therefore, the equation for handling paper was selected for the pulp-drying workers. Dermal exposure from handling dry paper or pulp is presented in Equation 4-11 (CPSC 1989).

$$DED\ (mg/day) = DC\ (ppt) \times PW\ (g) / PS\ (cm^2) \times R\ (h^{-1}) \times \%AD \times S\ (cm^2) \times ED\ (h/day) \qquad (4\text{-}11)$$

where:

DED = dermal exposure from handling dry material, mg/day
DC = 2,3,7,8-TCDD/2,3,7,8-TCDF concentration in the dry pulp, ppt
PW = weight of the dry pulp sheets, g
PS = surface area of the dry pulp sheets, cm^2
R = rate of transfer from pulp to the skin, h^{-1}
%AD = percent of 2,3,7,8-TCDD/2,3,7,8-TCDF available for dermal absorption; fractional value used in calculations
S = skin surface area, cm^2
ED = exposure duration, h/day.

Utility workers who handle the wet and dry pulp during cleanup of the production area were assumed to come into contact with the pulp two hours per day. The worst-case assumption that the workers do not wear any type of glove that effectively limits exposure to PCDDs and PCDFs was also used in this exposure assessment. The equation for handling wet pulp was selected for this operation since the workers have a higher potential for contacting wet pulp rather than dry pulp. This equation was presented previously in Equation 4-8.

Equations 4-5 and 4-6 are used to calculate the TEQ and the percent exposure due to 2,3,7,8-TCDD. In these two equations, the daily inhalation exposure from volatilization (Iv) is replaced with the daily dermal exposure (DEW or DED) from 2,3,7,8-TCDD and 2,3,7,8-TCDF.

(3) Papermaking. Papermaking operations include wet-end additives/stock preparations, coating preparation, paper machine wet-end, paper machine dry-end, and off-machine coating. There is potential for pulp exposure in the papermaking stage of pulp and papermaking operations. In this stage, bleached pulp containing PCDDs and PCDFs is processed, and it is assumed that the concentrations of PCDDs and PCDFs remain constant. PCDDs and PCDFs can also be introduced into this stage by the use of recycled white water from the paper machine to dilute the pulp slurry prior to its feeding into the wet end of the papermaking machine. The additional PCDD/PCDF contribution, however, should be small in comparison to the concentrations of PCDDs and PCDFs in the bleached pulp. In the papermaking and finishing operations, a potential for PCDD and PCDF exposure through dermal absorption exists if workers come in contact with either dioxin/furan-contaminated wet pulp which is to be fed into beaters or refining equipment or with the paper products.

The workers in dry-end operations are potentially exposed to PCDDs and PCDFs through dermal contact with paper products, inhalation of vapors during drying operations, and inhalation of paper dust during normal process operations. For a particular plant evaluated in a NIOSH study, engineering controls and work practices used to prevent or decrease the amount of dust inhaled included exhaust systems, the use of dust masks, and cleaning of equipment every shift to remove accumulated

paper dust. A NCASI study to determine the particulate size distribution of paper dust showed that total paper dust mass concentration levels ranged from 0.2 to 4.90 mg/m^3 during normal operations, and 31 to 109 mg/m^3 at the time of operation during blowdown (operations requiring approximately 15 minutes per shift for machinery cleaning) (NCASI 1988b).

(a) **Inhalation exposure**. Three job categories of workers in the paper mill (i.e., wet-end operator, dry-end operator, and utility operator) are potentially exposed to PCDDs and PCDFs.

There are two routes for inhalation exposure: (1) vapors from volatilization of PCDDs and PCDFs and (2) particulates (dust). The utility operator and dry-end operator may be exposed by both routes of inhalation exposure, while the wet-end operator is estimated to be exposed only from volatilization of PCDDs and PCDFs. It was estimated that wet-end operators, dry-end operators, and utility operators spend 2, 4, and 6 hours of their shifts, respectively, in areas of the plant in which there is a potential for inhalation exposure.

(i) Volatilization. The same calculation procedures previously employed to estimate inhalation exposure to PCDDs and PCDFs by vaporization (using Equations 4-4 and 4-10) for pulp drying workers was used to estimate inhalation exposure to paper mill workers.

(ii) Particulate matter. Equation 4-12 was used to calculate the inhalation exposure from PCDD and PCDF contained in particulate matter generated during paper mill operations. This equation is similar to Equation 4-4 except that the Cm in this equation is for the total particulate concentration rather than the 2,3,7,8-TCDD/2,3,7,8-TCDF concentration. In the absence of data on 2,3,7,8-TCDD/2,3,7,8-TCDF concentrations in the paper dust, the fraction of 2,3,7,8-TCDD/2,3,7,8-TCDF in the dry pulp was used to allocate the portion of the paper dust which is 2,3,7,8-TCDD/2,3,7,8-TCDF. The dry weight fraction of 2,3,7,8-TCDD/2,3,7,8-TCDF in the pulp, as reported in the 104-Mill Study, was used in the calculation because the paper dust that is emitted is dry.

$$Ip = Cm\ (mg/m^3) \times 1.25\ m^3/h \times ED\ (h/day) \times W \qquad (4\text{-}12)$$

where:

Ip = daily inhalation of particulate matter, mg/day
Cm = concentration of paper mill dust, mg/m^3 (with existing engineering controls)
ED = exposure duration, h/day
W = weight fraction 2,3,7,8-TCDD/2,3,7,8-TCDF in the paper dust.

Equations 4-5 and 4-6 are used to calculate the TEQ and the percent exposure due to 2,3,7,8-TCDD. In these two equations, the daily inhalation exposure from volatilization (TEQv) for 2,3,7,8-TCDD and 2,3,7,8-TCDF are replaced with the daily inhalation exposure from particulate matter (TEQp) for 2,3,7,8-TCDD and 2,3,7,8-TCDF.

(b) **Dermal exposure.** The same calculation procedures previously employed to estimate dermal exposure levels for the pulp mill operators and pulp-drying workers were used to estimate the dermal exposure levels for the paper mill operators.

The wet-end operator responsibilities include pulping, bleaching, beating, refining, application of additives, and blending proportioning. Dermal exposure duration was based on engineering judgment. The equation for handling wet material was selected for this operation, since the worker will be handling the wet end of the process line. This equation was presented in Equation 4-8.

The dry-end operator responsibilities include supercalendering, rewinding, slitting, cutting and sheeting, trimming, packaging, and shipping. Some shipping and packaging operations may be performed in a separate part of the same plant. Dermal exposure duration was based on engineering judgment. The equation for handling dry material was selected for this operation, since the worker handles dry paper. This equation was presented in Equation 4-11.

The utility operator responsibilities include machine cleaning (blowdown), and assisting dry-end and wet-end operators. Dermal exposure duration was based on engineering judgment. The equation for handling dry material was selected for this operation, since the worker has a higher potential for contacting dry paper than wet paper. This equation was presented in Equation 4-8.

(4) Paper converting. There is potential for PCDD and PCDF exposure in converting operations. The potential for dermal exposure arises from manual handling of dry bleached paper-based stock and finished product which contain PCDDs and PCDFs, whereas the potential for inhalation exposure arises from dry paper dusts created during the various converting operations. In the finishing stages, dermal contact may occur through arm and other skin surfaces during the following building operations: (1) building of the reel; (2) changing of the roll; and (3) trimming, cutting, transporting, wrapping, or packaging of the paper. For certain activities during converting operations (e.g., quality assurance), workers may also have skin contact with the final paper product.

Data from a NIOSH study of worker exposures to paper dust from the dry end of paper machines producing tissue paper, paper towels, and newsprint were used. The following is an estimation of inhalation exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF for pulp mill workers.

(a) **Inhalation exposure.** The various job categories in the converting operation include machine operators; 3rd, 4th, and 5th hands; back tenders; slitters; and cutters. In the paper converting industry, dust generated during cutting and trimming operations is the only potential route for inhalation exposure. All operators in the production area were assumed to be exposed to the paper dust for the entire 8 hours of their shift.

The same calculation procedure previously employed to estimate inhalation exposure from dust for the paper mill operators was used to estimate the inhalation exposure levels from dust for the paper converting workers. Values for the dust concentration were calculated for an 8-hour exposure period from concentrations given in the NIOSH study.

(b) **Dermal exposure.** Dermal exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF was based on the assumption that workers do not wear gloves that effectively limit exposure. The same calculation procedure as that used for the paper mill operators' dermal exposure was used to estimate the dermal exposure levels for the paper converting operator. Dermal exposure was based on engineering judgment. The equation for handling paper was selected for this operation since the worker will be handling dry paper rather than wet pulp. This equation was presented in Equation 4-11.

(5) <u>Nonwoven industry</u>. Workers in the nonwoven industry are potentially exposed to PCDDs and PCDFs through the handling of the pulp. Inhalation of dust may be possible during the machining of pulp; however, area dust samples for nonwoven operations are not available to identify any potential for dust inhalation.

Inhalation exposure to all workers in a nonwovens manufacturing facility may occur because of the mechanical processing of the dry pulp, which may create pulp dust in the workplace. Plants which produce personal and medical hygiene products such as diapers and surgical masks are required to follow Food and Drug Administration regulations on the amount of allowable area dust (Cunningham 1990). Typically, pulp dust resulting from the hammer mill is reclaimed through a vacuum screen formed by a vacuum filter. The filter has a 99+ percent efficiency and can filter particles as low as 1 to 1-1/2 microns. These filters recycle the pulp dust to the hammer mill (Lammers 1989). Dermal exposure may occur to the operator feeding the pulp sheet manually into the hammer mill, but in automated nonwoven facilities, the pulp is machine-fed into the hammer mill, and therefore there is no skin contact with the pulp. Non information was available on the number of manual versus automated plants (Cunningham 1990).

(a) **Inhalation exposure.** Inhalation exposure to PCDDs and PCDFs was based on the assumption that workers do not wear respiratory protection. The same calculation method as that used for paper converting operators was used to estimate the exposure levels for nonwoven workers. Exposure duration was assumed to be the entire 8-hour shift since no data were available.

(b) **Dermal exposure.** The same calculation method as that used for pulp mill operators' dermal exposure was used to estimate the exposure levels for the nonwoven workers. Since worker activity data were not available, it was assumed that the nonwoven worker would come into contact with the pulp 6 hours per day. Dermal exposure duration was based on engineering judgment, since no data were available.

(6) Commercial users. During the commercial use of paper products, workers may be exposed to PCDDs and PCDFs through dermal contact with bleached paper products. The skin surface area contacting the bleached paper product and the amount of contact time varies with each job category. Almost all workers contact paper at some point in the work day. A wide variety of worker categories including secretaries, librarians, teachers, and accountants use various types of paper products for a large portion of the work day. Since many of the variables for the calculation of dermal exposure are not known, some assumptions were made. The number of workers in certain job categories was obtained from the United States Census Bureau and was the most complete and recent data. The concentration of 2,3,7,8-TCDD and 2,3,7,8-TCDF was estimated to be that of the pulp concentration because the PCDD and PCDF levels in paper products measured in the NCASI study was not representative of the entire paper industry. For medical workers, the concentration of 2,3,7,8-TCDD and 2,3,7,8-TCDF was assumed to be half since it was assumed that the nonwoven garments these workers contact are 50 percent pulp/50 percent textile fibers. The surface areas contacted by nonwoven garments and masks were assumed to be half of the face, and the entire palm and finger surfaces. Nonwoven garments worn by the medical workers were assumed to be used as a covering over clothing or other cloth garments. No data were available for exposure frequency and duration except for clerical workers and managers. All other exposure durations were based on engineering judgment and general knowledge of job related tasks involving the handling of paper and nonwoven products. The skin contact area for workers who are required to handle sheets of paper was assumed to be 20 percent of the total of palm and finger surfaces of both hands. These values were derived from studies done by NCASI on typical commercial users of paper products. The equation for handling dry material was used for commercial users of paper since they handle the dry paper. This equation is presented in Equation 4-11.

Several of the job categories were combined because exposures were assumed to be similar. In Group 1, accountants, auditors, architects, librarians, archivists, and duplicating and mail/message distribution personnel were assumed to have similar exposures; Group 2 is made up of lawyers, judges, computer programmers and operators, record processors, management, and miscellaneous administrative support personnel. Secretaries, stenographers, and typists make up Group 3; Group 4 includes teachers and sales workers; and Group 5 includes medical workers.

4.2.3 Summary of Worker Inhalation and Dermal Exposure, Individual Cancer Risks, and Population Cancer Risks

Dioxins are formed during the production of bleached pulp, thereby resulting in a potential for worker exposure to these chemicals in the production, processing, and commercial use of pulp, paper, and paper products. Studies have shown that the use of chlorine and chlorine-based bleaching agents could result in the generation of dioxins such as 2,3,7,8-TCDD, 2,3,7,8-TCDF, and 1,2,7,8-TCDF. Concentrations of dioxin isomers have been detected in the bleached pulp and in the paper products themselves. Results from the 104 Mill Study showed that dioxin concentrations in bleached pulp ranged from 0.10 to 49 ppt for 2,3,7,8-TCDD and 0.25 to 2620 ppt for 2,3,7,8-TCDF.

Pulp and paper mill workers may be exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF primarily via two routes: 1) dermal contact with the bleaching wastewaters, bleached pulp or paper products; and 2) inhalation of paper dusts which are created during converting, rewinding, sizing, pulp-fluffing, cutting, or other operations. Inhalation of vapors is not a major route of exposure since 2,3,7,8-TCDD and 2,3,7,8-TCDF have a low vapor pressure and tend to bind with organic matter. The extent of dermal exposure to workers varies depending on the worker job category. Worker contact with the raw bleached pulp stock is minimal since many processes are automated. Brown stock washing operators, pulp testers, or utility employees can be exposed through dermal contact with either the brown stock washed with recycled paper machine white waters containing 2,3,7,8-TCDD and 2,3,7,8-TCDF formed during bleaching operations or directly with the recycled paper machine waters. Operators, tenders, helpers, and utility employees in pulp manufacturing plants are also potentially exposed through dermal contact with the bleached pulp and filtrate during bleaching operations. The potential for inhalation exposure during pulp manufacturing is low because the bleaching towers are often installed outdoors and the operations are well controlled. Exposure through dermal contact with the bleached pulp may also occur during papermaking and finishing operations. Specific worker job categories with the potential for exposure include operators, assistants, and utility employees. In the papermaking operations, the wet-end operators, assistants, and utility employees are potentially exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF through dermal contact with

pulp. The dry-end paper machine operators, assistants, and utility employees may be exposed through the inhalation of paper dust and dermal contact with the paper products. Both of these routes of exposure are also applicable to workers in converting operations.

Table 4-3 summarizes the individual risks and population risks for workers involved in pulp manufacture, pulp-drying, paper manufacture, paper converting, and nonwovens production.

4.3 Worker Exposure from Processing and Commercial Use of Pulp and Paper Mill Sludge

This section assesses worker exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF during the processing and commercial use of sludge generated from wastewater treatment operations in pulp and paper mills. Section 4.3.1 provides a discussion of industrial processes and operations that may result in exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF during the processing and commercial use of pulp and paper mill sludges. Section 4.3.2 presents the results of studies that have measured contaminant concentration in sludges. Section 4.3.3 provides a characterization of the worker population for different operations involving the processing and commercial use of pulp and paper mill sludge. Section 4.3.4 discusses the potential for dermal and inhalation exposure to dioxins. Section 4.3.5 provides a summary of the estimates of the levels of inhalation and dermal exposure for workers involved in different operations, individual cancer risks, and population cancer risks.

4.3.1 Sludge Formation, Processing, and Disposal Operations

This section describes sludge formation, processing, and disposal operations which may be employed at pulp and paper mills.

(1) Sludge formation and processing. Two kinds of sludge are generated by wastewater treatment at pulp and paper mills: primary and secondary sludge. Primary sludges consist of solids which are lost from the pulp and paper manufacturing process and are subsequently removed by primary clarification (Ledbetter 1976). These solids are composed of fibers, clay filler materials, coating clays, and other chemical additives (Kirk-Othmer 1981a). In addition to fibers, other organic components such as wood dust, fiber debris, starches, dextrine, resins, and protein may be present. Typical water contents of nondewatered primary sludges from pulp and paper manufacturing operations may range from 90 to 98 percent (Ledbetter 1976). Secondary sludges are largely biological in nature and are harder to handle and dewater (Kirk-Othmer 1981a). Secondary sludge water contents may range from 98 to 99.5 percent (Hammer 1975).

Table 4-3. Summary of Individual and Population Cancer Risks for Workers Involved in Manufacturing, Processing, and Commercial Usage of Pulp, Paper, and Paper Products

Job category	No. of workers	Exposure pathway[c]	Estimated risk[a]			
			Individual risk		Population risk[b] # cases/yr	
			Low	High	Low	High
Pulp manufacturing operations						
· Bleach plant operator	434	Inhalation-volatilization[f]	4×10^{-7} (0.08)	5×10^{-7} (0.08)	4×10^{-6}	5×10^{-6}
		Dermal[f]	2×10^{-13} (47)	5×10^{-10} (4)	3×10^{-12}	6×10^{-9}
· Pulp testers	433	Inhalation-volatilization[e]	1×10^{-19} (0.03)	2×10^{-15} (0.002)	1×10^{-18}	3×10^{-14}
		Dermal[i]	2×10^{-12} (47)	3×10^{-9} (4)	2×10^{-11}	4×10^{-8}
· Utility operator	433	Inhalation-volatilization[i]	1×10^{-6} (0.08)	1×10^{-6} (0.08)	1×10^{-5}	2×10^{-5}
		Dermal[i]	4×10^{-12} (47)	7×10^{-9} (4)	4×10^{-11}	7×10^{-8}
Pulp drying operations						
· Pulp drying operator	160	Inhalation-volatilization[f]	2×10^{-11} (0.03)	2×10^{-7} (0.002)	7×10^{-11}	9×10^{-7}
		Dermal[f]	9×10^{-10} (29)	3×10^{-6} (2)	4×10^{-9}	1×10^{-5}
· Pulp drying utility operator	80	Inhalation-volatilization[f]	2×10^{-11} (0.03)	2×10^{-7} (0.002)	3×10^{-11}	4×10^{-7}
		Dermal[f]	2×10^{-13} (29)	5×10^{-10} (2)	5×10^{-13}	1×10^{-9}
Paper manufacturing operations						
· Wet-end operator	10,667	Inhalation-volatilization[f]	2×10^{-11} (0.03)	2×10^{-7} (0.002)	5×10^{-9}	6×10^{-5}
		Dermal[f]	2×10^{-13} (47)	5×10^{-10} (4)	7×10^{-11}	1×10^{-7}

Table 4-3. (Continued)

Job category	No. of workers	Exposure pathway	Estimated risk[a] Individual risk Low	Individual risk High	Population risk[b] # cases/yr Low	Population risk[b] # cases/yr High
Paper manufacturing operations (continued)						
- Dry-end operator	12,445	Inhalation-volatilization[j]	3×10^{-11} (0.03)	4×10^{-7} (0.002)	1×10^{-8}	1×10^{-4}
		Inhalation-particulate matter[h]	2×10^{-10} (29)	1×10^{-5} (2)	6×10^{-8}	5×10^{-3}
		Dermal[h]	2×10^{-10} (29)	7×10^{-7} (2)	6×10^{-8}	2×10^{-4}
- Utility operator	8,888	Inhalation-volatilization[i]	5×10^{-11} (0.03)	7×10^{-7} (0.002)	1×10^{-8}	1×10^{-4}
		Inhalation-particulate matter[e]	8×10^{-9} (29)	8×10^{-5} (2)	2×10^{-6}	2×10^{-2}
		Dermal[i]	3×10^{-10} (29)	1×10^{-6} (2)	6×10^{-8}	2×10^{-4}
Paper converting operations						
- General worker	129,000	Inhalation-particulate matter[k]	4×10^{-10} (29)	5×10^{-5} (2)	1×10^{-6}	2×10^{-1}
		Dermal[k]	4×10^{-10} (29)	1×10^{-6} (2)	1×10^{-6}	4×10^{-3}
Nonwoven operations						
- General worker	15,000	Inhalation-particulate matter[k]	4×10^{-10} (29)	3×10^{-6} (2)	1×10^{-7}	1×10^{-3}
		Dermal[i]	8×10^{-13} (29)	3×10^{-9} (2)	3×10^{-10}	1×10^{-6}

Table 4-3. (Continued)

Job category	No. of workers	Exposure pathway	Estimated risk[a] Individual risk Low	Individual risk High	Population risk[b] # cases/yr Low	Population risk[b] # cases/yr High
Commercial users[d]						
Group 1	2,639,000	Dermal[i]	$7x10^{-11}$ (29)	$3x10^{-7}$ (2)	$5x10^{-6}$	$2x10^{-2}$
Group 2	26,933,000	Dermal[h]	$5x10^{-11}$ (29)	$2x10^{-7}$ (2)	$3x10^{-6}$	$1x10^{-2}$
Group 3	5,004,000	Dermal[g]	$2x10^{-11}$ (29)	$8x10^{-8}$ (2)	$1x10^{-6}$	$5x10^{-3}$
Group 4	14,095,000	Dermal[f]	$2x10^{-11}$ (29)	$8x10^{-8}$ (2)	$2x10^{-6}$	$6x10^{-3}$
Group 5	793,000	Dermal[f]	$3x10^{-10}$ (29)	$8x10^{-7}$ (2)	$2x10^{-5}$	$5x10^{-2}$

[a] Values in parentheses represent percent exposure to 2,3,7,8-TCDD; risk estimates are based on EPA's slope factor..
[b] Values represent cases per year.
[c] The frequency of exposure assumed to be 250 days per year.
[d] Group 1 includes accountants, auditors, architects, librarians, archivists, curators, and duplicating and mail/message distribution occupations.
Group 2 includes lawyers, judges, computer programmers, computer operators, records processing, management, miscellaneous administrative support occupations.
Group 3 includes secretaries, stenographers, and typists.
Group 4 includes teachers and sales representatives.
Group 5 includes medical workers who may come in contact with nonwoven products such as garments and masks.
[e] Duration of exposure assumed to be 1 hour per day.
[f] Duration of exposure assumed to be 2 hours per day.
[g] Duration of exposure assumed to be 3 hours per day.
[h] Duration of exposure assumed to be 4 hours per day.
[i] Duration of exposure assumed to be 6 hours per day.
[j] Duration of exposure assumed to be 7 hours per day.
[k] Duration of exposure assumed to be 8 hours per day.

Primary and secondary sludges must be processed for volume reduction prior to disposal. Processing techniques which involve sludge volume reduction include thickening, conditioning, and dewatering (Kirk-Othmer 1981a). Thickening is primarily accomplished using either gravity settling, which is applied to mixtures of primary and secondary sludges, or dissolved air flotation which is usually applied to secondary sludges only. Conditioning is performed to improve the sludge dewatering characteristics. Dewatering is accomplished using centrifugation, vacuum filtration, or pressure filtration.

No data are available in the literature to characterize the typical unit operations at the wastewater treatment facilities of pulp and paper mills. The vast majority employ primary treatment and some form of secondary treatment along with dewatering of sludges from these operations.

The mechanism by which contamination from 2,3,7,8-TCDD and 2,3,7,8-TCDF occurs in pulp and paper mill sludges is currently being investigated by the paper industry. 2,3,7,8-TCDD and 2,3,7,8-TCDF are known to be formed during chlorine or chlorine derivative bleaching operations employed in pulp mills, and may be present in bleach plant and paper machine wastewater. 2,3,7,8-TCDD and 2,3,7,8-TCDF have a strong affinity for organic matter, and binding with organic matter in primary and secondary sludge may occur (Olson 1988).

(2) Sludge disposal operations. Disposal methods for sludge from pulp and paper mills include landfilling, incineration, surface impoundments, land application, and distribution as a salable product. Landfilling is the predominant disposal operation of choice currently in the industry, although a number of mills are also using incineration and surface impoundments.

(a) Landfilling. Landfilling involves the disposal of sludge in company-owned, publicly-owned, or privately-owned landfills. In this disposal operation, sludge is almost universally dewatered to a solids content of 20 to 25 percent to reduce its volume and lower transportation costs (USEPA 1979). Typically, the dewatered sludge is transferred from the dewatering operations to storage or directly to heavy duty dump trucks. If storage operations are used, the dewatered sludge will be loaded into dump trucks by a front-end loader (Ledbetter 1976). The dewatered sludge is hauled to the landfill where the basic operations involve spreading, compacting, and covering the sludge with excavated soil daily (Hammer 1975).

(b) Incineration. Incineration is a two-step oxidation process involving drying of the sludge followed by combustion. This process may occur in separate pieces of equipment or successively in a single unit (USEPA 1979). Incineration converts the sludge into an inert ash which is handled in wet or dry form (Hammer 1975). In wet form, the

ash is slurried with scrubber water and is pumped to an ash lagoon. In dry form, the ash is conveyed mechanically by a bucket elevator to a storage hopper for discharge into a dump truck for eventual disposal in a landfill (Metcalf & Eddy 1979).

According to the 104-Mill Study data, all mills that practice sludge incineration burn the sludge in waste fuel- or combination-power boilers. Potential exposures and risks resulting from pulp and paper mill sludge incineration are addressed in Section 7 of this report.

(c) Land application. Land application involves disposal of sludge by surface spreading or subsurface injection, usually on agricultural lands. Dewatered sludge is usually applied to land by a conventional fertilizer or manure spreader. Liquid sludges are surface spread by tank truck or injected below the surface (Metcalf & Eddy 1979). Surface spreading may require movement and burying of the sludge below the soil by plows, graders, or bulldozers (USEPA 1979).

(d) Distribution of sludge as a salable product. Sludge is processed for distribution as a salable product by heat-drying or composting techniques. The sludge is usually dewatered prior to processing to reduce the water content (USEPA 1979). Heat-drying is employed to remove moisture from sludge so that it can be efficiently processed into fertilizer. Heat-drying is also employed prior to incineration to ensure proper combustion of the sludge. The drying of the sludge is necessary in fertilizer manufacturing to permit grinding of the dried sludge.

Composting is undertaken to biologically degrade the sludge into a stable end product which is used as a soil conditioner. The composting process involves three steps: (1) preparation of the wastes to be composted, (2) decomposition of the prepared wastes, and (3) preparation and marketing of the product.

4.3.2 Dioxins in Pulp and Paper Mill Sludges

Sampling of sludge from pulp and paper mills that produce and/or process bleached pulp and bleached pulp-based products has shown that 2,3,7,8-TCDD and 2,3,7,8-TCDF are predominantly present in primary and secondary sludges formed during wastewater treatment operations.

2,3,7,8-TCDD and 2,3,7,8-TCDF concentrations are much higher in secondary sludges as compared to primary and combined dewatered sludges. Combined dewatered sludges, which are a mixture of secondary and primary sludge, have higher 2,3,7,8-TCDD and 2,3,7,8-TCDF concentrations than primary sludges. Concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF may be higher in secondary sludges as compared to primary sludges because secondary solids are comprised of organic biological solids for which PCDDs and PCDFs have an affinity.

The concentrations of 2,3,7,8-TCDF are typically greater than those of 2,3,7,8-TCDD in such sludges. Median concentrations of 2,3,7,8-TCDD in combined dewatered sludges were 27.5 and 36.0 ppt for the 5-Mill Study and the 104-Mill Study, respectively, whereas the corresponding median concentrations of 2,3,7,8-TCDF were 359.5 and 149.0 ppt, respectively.

It must be noted, however, that concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF measured in the various sludges do not necessarily represent worker exposure to these compounds during sludge handling, processing, and disposal operations. Worker exposure depends on a variety of conditions (e.g., engineering controls, personal protective equipment, work practices) which are addressed in Section 4.3.4.

4.3.3 Sludge Handling/Disposal Workforce Characterization

No previous studies have been conducted specifically on the workforce population in sludge handling and disposal operations at pulp and paper mills (Fisher 1989). The workforce for these operations were estimated based on parallels with municipal sewage sludge handling and disposal operations. Many sources were used to estimate the number of workers. These sources included EPA literature, NIOSH data bases, industry data, and discussions with NCASI and the United Paperworkers International Union (UPIU).

(1) Sludge formation and processing operations. Three job categories of workers are postulated for the sludge formation and processing operations: waste treatment plant operators, sludge haulers, and front-end loader operators.

Sludge processing operations will involve preparation of the waste sludge for disposal. In most cases, the sludge is loaded into dump trucks by conveyors or a front-end loader and is hauled away for disposal. Typical activities of sludge haulers and front-end loader operators will center around their respective equipment. Front-end loader operators will operate, maintain, and clean their equipment, if necessary. The front-end loader operator drives the front-end loader and transfers sludge from storage piles into dump trucks. The sludge hauler drives the dump truck and may maintain and clean it if necessary. The sludge hauler will transport the sludge to the disposal site. The sludge hauler and front-end loader operator population exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF is estimated to be approximately 400 workers.

(2) Sludge disposal operations. The number of workers involved with sludge disposal operations depends on the specific disposal technique applied by the facility, the quantity of sludge disposed and the location of final disposal.

(a) **Landfilling operations.** Landfilling operations may involve pulp and paper mill employees if sludge is landfilled on company-owned land, or employees of waste disposal firms if the sludge is landfilled at a site not affiliated with the pulp and paper mill. Only one job category of workers is postulated for landfilling operations: equipment operators.

At the landfill site, the number of equipment operators depends on the landfill technique used and quantity of sludge to be disposed. The total pieces of equipment may range between one and five and may even be greater in the case of large quantities of sludge (USEPA 1979). The landfill equipment operator population exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF is estimated to be approximately 400 workers.

(b) **Incineration.** All of the mills involved in the 104-Mill Study which incinerate sludge do so on-site, and thus sludge incineration operations will involve only pulp and paper mill employees. Disposal of residual bottom ash and fly ash, however, will also involve landfilling personnel as previously described. Sludge is typically burned with wood waste and fuel in power boilers for energy recovery. Thus, incineration of sludge and subsequent disposal of bottom ash and fly ash will involve power plant operators and maintenance staff. The number of workers will depend on the size of the mill and the number of boilers operated.

(c) **Land application.** One job category of workers is postulated for land application operations: equipment operators. Sludge application will involve equipment operators to operate fertilizer spreaders, tank trucks, plows, bulldozers, or other equipment depending on the type of sludge application. The number of equipment operators will depend on the quantity of sludge to be disposed, the sludge application techniques, and whether one operator is exclusively dedicated to one piece of equipment. The land application equipment operator population exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF is estimated to be approximately 20 workers.

(d) **Distribution of sludge as a salable product.** Sludge is converted into a salable product by heat-drying or composting. Heat-drying will require skilled operators for the dryers (USEPA 1979). Other employees may be used to aid in conveying and bagging of the dried sludge prior to shipment to fertilizer manufacturers. The fertilizer manufacturing operations will employ various personnel in the processing of the dried sludge.

Composting operations may involve personnel not affiliated with the pulp and paper mill when composting is done off-site, as is generally the case. There may be three job categories of workers in composting operations: equipment operators, screen operators, and compost haulers. Equipment operators perform unloading and placement of the sludge into windrows or aerated piles. Equipment operators remove the compost from

the windrows and piles and load it into screens. They also load the finished compost into dump trucks for distribution. If the compost is screened, an equipment operator and screen operator are necessary (USEPA 1979). Other personnel may be required if the compost is ground, blended or further processed in some manner prior to distribution. The screen operator is responsible for proper maintenance and operation of the screen, e.g., unplugging the screen if the compost is too wet. The compost hauler transports the finished product to farms or other facilities which may further process the compost.

The equipment operator population exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF is estimated to be approximately 150 workers. The screen operator population exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF is estimated to be approximately 20 workers. The compost hauler population exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF is estimated to be approximately 50 workers.

4.3.4 Worker Exposure Estimating Methodologies

Although considerable data have been collected on concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF for pulp, sludge, and wastewaters in the pulp and paper industry, no inhalation or dermal exposure data for 2,3,7,8-TCDD and 2,3,7,8-TCDF are currently available. Furthermore, little information is available on the effectiveness of engineering controls or the use of personal protective equipment in this industry. Therefore, modeling techniques and assumptions were used to estimate worker exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF.

Workers processing and commercially using pulp and paper sludge may be exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF either through dermal contact with the sludge or through inhalation exposure of either volatilized 2,3,7,8-TCDD/2,3,7,8-TCDF or particulates containing these chemicals. The potential for volatilization exposure should be minimal because these chemicals have a low vapor pressure and have a tendency to bind with organic matter rather than volatilize freely. The inhalation exposure levels computed in this report provide an estimate of the quantities of 2,3,7,8-TCDD and 2,3,7,8-TCDF that would freely volatilize; the values hence represent worst-case estimates since the effects on volatilization of 2,3,7,8-TCDD and 2,3,7,8-TCDF due to binding with organic matter and from interferences due to other chemicals present in the sludge are not considered. There is some potential for inhalation exposure from particulates during sludge handling operations such as loading/unloading, spreading, compacting, plowing into soil, and composting. There is some potential for dermal exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF through handling of contaminated sludge, although this would be minimal because most operations are highly automated with generally little contact between the sludge and employees except in cases of emergency maintenance and cleanup. The modeling technique presented in this section for sludge handling and processing were also used for all other sludge disposal operations in the subsequent section.

(1) Sludge formation and processing. In sludge formation and processing, the potential for 2,3,7,8-TCDD and 2,3,7,8-TCDF exposure for waste treatment plant operators exists through dermal contact with sludge when taking samples and when performing maintenance. Dermal contact is otherwise limited by the high degree of automation of sludge processing operations. Contact with sludge can occur through cleanup of spills of wet sludge or dewatered sludge that has fallen off the conveyors. Cleanup is usually done with shovels, minimizing dermal contact. Repair of dewatering equipment, pumps, and conveyors may also result in dermal contact. Since the process is highly automated and cleanup is usually done with a shovel, dermal exposure should be minimal (Hawks 1989). Therefore, as a worst case, it was estimated that typical maintenance and cleaning activities may result in 2 hours of dermal contact with the sludge during a shift.

The potential for inhalation exposure to waste treatment operators exists through volatilization of 2,3,7,8-TCDD and 2,3,7,8-TCDF in the sludge and inhalation of particulate matter generated during sludge handling operations. Waste treatment plant operators may be exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF through volatilization when inspecting and/or repairing equipment, taking samples, and cleanup of spilled sludge. Waste treatment plant operators may also be exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF through the generation of particulate matter during sludge handling operations. The quantity of particulate matter generated is estimated to be minimal because of the high moisture content of the sludge (approximately 70 percent).

Sludge haulers and front-end loader operators may be exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF through handling and loading of the sludge. Exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF may occur through inhalation of particulate matter generated during sludge handling and loading, volitilization from the wet sludge, and dermal contact during maintenance activities.

(a) **Inhalation exposure.** In sludge handling/processing operations, there are three job categories of workers (waste treatment plant operators, sludge haulers and front-end loader operators) who are potentially exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF through inhalation of vapors and particulate matter containing dioxin and dibenzofuran.

(i) Volatilization. There are no existing data available to determine inhalation exposure from volatilization of 2,3,7,8-TCDD/2,3,7,8-TCDF during sludge handling/processing. On-site 2,3,7,8-TCDD/2,3,7,8-TCDF concentrations in air were estimated based on a "box model" approach, which was used to simulate the dilution impacts of the wind in an outdoor setting, by using Equation 4-13.

$$Cm = \frac{G}{(L)\ (MH)\ (V)} \times 1000 \qquad (4\text{-}13)$$

where:

Cm = 2,3,7,8-TCDD/2,3,7,8-TCDF vapor concentration in air, mg/m^3
G = generation rate of 2,3,7,8-TCDD/2,3,7,8-TCDF, g/sec
L = equivalent side length of the area source perpendicular to the direction of the winds, m
MH = mixing height of vapors before inhalation by an individual, m
V = average wind speed at the inhalation height, m/sec.

The average wind speed chosen was 2.2 m/s and the mixing height chosen was 1.5 m (USEPA 1988b). The "box model" approach used assumes that 2,3,7,8-TCDD/2,3,7,8-TCDF present at the site travels only a short distance before it is inhaled by the worker.

There are many models which can be used to estimate generation rates. The selected model is based on a set of equations presented in USEPA (1986), and Hwang and Falco (1986) as described in EPA (1988b). The daily inhalation exposure from volatilization is then calculated using Equation 4-14. It should be noted that the calculated inhalation exposure levels from volatilization are biased high because no percent reductions are incorporated in the calculations for 2,3,7,8-TCDD/2,3,7,8-TCDF binding with organic matter and the presence of other chemicals in the matrices that could interfere with the volatilization of the 2,3,7,8-TCDD/2,3,7,8-TCDF. No estimates could be found in the literature or provided by knowledgeable contacts in the field regarding the quantitative reductions in volatilization from such interferences.

$$Iv = Cm \times 1.25\ m^3/h \times ED \qquad (4\text{-}14)$$

where:

Cm = concentration of 2,3,7,8-TCDD/2,3,7,8-TCDF in the vapor, mg/m^3
Iv = daily inhalation exposure from volatilization, mg/day
ED = exposure duration, h/day

The relative toxicity of 2,3,7,8-TCDF with respect to 2,3,7,8-TCDD can be determined by calculating toxicity equivalents (TEQ). In addition, the percent exposure due to 2,3,7,8-TCDD can also be calculated. Equation 4-15 presents the equation for calculating TEQ while Equation 4-16 is used for calculating the percent of the exposure due to 2,3,7,8-TCDD. These equations are found in USEPA (1989b).

$$DTEQv = Iv_{TCDD} + 0.1\ Iv_{TCDF} \qquad (4\text{-}15)$$

$$\%TCDDv = \frac{Iv_{TCDD}}{Iv_{TCDD} + Iv_{TCDF}} \times 100 \qquad (4\text{-}16)$$

In addition to daily TEQ, the lifetime average daily TEQ (LTEQv) for workers was also calculated. This is presented in Equation 4-17:

$$LTEQv = DTEQv \times DY \times LF/(BW \times LE) \quad (4\text{-}17)$$

where:

LTEQv = lifetime average daily TEQ from volatilization, mg/day-kg
DTEQv = daily TEQ from volatilization, mg/day
DY = number of days per year exposed, day/year
LF = number of years of exposure per lifetime, years/lifetime
BW = average body weight for a worker, kg
LE = lifetime expectancy, days/lifetime

The number of years of exposure per lifetime (LF) was assumed to be 40 years and the lifetime expectancy (LE) was assumed to be 25,550 days (i.e., 70 years). The average body weight for male workers (BW) is 70 kg and a female worker is 58 kg (NCASI 1988c). It was assumed that the worker would be in the plant for 250 days per year.

(ii) Inhalation exposure from particulate matter. Equation 4-18 was used to calculate the inhalation exposure from 2,3,7,8-TCDD and 2,3,7,8-TCDF contained in particulate matter generated during sludge handling/processing operations. This equation is similar to Equation 4-14 except that Cm in this equation represents the total particulate concentration rather than the 2,3,7,8-TCDD and 2,3,7,8-TCDF concentration.

$$Ip = Cm\ (mg/m^3) \times 1.25\ m^3/h \times ED\ (h/day) \times WF \quad (4\text{-}18)$$

where:

Ip = daily inhalation from particulate matter, mg/day
Cm = concentration of particulate matter, mg/m^3
WF = weight fraction of 2,3,7,8-TCDD/2,3,7,8-TCDF in the dry sludge
ED = exposure duration, h/day.

The concentration of 2,3,7,8-TCDD/2,3,7,8-TCDF in the particulate matter was assumed to be equal to the concentration of 2,3,7,8-TCDD/2,3,7,8-TCDF in the sludge. The dry weight concentration of 2,3,7,8-TCDD/2,3,7,8-TCDF, as reported in the 104-Mill Study, was used in the calculations because the particulate matter which is emitted is assumed to be dry.

The concentration of particulate matter was calculated using Equation 4-19:

$$Cm = \frac{Qp}{(L)(MH)(V)} \quad (4\text{-}19)$$

where:

Cm = particulate concentration, mg/m^3
Qp = total particulate matter emission rate, mg/s
L = equivalent side length of the site perpendicular to the direction of the winds, m
MH = mixing height of the particulate matter before inhalation by an individual (assumed to be 1.5 m)
V = average wind speed at the inhalation height (assumed to be 2.2 m/s).

Total particulate emission rates for a given disposal scenario, which in this case is dewatered sludge loading and unloading, are used in the equation. The length of the area from which the particulate matter is being emitted corresponds to the length of an open area of the dump truck, length of daily landfill and land application areas, and length of the area of composting activities. Particulate matter emission rate for sludge handling were calculated using AP-42 emission factors. These AP-42 emission factors are widely used for estimating particulate emission rates for similar applications.

(b) Dermal exposure. Dermal exposure levels to 2,3,7,8-TCDD and 2,3,7,8-TCDF were computed based on the assumption that workers do not wear any types of gloves that are effective in limiting exposure to PCDDs and PCDFs. Dermal exposure levels for sludge haulers and front-end loader operators were assumed to be equal because of their similar job functions.

There are a few different approaches available for estimating dermal exposure. The approach selected was agreed upon by EPA, Federal Drug Administration, and the Consumer Product Safety Commission (Babich et al. 1989). This approach considers the partitioning of PCDD/PCDF from the appropriate matrix (e.g., soil, sludges, pulp, paper) to a liquid (i.e., water, skin oils, urine, blood) and percutaneous absorption of PCDDs and PCDFs from the liquid. In this reference, common assumptions for the assessment of dermal exposure are presented. However, in this reference, equations for estimating dermal exposures were not present. CPSC supplied three equations for estimating dermal exposure (CPSC 1989). These equations are for estimating dermal exposure to pulp, paper, and sludge/soil. The equation for handling wet sludge/soil was selected for the sludge haulers and front-end loader operator handling wet sludge and is presented in Equation 4-20.

$$DEW = DC\ (ppt) \times \rho\ (mg/cm^3) \times FT\ (cm) \times B \times AD\ (h^{-1}) \times S\ (cm^2) \times ED\ (hr/day) \qquad (4\text{-}20)$$

where:

DEW = dermal exposure from handling wet material, mg/day
DC = adjusted 2,3,7,8-TCDD/2,3,7,8-TCDF concentration to account for handling of wet sludge, ppt
ρ = density of the dewatered sludge, mg/cm^3
FT = liquid film thickness, cm
B = bioavailability factor for sludge, unitless
AD = absorption coefficient of TCDD/TCDF through the skin, h^{-1}
S = skin surface area, cm^2
ED = exposure duration, h/day.

No data were available on duration or extent of dermal exposure. Therefore, dermal exposure duration was based on engineering judgment. It was assumed that only the palms and fingers of both hands of the hauler/front-end loader operator would be in contact with the wet sludge, whereas it was assumed that both hands and forearms of the waste treatment plant operator would be in contact with the wet sludge. The density for dewatered sludge is 1058 mg/cm^3 (USEPA 1979) and a liquid film thickness for the wet sludge was estimated to be 0.025 cm. It was estimated that 2,3,7,8-TCDD was absorbed at an average rate of approximately 0.012 h^{-1} (AD) over the time period from 0.5 to 17 hours (Babich et al. 1989).

The relative toxicity of 2,3,7,8-TCDF in respect to 2,3,7,8-TCDD can be determined by calculating TEQ values. The percent exposure due to 2,3,7,8-TCDD can also be calculated. Equations 4-15 and 4-16 are used to calculate these two variables. In these two equations, the daily inhalation exposure from volatilization (Iv) for 2,3,7,8-TCDD and 2,3,7,8-TCDF are replaced with the daily dermal exposure (DEW) for 2,3,7,8-TCDD And 2,3,7,8-TCDF.

(2) Sludge disposal operations. The route and amount of exposures to 2,3,7,8-TCDD and 2,3,7,8-TCDF in sludge disposal operations will depend on the specific sludge disposal technique, the amount of sludge disposed, and the degree of processing of the sludge. In addition, atmospheric events such as wind and rain will affect the degree of exposure in sludge disposal operations conducted outdoors.

(a) Landfilling operations. Inhalation is the primary route of exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF in landfilling operations. During unloading, spreading, compacting, and burying of the sludge with various pieces of equipment, particulate matter is generated which may be inhaled by the equipment operators. In addition, 2,3,7,8-TCDD and 2,3,7,8-TCDF may volatilize from the sludge and be inhaled.

Dermal exposure to the sludge is minimal because workers are generally not in contact with the sludge unless maintenance of the equipment is required.

(b) **Land application.** Exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF through land application is very similar to that of landfilling operations. Inhalation is the primary route of exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF in land application operations. Particulate matter generated during unloading, spreading, and plowing operations may be inhaled by the equipment operators. In addition, 2,3,7,8-TCDD and 2,3,7,8-TCDF may volatilize from the sludge and consequently be inhaled.

Dermal exposure to the sludge is minimal because equipment operators are generally not in contact with the sludge unless maintenance of the equipment is required.

(c) **Distribution of sludge as a saleable product.** In heat-drying operations, which are used to dry the sludge so that it can be further processed into fertilizer, exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF may occur primarily through inhalation of particles entrained in the air during handling and conveying of the dried sludge to storage and distribution. Inhalation exposure will also occur in fertilizer manufacturing operations in which grinding, screening, and bagging of the dried sludge will produce particulate matter emissions. Dermal exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF may occur at the mills through cleanup and emergency maintenance, but is more likely in fertilizer manufacturing operations in which the sludge is much further processed. Dermal and inhalation exposures for this type of operation are not estimated because only one of the mills practices flash drying.

Equipment operators may be exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF by inhalation of particulate matter generated during composting operations. Particulate matter, which may be inhaled by the equipment operator, is generated during unloading of sludge, its placement in a windrow or pile, mixing bulking agents with sludge, turning and mixing of the compost piles, removal of compost from piles and unloading at the screening operations, loading compost into the screens, and loading screened compost into piles and eventually into trucks for distribution. In addition, the equipment operators may inhale 2,3,7,8-TCDD and 2,3,7,8-TCDF which has volatilized from the sludge.

Dermal exposure of equipment operators to the sludge/compost is minimal because operators are generally not in contact with the sludge/compost unless maintenance of the equipment is required.

The screen operators may be exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF via inhalation of particulate matter generated during screening of the compost. In addition, exposure may also occur from volatilization of 2,3,7,8-TCDD and 2,3,7,8-TCDF from the compost. Dermal exposure of screen operators to the compost occurs during routine maintenance of the screen, such as unplugging, which occurs when the compost is too wet, and cleaning of any spilled material.

Compost haulers may be exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF through loading and unloading of the compost from the dump truck. Exposure may occur through inhalation of particulate matter generated during compost loading and unloading, and from volatilization from the compost. Dermal exposure occurs during maintenance activities.

4.3.5 Summary of Worker Exposure, Individual Cancer Risks, and Population Cancer Risks from Processing and Commercial Use of Sludge

2,3,7,8-TCDD and 2,3,7,8-TCDF are present in primary and secondary sludges formed during wastewater treatment operations at pulp and paper mills, thereby resulting in a potential for worker exposure to these chemicals in the processing and commercial use of pulp and paper mill sludge. Two studies have quantified their presence in sludge: the 5-Mill and 104-Mill studies. Results from the 5-Mill Study showed that concentrations in primary, secondary, and combined dewatered sludge ranged from 17 to 710 ppt for 2,3,7,8-TCDD and 32, to 10,900 ppt for 2,3,7,8-TCDF, with the largest concentrations reported in secondary sludge samples. Results from the 104-Mill Study showed that concentrations corresponding to the low and high TEQ in combined dewatered sludges ranged from 0.7 to 1390 ppt for 2,3,7,8-TCDD and 3 to 17,100 ppt for 2,3,7,8-TCDF. It also showed that concentration in nondewatered sludges ranged from 6 to 4500 ppq for 2,3,7,8-TCDD and 6 to 14,000 ppq for 2,3,7,8-TCDF.

Workers involved in pulp and paper mill sludge processing and commercial use of the sludge be exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF via three major routes: (1) dermal contact with wet or dry sludge and compost during maintenance of equipment and performance of job functions; (2) inhalation of particulate matter generated by sludge and compost handling during sludge processing, landfilling, land application, and composting operations involving unloading, loading, spreading, burying, or screening of the materials; and (3) inhalation of 2,3,7,8-TCDD and 2,3,7,8-TCDF volatilized from the sludge and/or compost.

Estimated risks for workers exposed during the processing and commercial use of pulp and paper mill sludge are summarized in Table 4-4.

4.4 Analysis of Uncertainties

4.4.1 Worker Exposure from Manufacture, Processing, and Commercial Use of Pulp, Paper, and Paper Products

Considerable research has been conducted by EPA and API to understand the formation of and subsequently reduce the generation of dioxins in the pulp and paper industry. Additional information, however, is needed in the following areas: (1) potential for worker exposure in paper converting operations; (2) the frequency and duration of potential dermal

Table 4-4. Summary of Outer Bounds of Individual and Population Cancer Risks for Workers Involved in Processing and Commercial Usage of Pulp and Paper Mill Sludge

Job category	No. of workers	Exposure type[c]	Estimated risk[a]			
			Individual risk		Population risk[b] # cases/yr	
			Low	High	Low	High
Sludge handling/processing						
- waste treatment plant operators	1300	Inhalation-volatilization[f]	2×10^{-12} (0.6)	9×10^{-9} (0.2)	2×10^{-11}	9×10^{-8}
		Inhalation-particulate matter[d]	2×10^{-11} (19)	7×10^{-8} (8)	2×10^{-10}	7×10^{-7}
		Dermal[e]	1×10^{-7} (19)	4×10^{-4} (8)	1×10^{-6}	4×10^{-3}
- Sludge haulers/front-end loader operators	400	Inhalation-volatilization[f]	2×10^{-11} (0.6)	2×10^{-7} (0.2)	8×10^{-10}	5×10^{-6}
		Inhalation-particulate matter[f]	1×10^{-10} (19)	5×10^{-7} (8)	4×10^{-9}	2×10^{-5}
		Dermal[d]	6×10^{-9} (19)	3×10^{-5} (8)	2×10^{-7}	8×10^{-4}
Landfilling operations						
- Equipment operators	400	Inhalation-volatilization[g]	5×10^{-10} (0.6)	2×10^{-6} (0.2)	5×10^{-9}	2×10^{-5}
		Inhalation-particulate matter[g]	1×10^{-8} (19)	2×10^{-5} (6)	1×10^{-7}	2×10^{-4}
		Dermal[d]	6×10^{-9} (19)	1×10^{-5} (6)	6×10^{-8}	1×10^{-4}
Land application operations						
- Equipment operators	20	Inhalation-volatilization[g]	3×10^{-8} (0.6)	9×10^{-7} (1)	1×10^{-8}	4×10^{-7}
		Inhalation-particulate matter[g]	1×10^{-6} (19)	6×10^{-5} (35)	5×10^{-7}	3×10^{-5}
		Dermal[d]	1×10^{-7} (19)	7×10^{-6} (35)	5×10^{-8}	4×10^{-6}

Table 4-4. (Continued)

Job category	No. of workers	Exposure pathway[c]	Estimated risk[a] Individual risk Low	Individual risk High	Population risk[b] # cases/yr Low	Population risk[b] # cases/yr High
Composting operations						
· Equipment operators	150	Inhalation-volatilization[g]	$1x10^{-7}$ (0.2)	$6x10^{-6}$ (0.2)	$5x10^{-7}$	$2x10^{-5}$
		Inhalation-particulate matter[g]	$3x10^{-7}$ (8)	$1x10^{-5}$ (6)	$1x10^{-6}$	$4x10^{-5}$
		Dermal[d]	$4x10^{-9}$ (8)	$2x10^{-7}$ (6)	$2x10^{-8}$	$9x10^{-7}$
· Compost haulers	50	Inhalation-volatilization[f]	$3x10^{-10}$ (0.2)	$1x10^{-8}$ (0.2)	$4x10^{-10}$	$2x10^{-8}$
		Inhalation-particulate matter[f]	$3x10^{-8}$ (8)	$1x10^{-6}$ (6)	$4x10^{-8}$	$2x10^{-6}$
		Dermal[d]	$4x10^{-9}$ (8)	$2x10^{-7}$ (6)	$5x10^{-9}$	$3x10^{-7}$
· Screen operators	20	Inhalation-volatilization[g]	$1x10^{-9}$ (0.20)	$7x10^{-8}$ (0.2)	$7x10^{-10}$	$3x10^{-8}$
		Inhalation-particulate matter[g]	$2x10^{-6}$ (8)	$7x10^{-5}$ (6)	$9x10^{-7}$	$4x10^{-5}$
		Dermal[f]	$2x10^{-7}$ (8)	$8x10^{-6}$ (6)	$9x10^{-8}$	$4x10^{-6}$

[a] Values in parentheses are percent risk due to 2,3,7,8-TCDD; risk estimates are based on EPA's slope factor.
[b] Values represent cases per year.
[c] The frequency of exposure assumed to be 250 days per year.
[d] Duration of exposure assumed to be 1 hour per day.
[e] Duration of exposure assumed to be 2 hours per day.
[f] Duration of exposure assumed to be 4 hours per day.
[g] Duration of exposure assumed to be 8 hours per day.

and inhalation worker exposure to dioxins in the pulp and paper industry; (3) the extent of the use of personal protective equipment and engineering controls; and (4) the number of workers in job categories potentially exposed to dioxins. Additional information is also needed on the potential for exposure to pulp and paper workers during infrequent activities such as bi-yearly cleaning of grinding pit and paper roll residuals which may contain high dioxin concentrations.

There are some ongoing as well as planned studies which may clarify some of the uncertainties found in this report. A NIOSH study to characterize worker exposure to dioxins at a pulp/paper manufacturing plant is currently underway. The 104 Mill Study has been completed; however, the data that were collected need to be analyzed with respect to plant operating parameters such as production rate, type of wood used (e.g., softwood, hardwood), and quantity of bleaching chemical used. The 25 Bleach Line Study conducted by NCASI will provide dioxin concentration data for 25 bleach lines in 31 mills. Additional information will also be available soon on workforce characterization, converting operations, and production data from the American Paper Institute. That data should assist in filling some of the data gaps. Site visits to pulp manufacturing, papermaking, and paper converting operations would also provide additional insight on the potential for exposure to workers when performing different activities.

4.4.2 Worker Exposure from Sludge Processing and Commercial Use

No previous studies have been conducted regarding workforce characterization and worker exposure to PCDDs and PCDFs for pulp and paper mill sludge processing and commercial use. Information is needed in the following areas: (1) worker job category descriptions; (2) the number of workers in different job categories; (3) the potential for worker exposure in the various sludge processing and disposal operations; (4) the frequency and duration of potential dermal and inhalation exposure to PCDDs and PCDFs from pulp and paper mill sludge processing and use; and (5) the extent of use of personal protective equipment and engineering controls. This information is also needed for operations which may not be affiliated with pulp and paper mills such as composting operations and processing of dried sludge for fertilizer manufacture. Most of the estimates regarding types and numbers of workers and duration of exposure used in this report were based upon parallels with municipal sludge-handling operations and engineering judgment.

There are some ongoing studies which may clarify some of the uncertainties and fill some data gaps. The 104-Mill Study has been completed; however, the data that was collected needs to be analyzed with respect to plant operating parameters such as sludge and pulp production rates, type of wood used (e.g., softwood, hardwood), pulping technology used (e.g., Kraft, sulfite), and quantity of bleaching chemical used. The 25 Bleach Line Study by NCASI will provide TCDD and TCDF

concentration data for sludges in addition to effluent, bleach plant filtrates, and intermediate and final pulps. NCASI may be able to provide additional information, if any is available, regarding workforce characterization and worker exposure from sludge processing and commercial use which should assist in filling some data gaps. Site visits to pulp and paper manufacturing operations as well as to affiliated or nonaffiliated disposal or commercial sludge processing sites would also provide additional insight on the potential for exposure to workers when performing different activities.

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5. ASSESSMENT OF RISKS TO THE GENERAL POPULATION FROM EXPOSURE TO DIOXINS AND FURANS DURING USE AND DISPOSAL OF PULP AND PAPER MILL SLUDGE AND DISPOSAL OF PAPER WASTES

5.1 Introduction

Sludge from pulp and paper mills that use chlorine in the bleaching process contains measureable amounts of PCDDs and PCDFs as evidenced by data submitted pursuant to the 104-Mill Study (USEPA 1989a) (see Table 5-1). Other information submitted pursuant to the 104 Mill Study indicates that approximately 2.5 million metric tons of such sludge is generated annually. Most is landfilled (44 percent) or placed in surface impoundments (24 percent); the remainder is incinerated (12 percent), land-applied (12 percent), or distributed and marketed (8 percent). Each of these five use or disposal practices presents potential risks of human health impacts resulting from exposure to dioxins in the sludge.

Exposure to dioxins may also occur during the disposal of paper wastes. EPA estimates that about 45 million metric tons of pulp and paper products are disposed annually (USEPA 1988a). When discarded paper products containing PCDDs/PCDFs are buried in municipal landfills or burned in municipal incinerators, PCDDs and PCDFs may be released into the environment, resulting in potential exposure.

This chapter estimates human exposure and risks associated with the use and disposal of sludge from pulp and paper mills, as well as with the disposal of paper wastes in municipal landfills. Exposures and risks associated with the potential release into the environment of PCDDs/PCDFs contained in sludge during incineration are assessed in Section 7 of this report. The information presented here was compiled from:

> USEPA. 1990. U.S. Environmental Protection Agency. Assessment of risks from exposure of humans, terrestrial and avian wildlife, and aquatic life to dioxins and furans from disposal and use of sludge from bleached kraft and sulfite pulp and paper mills. Washington, DC: Office of Toxic Substances and Office of Solid Waste. EPA 560/5-90-013.

The following five waste disposal practices are addressed in this chapter:

- Landfilling of pulp and paper sludge (Section 5.2.1),
- Landfilling of paper wastes (Section 5.2.2),
- Surface impoundment of pulp and paper sludge (Section 5.2.3),

Table 5-1. Distribution of 2,3,7,8-TCDD and 2,3,7,8-TCDF Sludge Concentrations for All Plants in 104-Mill Study

Distribution descriptor	2,3,7,8-TCDD concentration (ng/kg or ppt)	2,3,7,8-TCDF concentration (ng/kg or ppt)
Number	79	79
Mean	162.9	885.4
Std. Dev.	464.7	2,303
Percentiles:		
100% (maximum)	3,800	17,100
99%	3,800	17,100
95%	680	2,940
90%	293	1,760
75%	119	799
50%	51	158
25%	12	34
10%	3	6
5%	1.9	2.4
1%	0	0
0% (minimum)	0	0

Source: USEPA (1990)

- Land application of pulp and paper sludge (Section 5.2.4), and
- Distribution and marketing of pulp and paper sludge (Section 5.2.5).

Exposure pathways evaluated for each sludge/waste use and disposal practice considered in this assessment are presented in Table 5-2. Section 5.3 provides a discussion on uncertainties inherent in the analyses described in Sections 5.2.1 through 5.2.5, and a summary of the risk estimates developed in this chapter is presented in Section 5.4.

Conclusions to this report are applicable only to pulp and paper mill sludges. At this time, the Agency has not assessed risks to human health and the environment from the use and disposal of sludges generated at publicly and privately owned treatment plants that treat domestic sewage. This evaluation with potential subsequent regulation of sewage sludges will be performed in the next two to three years in the second round of sewage sludge regulations under 40 CFR Part 503.

5.2 Estimates of Exposure and Risks to the General Populations from Disposal and Use of Sludge from the Pulp and Paper Industry and Disposal of Paper Products

In the assessment, exposures and risks to a hypothetical maximum exposed individual (MEI) as well as exposures and risks to an individual considered to be typical were addressed. The "typical" individual risk estimates were used in conjunction with estimates of potentially exposed populations to predict potential population risks.

Two approaches were used for estimating exposures and risks. These differ primarily in the selection of 2,3,7,8-TCDD/2,3,7,8-TCDF sludge concentration data used to estimate exposures and risks. One approach used the mean and highest concentration data reported in the 104-Mill Study (USEPA 1989a) for those mills employing the particular disposal practice being assessed (i.e., when assessing risks from sludge landfilling, the mean and highest concentrations reported for any of the mills using this disposal method were used). The other approach, hereinafter referred to as the "generic" approach, used values representative of concentrations in sludge from all mills, regardless of current disposal practices. By assuming a constant level of sludge contamination regardless of the method of disposal, pathways and disposal methods that are intrinsically more risky could be pointed out. For estimating typical exposure, this generic assessment assumed a 2,3,7,8-TCDD/2,3,7,8-TCDF concentration equal to the mean concentration reported over all plants in the 104-Mill Study. For the assessment of MEI exposure, the 90th percentile concentrations were used. The "generic" approach further did not divide the slope factor for dioxin by 0.55 which served to account for absorption in the animal studies which

Table 5-2. Exposure Pathways Evaluated for Each Pulp and Paper Mill Sludge Disposal or Use Practice

	Landfilling of sludge	Landfilling of paper	Surface impoundment of sludge	Land application of sludge	Distribution and marketing of sludge
Ingestion exposure					
From drinking contaminated ground water	•	•	•	•	
From drinking surface water contaminated by runoff	•		•	•	
From foods produced with contaminated soil				•	•
From consumption of fish caught in contaminated surface water	•		•	•	
From direct ingestion of contaminated soil				•	•
Inhalation exposure					
To volatilized contaminants	•	•	•	•	•
To particulate from contaminated soils				•	•
Dermal exposure					
From contact with contaminated soil				•	•

were used to derive the slope factor. For the purpose of this integrated report, results based on the generic approach used are presented.

Physical/chemical property and fate/transport information on 2,3,7,8-TCDD and 2,3,7,8-TCDF that were used in USEPA (1990) assessment are presented in Table 5-3..

5.2.1 Exposure and Risks from Disposal of Pulp and Paper Sludge in Landfills

Landfilling of sludge from the pulp and paper industry is defined as the burial of sludge on land, usually accompanied by the regular application of soil cover. Fifty-nine of the pulp and paper mills in the 104-Mill Study (USEPA 1989a) reported using landfilling to dispose of their sludge. Of these, at least 15 dispose of their sludge in municipal landfills. Because specific information at each site was not available, all landfill sites were assumed to be of similar size, to contain sludge with the same concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF, to use the same management practices, and to emit contaminants at similar rates.

Table 5-4 summarizes the characteristics of these hypothetical landfills and the landfilled sludge. The landfill design is intended to be representative of industrial landfills that receive pulp and paper sludge. For this analysis, it was assumed that risks from these hypothetical industrial landfills can be generalized to municipal landfills.

This analysis estimated exposures and risks to 2,3,7,8-TCDD and 2,3,7,8-TCDF through four exposure pathways associated with sludge landfills:

- Contaminants volatilize from the landfill and are transported by wind to neighboring areas. Humans inhale contaminated air and are exposed.

- Storm runoff carries contaminant-laden particles of soil from the surface of the landfill to nearby surface water bodies. Contaminants are then released from stream or lake sediments into surface water, which is withdrawn for drinking water supplies. Humans ingest the contaminated water and are exposed.

- Storm runoff carries 2,3,7,8-TCDD and 2,3,7,8-TCDF to surface water bodies, as described above. Fish accumulate the contaminants from the water or sediment. Humans ingest fish and are exposed.

- Rain water or sludge moisture carry dissolved contaminants from the bottom of the landfill to an aquifer underneath a landfill. Dissolved contaminants are then transported by the aquifer to

Table 5-3. Physical/Chemical Properties and Fate/Transport Assumptions: All Exposure Pathways

Input parameter	Estimate for typical individual	Estimate for MEI individual	Notes/explanation/(reference)
TCDD diffusivity in air (cm^2/sec)	0.05	0.05	Eduljee (1987); USEPA (1988b)
TCDD diffusivity in water (cm^2/sec)	5.5×10^{-6}	5.5×10^{-6}	Estimated w/Hayduk and Laudie method (Lyman et al. 1982).
TCDD solubility in water (ug/g)	0.02	0.02	TCDD concentrations do not reach the solubility limit in model simulations (see Section 2 of this report).
TCDD photolysis half-life (min)	4,320	4,320	(see Section 2 of this report).
TCDD molecular weight	322	322	
TCDD K_{oc} (cm^3/g)	1×10^{7}	1×10^{7}	(see Section 2 of this report).
TCDD Henry's Law constant (atm m^3/mol)	1.6×10^{-5}	1.6×10^{-5}	(see Section 2 of this report).
TCDF diffusivity in air (cm^2/sec)	0.05	0.05	Estimated w/Wilke and Lee method (Lyman et al. 1982).
TCDF diffusivity in water (cm^2/sec)	5.6×10^{-6}	5.6×10^{-6}	Estimated w/Hayduk and Laudie method (Lyman et al. 1982).
TCDF solubility in water (ug/g)	4.3	4.3	TCDF concentrations do not reach the solubility limit in model simulations (see Section 2 of this report).
TCDF photolysis	4,320	4,320	Assumed same as TCDD.
TCDF molecular weight	306	306	
TCDF K_{oc}	3.5×10^{4}	3.5×10^{4}	(see Section 2 of this report).
TCDF Henry's Law constant (atm m^3/mol)	8.6×10^{-5}	8.6×10^{-5}	(see Section 2 of this report).

Table 5-4. Assumptions and Parameter Values - Landfills: All Exposure Pathways

Input parameter	Estimate for typical individual	Estimate for MEI individual	Notes/explanation/(reference)
Area of landfill (cm^2)	1.2×10^9	2.4×10^9	30 acres is thought to be typical. 60 acres is assumed for high estimate (USEPA 1985, 1989a).
Quantity of sludge received (DMT/yr)	19,500	76,700	Average and highest values reported in 104 Mill Study (USEPA 1989a).
Landfill dimensions (m x m)	350 x 350	500 x 500	Assumes square site for 30 and 60 acre landfills.
Facility lifetime (yrs)	14	14	(USEPA 1985a).
Depth of landfill (m)	6	6	One landfill reported depth of 20 feet (USEPA 1989a). Assumed to be representative due to lack of additional information.
Site locations	Location of town containing each landfill.		
Depth of cover	0	0	(USEPA 1988b).
Fraction organic carbon in cover	1%	1%	(USEPA 1988b).
Fraction organic carbon in sludge	25%	25%	Median nitrogen content in combined sludges is 0.85%. Typical organic carbon-to- nitrogen ratios range from 17:1 to 45:1 (NCASI 1984). Thus, fraction of organic carbon ranges from 14 to 38% (midpoint of 25%).
Fraction organic carbon in landfill soil	20%	20%	Appropriate for sludge.
Soil porosity	0.25	0.25	Typical values.
Soil disconnectiveness index	3.7	3.7	Typical values.

Table 5-4. (continued)

Input parameter	Estimate for typical individual	Estimate for MEI individual	Notes/explanation/(reference)
Soil intrinsic permeability (cm^2)	1×10^{-8}	1×10^{-8}	Within range of permeabilities reported for "silty sand" and "clean sand" (Freeze and Cherry 1979).
True density of sludge/soil	2.65	2.65	Density of quartz.
Bulk density of soil (kg/m^3)	1,400	1,400	Typical value for sand (Yeh 1981).
TCDD concentration in sludge (ng/kg)	163	293	Average and 90th percentile reported values for all mills (USEPA 1989a).
TCDF concentration in sludge (ng/kg)	885	1,760	Average and 90th percentile reported values for all mills (USEPA 1989a).

nearby drinking water wells. Humans ingest contaminated water withdrawn from the wells and are exposed.

Each of these potential pathways of exposure is discussed below.

(1) Estimates of exposures and risks from inhalation of vapors. Estimating human exposure and risk through the volatilization pathway involved the three steps described below. Information about models used to estimate volatile emissions and a few key assumptions used are presented in Table 5-5.

(a) **Estimating volatile emissions.** Criteria for design and operating characteristics for industrial landfills are not currently available. Consequently, the extent to which daily and final cover are used at landfills is not known. For this analysis, it was assumed that a typical landfill applies no cover to its sludge. The Hwang and Falco (1986) equations were used to estimate the emission of dioxins and furans from the soil for both the MEI and typical exposure scenarios.

(b) **Estimating wind transport of volatile emissions.** This analysis used the Industrial Source Complex, Long Term (ISCLT) model (a Gaussian plume dispersion model) to estimate air concentrations of volatilized 2,3,7,8-TCDD and 2,3,7,8-TCDF to which populations downwind may be exposed. The ISCLT model estimated ambient air concentrations extending 50 km in all directions from the landfill sites. Site-specific meteorological data were used.

(c) **Methods for estimating human exposure and risk.** Individual exposure and cancer risk were calculated by:

$$E_D = \frac{(C_D)(I_H)(F_A)(L_F)}{(BW)(LE)} \qquad (5\text{-}1)$$

where:

BW = average body weight (assumed to be 70 kg)
C_D = estimated air concentration at distance D (pg/m^3)
E_D = average lifetime individual dose for person residing at distance D (pg/kg/day)
F_A = fraction of contaminant absorbed from inhaled air by humans (unitless) (assumed to be 1)
I_H = volume of air inhaled daily (assumed to be 23 m^3/day)
LE = life expectancy in days/lifetime (assumed to be 70 years)
L_F = number of days of exposure in a lifetime (assumed to be 70 years x 365 days/year)

and:

Table 5-5. Assumptions and Parameter Values - Landfills: Volatilization Pathway

Input parameter	Estimate for typical individual	Estimate for MEI individual	Notes/explanation/(reference)
Model used to estimate emissions	USEPA (1988b)	USEPA (1988b)	Hwang and Falco (1986) and USEPA (1986) in USEPA (1986b)
Model used to estimate wind transport, air concentrations, and human exposure	Industrial Source Complex, Long Term (ISCLT) model (area source)	ISCLT	Location - specific modeling for each site (Bowers et al.1980).
Metereological data for ISCLT	Stability array (STAR) summary data accessed through GEMS (site specific)		
Emission period (yrs.)	70	70	Corresponds to length of exposure for cancer risk calculations.
Landfill status at beginning of emission period	Full	Full	Will tend to overestimate emissions.

$$I_{RD} = (E_D)(q_1^*) \quad (5\text{-}2)$$

where:

I_{RD} = Lifetime individual cancer risk for person residing at distance D
q_1^* = Cancer potency for 2,3,7,8-TCDD or 2,3,7,8-TCDF $(pg/kg/day)^{-1}$

ISCLT also provided estimates of population-weighted average concentrations of the 2,3,7,8-TCDD or 2,3,7,8-TCDF in ambient air surrounding all landfill facilities. Aggregate cancer risks were calculated with the following expression:

$$E_{AVE} = \frac{(C_{AVE})(I_H)(F_A)(L_F)}{(BW)(LE)} \quad (5\text{-}3)$$

where:

C_{AVE} = average air concentration of contaminant, computed by weighting each level of contaminant concentration by the number of persons exposed to that level (pg/m^3)
E_{AVE} = population-weighted average exposure for all persons living within 50 km of a pulp and paper sludge landfill (pg/kg/day)

and:

$$R_T = [(Eave)(q_1^*)(POP)]/LE \quad (5\text{-}4)$$

where:

POP = Total exposed population
R_T = Aggregate cancer risk for exposed population (incremental cancer cases/year)

(d) Estimates of populations exposed. The ambient air concentrations estimated by ISCLT were overlayed onto actual human populations within 50 km of the sites examined. Population data were drawn from the 1980 Census, mapped to the level of Census block group and enumeration district.

(2) Estimates of exposures and risks from ingestion of ground water. For lack of sufficient empirical data, this analysis relied on mathematical models to estimate the extent of possible human exposure and risk. Table 5-6 lists key assumptions and input parameters used to estimate potential ground-water contamination. The estimation of exposure and risk involved three steps as described below.

Table 5-6. Assumptions and Parameter Values - Landfills: Ground-Water Pathway

Input parameter	Estimate for typical individual	Estimate for MEI individual	Notes/explanation/(reference)
Model used to estimate loading to aquifer	SESOIL	SESOIL	(Bonanzountas and Wagner 1984).
Result used for exposure estimates	Steady state	Steady state	
Model used for transport through saturated zone	AT123D	AT123D	(Yeh 1981).
Result used for exposure estimates	Steady state	Steady state	
Distance to aquifer (m)	3	3	Assumes 6 meter depth to water table. For best estimate scenario, sludge layer extends to 3m below landfill surface, but contaminants and organic carbon are distributed throughout 6m landfill depth (DRASTIC data base).
Aquifer medium	Sand and gravel	Sand and gravel	Most common aquifer medium reported in DRASTIC data base for counties containing landfills.
Depth of aquifer (m)	100	100	
Width of aquifer (m)	infinite	infinite	
Distance to model wells (m)	200 1,200 3,000	200	Midpoints of ranges listed below.
Distances represented	0-400 400-2,000 2,000-4,000	200	

Table 5-6. (continued)

Input parameter	Estimate for typical individual	Estimate for MEI individual	Notes/explanation/(reference)
Exposed populations			Assumes density of 68 persons per square mile.
0-400 m	195		
400-2000 m	4,675		
2000-4000 m	14,600		
Decay	0	0	No decay rate available for TCDD or TCDF in groundwater.
Effective porosity of aquifer medium	0.25	0.25	Appropriate for sand and gravel (Freeze and Cherry 1979).
Hydraulic conductivity (m/hr)	10	10	Appropriate for productive sand and gravel aquifer (Freeze and Cherry 1979).
Hydraulic gradient	1%	1%	Typical value.
Longitudinal dispersivity (m)	20	20	Within range of "typical" values reported for sand or silt. Values equal approximately 1/10 distance to receptor well (Yeh 1981).
Transverse dispersivity (m)	7	7	Within range of "typical" values reported for sand or silt. 1/20 and 1/10 of longitudinal dispersivity (Yeh 1981).
Vertical dispersivity (m)	1	1	Within range of "typical" values reported for sand.
TCDD K_D in aquifer	10,000	10,000	Does not affect steady state results.
TCDF K_D in aquifer	35	35	Does not affect steady state results.
Annual recharge rate (cm)	43	43	Based on meteorological data for a landfill site in Wisconsin.

(a) **Estimating contaminant loadings to ground water.** The SESOIL model was used to simulate contaminant transport through soil layers. The model considered monthly climate data, and maintained a mass balance for contaminant transport through multiple soil layers. Estimates of potential human exposure and risks were derived once steady state conditions had been reached in the unsaturated zone. The simulation assumed that the upper three meters of the landfill contain pure sludge, and the lower three meters contain soil into which 2,3,7,8-TCDD, 2,3,7,8-TCDF, and organic carbon from the sludge have migrated. At steady state, the top three meters were assumed to contain 20 percent organic carbon; the lower three meters contain 10 percent.

(b) **Use of AT123D to predict contaminant transport through the aquifer.** From the estimated loadings to ground water, the AT123D model (Analytical Transport One-, Two-, and Three-Dimensional Model) was used to predict contaminant concentrations at wells downgradient of the site. Exposure estimates in this analysis were based on "steady state" results from AT123D.

(c) **Estimates of exposures and risks.** Individual exposure and cancer risk were calculated by:

$$E_D = (W)(F_A)(C_D)(L_F)/(BW)(L_E) \qquad (5\text{-}5)$$

where:

W = amount of water consumed daily (assumed to be 2 liters)
F_A = fraction of contaminant absorbed from ingested water (unitless) (assumed to be 1)
E_D = average lifetime individual dose (pg/kg/day)
C_D = estimated water concentration (pg/l)
BW = average body weight (assumed to be 70 kg)
L_E = life expectancy in days/lifetime (assumed to be 70 years)
L_F = number of days of exposure in a lifetime (assumed to be 70 years/lifetime)

and:

$$I_{RD} = (E_D)(q_1^*) \qquad (5\text{-}6)$$

where:

I_{RD} = lifetime individual cancer risk
q_1^* = cancer potency for 2,3,7,8-TCDD or 2,3,7,8-TCDF $(pg/kg/day)^{-1}$

Exposure and risk were estimated separately for persons taking drinking water at each of the three model distances from a surface landfill site.

Maximum exposure and risk were assumed to occur at the nearest well location.

(d) **Estimates of populations exposed.** It was assumed that the density of persons drinking groundwater is constant near each landfill, and that this density corresponds to the average for the United States as a whole, or approximately 68 persons per square mile (0.26 per hectare) (USDOC 1987). This value is used to estimate population sizes in Table 5-6.

(3) Estimates of exposure and risks from ingestion of drinking water from surface water sources. Where pulp and paper sludge is deposited in uncovered landfills, particles of sludge or soil from the landfill surface can be transported by erosion to nearby lakes or streams. If humans consume water from these lakes or streams, they may be exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF from the landfilled sludge. Methods used to estimate the extent of this potential exposure and its associated risks to human health are discussed in this section. Table 5-7 presents the assumptions and input parameters used.

(a) **Estimating 2,3,7,8-TCDD and 2,3,7,8-TCDF concentrations in surface water.** This analysis used the Universal Soil Loss Equation, together with estimates of sediment delivery ratios, to estimate the fraction of a lake or stream's sediment that originates from the landfill. By multiplying this fraction by the original concentration of 2,3,7,8-TCDD and 2,3,7,8-TCDF in sludge or soil particles on the landfill surface, the methodology derived estimates of the concentration of contaminants in the sediment. This contaminant load was then partitioned between adsorbed and dissolved phases, based on the assumption of equilibrium partitioning between the two phases.

(b) **Estimates of exposures and risks.** Individual exposure and cancer risk were calculated using Equations 5-5 and 5-6, respectively.

(c) **Estimates of populations exposed.** It was assumed that the density of persons potentially exposed corresponds to the average for the United States as a whole, or approximately 68 persons per square mile (0.26 per hectare) (USDOC 1987).

(4) Estimates of exposure and risks from ingestion of fish from surface water sources. Where pulp and paper sludge is deposited in uncovered landfills, particles of sludge or soil from the landfill surface can be transported by erosion to nearby lakes or streams. Fish living in the lakes or streams can take up sludge contaminants into their tissues; if humans then consume those fish, they can be exposed.

Table 5-7. Assumptions and Parameter Values - Landfills: Surface Water Pathways

Input parameter	Estimate for typical individual	Estimate for MEI individual	Notes/explanation/(reference)
Cover	None	None	
Vegetation	90%	None	USEPA (1988b).
Distance from site to surface water (meters)	152	30	USEPA (1988b).
"P" ratio	1:1	1:1	Assumes no support practices.
"C" ratio	1:1	10:1	All scenarios assume that the surrounding drainage area is pasture land (USDA 1978).
Drainage area (hectares)	1,295,000	4,047	Typical scenario assumes the sites drain into a major waterway. The MEI scenario assumes the sites drain into a relatively small stream.
Population density (people/square mile)	68	NA	USDOC (1987).
Percent of population served surface water	49	NA	USGS (1985).
Fish to sediment ratio - dioxin	1	10	USEPA (1988b).
Fish to sediment ratio - Furan	1	10	USEPA (1988b).
Percent organic carbon in soil	0.01	0.001	USEPA (1988b).
Concentration in filet vs. concentration in whole fish	0.5	0.5	See Section 2 of this report.
Percent of water and fish ingested that is contaminated	100	100	Assumes that the affected population that is served by surface water consumes 100% contaminated water.
Fish consumption (g/day)	6.5	140	Average U.S. consumption; subsistence fisher.

methodology used here is quite similar to that discussed in (3), in that both methodologies begin by estimating sediment concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF in water bodies as a result of runoff from landfills. Once sediment concentrations have been estimated, however, the methodology departs from that described in (3), and uses fish to sediment bioconcentration factors and estimates of human fish consumption contaminant doses to humans.

Table 5-6 presents assumptions and input parameters used to calculate concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF in surface water and fish tissue.

(a) **Estimating 2,3,7,8-TCDD and 2,3,7,8-TCDF concentrations in sediments and fish tissue.** This analysis used the Universal Soil Loss Equation, together with estimates of sediment delivery ratios, to estimate the fraction of a lake or stream's sediment that originates from the landfill. By multiplying this fraction by the original concentration of 2,3,7,8-TCDD and 2,3,7,8-TCDF in sludge or soil particles on the surface of the landfill, estimates of the concentration of contaminants in the sediment were derived. Empirical sediment to fish bioconcentration factors were used to estimated concentrations of the contaminant in fish. The concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF in the muscle tissues of fish (consumed by humans) were assumed to be fifty percent lower than the whole body concentrations of these contaminants.

(b) **Estimates of exposures and risks.** Individual exposure and cancer risk were calculated as follows:

$$Dose_F = \frac{(C_F)(Q_F)(BA_F)(L_F)}{(BW)(LE)} \qquad (5\text{-}7)$$

where:

$Dose_F$ = Average lifetime dose from consumption of fish (pg/kg/day)
C_F = Concentration of contaminant in fish tissue (pg/g)
Q_F = Individual's daily fish consumption (g/day)
BA_F = Bioavailability of 2,3,7,8-TCDD or 2,3,7,8-TCDF from fish (unitless); assumed to be 1.0.
BW = Human body weight (assumed to be 70 kg)
L_F = number of days of exposure in a lifetime (assumed to be 70 years x 365 days/year)
L_E = life expectancy in days in a lifetime (assumed to be 70 years)

and:

$$I_{RF} = (Dose_F)(q_1^*) \qquad (5\text{-}8)$$

where:

I_{RF} = Lifetime individual cancer risk from ingestion of fish
q_1^* = Cancer potency factor for 2,3,7,8-TCDD or 2,3,7,8-TCDF $(pg/kg/day)^{-1}$

The MEI analysis assumes a subsistence fisher consuming 140 grams/day. For the typical individual exposure analysis, fish consumption was assumed to be 6.5 grams per day.

(c) **Estimates of populations exposed.** As a reasonable worst case, the size of the total general population potentially exposed to contaminated fish was assumed to be the same size as estimated in Section 5.2.1.3 above.

(5) Summary of exposures and risks. Table 5-8 presents estimates of human risk for the potential pathways of exposure considered for landfilling of sludge. As can be seen from the tables, exposure and risks to the "most exposed individual" from the landfilling of sludge are highest from pathways associated with surface runoff. Estimated risks through these pathways for the MEI are based on an extreme scenario in which runoff from the site reaches a stream of relatively small drainage area, and the MEI is assumed to take drinking water or fish from the most contaminated segment of the stream. Average risks through surface water pathways are estimated based on the assumption of larger drainage areas, and are considerably lower.

5.2.2 Exposure and Risks from Disposal of Paper Products in Municipal Landfills

As shown in Table 5-9, the U.S. generates about 130 million metric tons of municipal waste per year, of which about 45 million tons, or 36 percent, is pulp and paper. After they are used, these products are recycled, incinerated, landfilled, or otherwise disposed. About 30 percent of the paper products generated can be expected to contain 2,3,7,8-TCDD and 2,3,7,8-TCDF. To the extent that humans come into contact with contaminants released from these wastes, risks to human health may result. This subsection considers potential health risks from the disposal of paper products in municipal landfills.

Table 5-8

Two pathways of potential human exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF from paper products in municipal landfills are considered:

- Contaminants from the paper are released into leachate within the landfill and seep into an aquifer beneath the facility. Nearby residents ingest drinking water from the aquifer and are potentially exposed.

Table 5-8. Estimates of Health Risks to the General Population from Landfill Disposal of Pulp and Paper Sludges Contaminated with 2,3,7,8-TCDD and 2,3,7,8-TCDF

Exposure pathway	MEI risk[a] (per lifetime)	Typical risk[a] (per lifetime)	Exposed population	Total risk (cases/ year)[b]
Inhalation exposure from volatilization from landfills in which sludge is disposed	5×10^{-7}	1×10^{-9}	12,800,000	2×10^{-4}
(Percent TCDD)[c]	(4.0)	(4.4)		(4.4)
Ingestion exposure from drinking ground water contaminated by leachate from landfills in which sludge is disposed	1×10^{-9}	1×10^{-10}	19,000	3×10^{-8}
(Percent TCDD)[c]	(2.0)	(8)		(8)
Ingestion exposure from drinking surface water contaminated by surface runoff from landfills in which sludge is disposed	8×10^{-4}	5×10^{-8}	6,980,000	5×10^{-3}
(Percent TCDD)[c]	(0.6)	(0.6)		(0.6)
Ingestion exposure from fish caught in surface water contaminated by runoff from landfills in which sludge is disposed	5×10^{-2}	8×10^{-8}	14,200,000	2×10^{-2}
(Percent TCDD)[c]	(63)	(65)		(65)

[a] Calculated as: (Estimated Dose) x 1.6×10^{-4} per (pg/kg)/day.

[b] Calculated as: (Typical Risk x Exposed Population) / (Life Expectancy)

[c] Calculated as: $100 \times \frac{\text{(Exposure to TCDD)}}{\text{(Exposure to TCDD) + (1/10) (Exposure to TCDF)}}$

Table 5-9. Site and Waste Characteristics for Municipal Landfills Receiving Waste Paper Contaminated With TCDD and TCDF

Site/Waste Characteristic	Value
Total municipal solid waste (MSW) (kkg/yr)[e]	1.3×10^8
Total pulp and paper waste (kkg/yr)[e]	4.5×10^7
Total bleached kraft paper, paperboard, pulp (kkg/yr)[f]	2.3×10^7
Bleached kraft paper as fraction of MSW (percent)	10.7
Maximum concentration of 2,3,7,8-TCDD in paper (ppt)	36[a]
Maximum concentration of 2,3,7,8-TCDD in MSW (ppt)	4[b]
Maximum concentration of 2,3,7,8-TCDF in paper (ppt)	333[a]
Maximum concentration of 2,3,7,8-TCDF in MSW (ppt)	36[b]
Area potentially affected for each landfill (ha)	500[c]
Density of persons using ground water (persons/ha)	4
Number of landfills	9,284
Maximum size of exposed population (persons)	18,500,000[d]

[a]Highest pulp concentration reported 104 Mill Study (USEPA).
[b]Includes only contribution from paper.
[c]Conservative assumption.
[d]Maximum size of exposed population = (area affected per landfill) x (number of landfills) x (population density).
[e]USEPA (1988a).
[f]ADL (1987).

- Contaminants from the paper are released into soil moisture and then volatilize from the landfill site to ambient air. Nearby residents inhale the contaminated air and are potentially exposed.

Human exposure through both of these pathways depends, in part, on the fraction of each landfill's contents consisting of contaminated paper products. If it is assumed that only bleached paper products contain 2,3,7,8-TCDD or 2,3,7,8-TCDF, then one can estimate the fraction of total disposed paper products likely to contain these contaminants. No information was found describing the fraction of all paper wastes originating from the bleached kraft process. Consequently, it was assumed that the fraction of paper product waste that originated from the bleached kraft process is the same as the fraction of bleached paper product production. To the extent that bleached paper products are exported, recycled, or otherwise disposed at higher or lower rates than other paper products, this assumption may over- or under-predict the fraction of municipal waste consisting of bleached paper products.

About 30 percent of all pulp and paper is produced from the bleach kraft process. If this same fraction also applies to solid wastes, then one would expect bleached paper wastes to account for about 11 percent of total municipal waste. The further assumption that bleached paper products' contribution to municipal landfills does not differ from its contribution to the total municipal waste stream would imply that bleached kraft accounts for about 11 percent of a typical municipal landfill's contents.

Consequently, since roughly one-tenth of the total paper products disposed of in a municipal landfill comprises bleached paper products, the average concentration over all disposed paper products can be assumed to be one-tenth the concentration in bleach paper products. The highest reported concentrations for 2,3,7,8-TCDD and 2,3,7,8-TCDF in bleached pulp from the 104 Mill Study are 36 ppt and 333 ppt, respectively. If these same concentrations are assumed to apply to the pulp and paper products disposed in landfills, then one would expect the average concentration of 2,3,7,8-TCDD and 2,3,7,8-TCDF to be 3.6 ug and 33.3 ug per metric ton of paper wastes, respectively.

To assess potential human exposures and risks from disposal of waste paper in municipal landfills, a series of conservative assumptions was used to estimate contaminant concentrations in air above a landfill and in ground water beneath it. Details of these calculations are presented in Subsections (1) and (2).

(1) Estimates of exposures and risks from inhalation of vapors. Humans can be exposed to potential health risks if 2,3,7,8-TCDD and 2,3,7,8-TCDF volatilize from paper in municipal landfills. An upper bound estimate of the extent of these risks can be derived by combining

consistently conservative assumptions into mathematical models for estimating the rate of emissions from these landfills and then estimating the extent to which emitted contaminants are diluted before inhalation. In Table 5-9, conservative estimates of 2,3,7,8-TCDD and 2,3,7,8-TCDF concentrations in a municipal landfill that might result from the disposal of paper products are presented. Based on these concentrations, this analysis uses a set of equations to predict emissions from a landfill site.

(a) Methods for estimating volatile emissions from surface impoundments. A numerical solution of a partial differential equation was used to determine the reduction in volatile emissions that would be expected following the addition of a soil layer to the top of a landfill (USEPA 1988b). For a 10 to 25 centimeter soil cover, emission rates were estimated to be reduced by 75 to 80 percent, given a contaminated layer thickness of 8 feet. This analysis assumed that municipal landfills apply such a cover and that emissions are reduced accordingly.

As shown in Table 5-3, organic carbon partition coefficients for 2,3,7,8-TCDD and 2,3,7,8-TCDF are assumed to be 1×10^7 and 3.5×10^4, respectively. The contents of the landfill are (conservatively) assumed to be only one percent organic carbon, resulting in K_D estimates of only 1×10^4 and 3.5×10^1 for 2,3,7,8-TCDD and 2,3,7,8-TCDF, respectively. For 2,3,7,8-TCDD, the K_D estimate is further reduced to correspond to the lowest reported partition coefficient between 2,3,7,8-TCDD in paper and in liquid or about 2,000 g/g. After adjustment for a 10 to 25 centimeter cover layer, it was estimated that emissions of 1×10^{-17} and 7×10^{-16} g/m^2/second will occur for 2,3,7,8-TCDD and 2,3,7,8-TCDF, respectively.

(b) Methods for estimating wind transport of volatile emissions. As a conservative estimate of ambient air concentrations of these contaminants near the landfill, an atmospheric box model (USEPA 1988b) was used to estimate concentrations of contaminants in air above the site. The equation used as the basis of the model is as follows:

$$C_a = \frac{Q}{(LS)(V)(MH)} \qquad (5\text{-}9)$$

where:

- Q = total emissions from site (g/sec)
- C_a = ambient air concentration of 2,3,7,8-TCDD or 2,3,7,8-TCDF at the exposure location (g/m^3),
- LS = equivalent side length of the site perpendicular to the wind (m),
- MH = mixing height before being inhaled by an individual (m), and
- V = average wind speed at the inhalation height (m/s)

It has been reported that 95 percent of all municipal landfills are less than 100 acres (41 hectares) in area (USEPA 1988c). This analysis considers a landfill of 41 hectares, represented as a square 640 meters wide. If the average wind velocity at the site is 4.5 meters per second, then the mixing volume of air within a height of 1.5 meters above the site is about 1,340 cubic meters per second. Given total emissions of 2,3,7,8-TCDD and 2,3,7,8-TCDF of 1×10^{-17} and 7×10^{-16} grams per second from the landfill area, the box model predicts ambient air concentrations of about 1×10^{-3} and 9×10^{-2} pg/m^3, respectively.

(c) Estimates of exposures and risks. Based on the estimated concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF in ambient air, individual exposures and cancer risks were calculated using Equations 5-1 and 5-2.

Based on assumed inhalation rates of 23 m^3 per day of outdoor air directly above the landfill site, these concentrations would result in upper bound cancer risks of less than 1×10^{-7}. Because the risk to a most exposed individual for this very conservative "high risk" scenario is so low, estimate of risk to the typical individual was not derived.

(2) Estimates of exposures and risks from ingestion of ground water. Landfill disposal of paper or paperboard containing 2,3,7,8-TCDD and 2,3,7,8-TCDF can result in human exposure and risk if these contaminants migrate from the landfill to groundwater, and are then transported to nearby drinking water wells.

Table 5-10 presents information about models used to estimate concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF in groundwater and a few key assumptions used to estimate those concentrations.

(a) Method for estimating contaminant loadings to groundwater. The estimated average concentration in municipal solid waste of 2,3,7,8-TCDD and 2,3,7,8-TCDF, adjusted by 10.7 percent to account for other waste in the landfill, were 4 and 36 ng/kg, respectively (see Table 5-9). The partition coefficients for 8 percent ethanol were used (2,000 cm^3/g) (see Appendix A.1). The maximum equilibrium concentration of the two chemicals in leachate, estimated from their concentration in municipal solid waste divided by their partition coefficient, was estimated to be 2×10^{-6}ug/l for 2,3,7,8-TCDD and 2×10^{-5}ug/l or 2,3,7,8-TCDF.

(b) Method for estimating groundwater concentrations. Ninety-five percent of all municipal landfills are less than 100 acres (41 hectares) in area (USEPA 1988c). If leachate from the landfill contains 2,3,7,8-TCDD and 2,3,7,8-TCDF at the estimated maximum concentrations, a 100 acre landfill site with 43 cm/year of recharge would release a maximum loading of 1.6×10^{-11} kg/hour of TCDD and

Table 5-10. Assumptions and Parameter Values - Paper Wastes in Municipal Landfills: Ground-Water Pathway

Input parameter	Estimate for typical individual	Estimate for MEI individual	Notes/explanation/(reference)
Model used for transport through saturated zone	NA	AT123D	
Results used for exposure estimates	NA	Steady state	Conservative.
Distance to aquifer (m)	NA	0	Conservative.
Aquifer medium	NA	Silty sand	
Depth of aquifer	NA	10	
Width of aquifer (m)	NA	500	
Distance to model wells (m)	NA	200	
Decay	NA	0	No decay rate available for TCDD or TCDF in ground water.
Effective porosity of aquifer medium	NA	0.1	Appropriate for silty sand.
Hydraulic conductivity	NA	1	"High risk" estimate appropriate for silty sand.
Hydraulic gradient	NA	1%	Typical value.
Longitudinal dispersivity (m)	NA	10	Within range of "typical" values reported for sand or silt.
Percent organic carbon in soil	0.01	0.001	
Water consumption (liters/day)	2	2	

Table 5-10. (continued)

Input parameter	Estimate for typical individual	Estimate for MEI individual	Notes/explanation/(reference)
Human absorption of water	100	100	
Transverse	NA	1	Within range of "typical" values reported for sand or silt.
Vertical dispersivity (m)	NA	1	
TCDD K_D in aquifer	NA	10,000	Does not affect steady state results.
TCDF K_D in aquifer	NA	35	Does not affect steady state results.

NA = Not applicable.

1.5×10^{-10} kg/hour of TCDF (from paper wastes) to an underlying aquifer. For a hypothetical landfill of 410 meters width and length (100 acres), and for an aquifer with characteristics described by the "high risk" scenario in Table 5-9, the AT123D model was used to estimate estimate well concentrations at 200 meters from the edge of the facility. Maximum predicted concentrations at 200 meters distance were 1×10^{-4} pg/l for 2,3,7,8-TCDD and 1×10^{-3} pg/l for 2,3,7,8-TCDF.

(c) Estimates of exposures and risks. Based on assumed rates of individual water ingestion per day (2 liters/day) and duration of exposure (70 years) individual exposures and cancer risks were calculated using Equations 5-4 and 5-5, respectively.

(3) Summary of results. Table 5-11 summarizes estimates of human risk for the disposal of waste paper in municipal landfills. Based on these results, 2,3,7,8-TCDD and 2,3,7,8-TCDF in waste paper products do not appear to result in significant human risks to human health when received by municipal landfills. Using conservative assumptions, it was estimated that maximum cancer risks to human health from the volatilization of 2,3,7,8-TCDD and 2,3,7,8-TCDF from municipal landfills would be expected to be lower than 1×10^{-6} for a "most exposed individual" who lives 24 hours per day on the top of the landfill. Risks from groundwater contamination appear to be lower still, with cancer risks to the MEI of less than 1×10^{-9}.

5.2.3 Exposures and Risks from Disposal of Pulp and Paper Sludge in Surface Impoundments

Surface impoundments are defined as facilities in which pulp and paper mill sludge is stored or disposed on land without a cover layer of soil. For this analysis, it was assumed that sludge contained in such facilities was of higher moisture content than the sludge deposited in landfills, at least in the active phase of the surface impoundment. Twenty facilities in the 104-Mill Study (USEPA 1989a) reported using surface impoundments for their sludge.

This analysis estimated general population exposures and risks to 2,3,7,8-TCDD and 2,3,7,8-TCDF through four exposure pathways associated with surface impoundments:

- Volatilized 2,3,7,8-TCDD and 2,3,7,8-TCDF are emitted from the impoundment surface. These chemicals are transported downwind to nearby areas. Humans inhale the contaminated ambient air and are exposed.

- Contaminants from sludge placed in the impoundment are dissolved in water seeping through the bottom of the impoundment. Contaminated water enters an aquifer beneath the impoundment, and

Table 5-11. Estimates of Health Risks to the General Population from Landfill Disposal of Paper Contaminated with 2,3,7,8-TCDD and 2,3,7,8-TCDF

Exposure pathway	MEI risk[a] (per lifetime)	Typical risk[a] (per lifetime)	Exposed population	Total risk (cases/year)[b]
Inhalation exposure from volatilization from municipal landfills in which paper is disposed:	$<6\times10^{-7}$	$<6\times10^{-7}$	NA[d]	NA[d]
(Percent TCDD disposed)[c]	(16)	(16)		
Ingestion exposure from drinking ground water contaminated by leachate from municipal landfills in which paper is disposed:	1×10^{-9}	$<1\times10^{-9}$	18,500,000	$<2\times10^{-2}$
(Percent TCDD)[c]	(53)	(53)		(53)

[a] Calculated as: (Estimated Exposure) x 1.6×10^{-4} per (pg/kg)/day

[b] Calculated as: (Average Risk x Exposed Population) / (Life Expectancy)

[c] Calculated as: $100 \times \frac{\text{(Exposure to TCDD)}}{\text{(Exposure to TCDD)} + (1/10)\ \text{(Exposure to TCDF)}}$

[d] Not applicable.

flows down-gradient to drinking water wells. Humans withdraw drinking water from the contaminated aquifer and are exposed.

- Surface runoff carries particles of sludge from the surface of the impoundment to a nearby lake or stream. 2,3,7,8-TCDD and 2,3,7,8-TCDF adsorbed to these particles enter the surface water body, and some of it dissolves in the surface water. Humans withdraw surface water for drinking and are exposed.

- Surface runoff carries particles of sludge from the surface of the impoundment to a nearby lake or stream, where the particles are suspended or settle to bottom sediment. Fish absorb and bioconcentrate 2,3,7,8-TCDD and 2,3,7,8-TCDF from the sludge particles. Humans ingest the fish, and are exposed.

Each of these potential pathways of exposure to the general population is described below. Table 5-12 summarizes the characteristics of hypothetical surface impoundments intended to be representative of those that receive pulp and paper sludge.

(1) Estimates of exposure and risk from inhalation of vapors. Modeling potential exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF vapor from surface impoundments involved two steps. First, the rate at which each contaminant is emitted from the surface of an impoundment facility was estimated. Second, wind transport of emitted contaminants affecting ambient air concentrations in surrounding areas was estimated. Information about models used to estimate volatile emissions and a few key assumptions used to estimate release from volatilization are presented in Table 5-13.

(a) **Methods for estimating volatile emissions from surface impoundments.** Several authors have proposed methods for estimating emissions from liquids contained in surface impoundments. Common to most methodologies is the use of a two-layer resistance model to estimate volatile emissions from a liquid surface. In general, the methodologies assume that emissions occur by molecular diffusion through non-turbulent, viscous sublayers on either side of the air/liquid interface, and that these two films form the dominant resistances to mass transfer across the interface. Background contaminant concentrations in ambient air are assumed negligible. This study used a two-layer resistance model with estimated mass transfer coefficients (USEPA 1989b).

Estimation of emissions required additional assumptions for estimating the dissolved concentration of 2,3,7,8-TCDD and 2,3,7,8-TCDF. For example, concentrations of dissolved contaminant may vary with depth in the lagoon or over time. If the contents of the impoundment were well mixed, if sludge was deposited only once, and if no contaminant was lost to other loss processes, then the concentration of TCDD in the

Table 5-12. Assumptions and Parameter Values - Surface Impoundments: All Exposure Pathways

Input parameter	Estimate for typical individual	Estimate for MEI individual	Notes/explanation/(reference)
Number of impoundments per site	3	3	USEPA (1985a).
Area of each impoundment (hectares)	25	50	Typical estimate from USEPA (1985). MEI estimate is typical estimate + 100%.
Depth of impoundment (m)	3	3	
Fraction organic carbon in sludge (% dry wt)	25%	25%	Midpoint of range reported by NCASI (1984).
Sludge concentration TCDD (ppt)	163	293	Mean and 90th percentile reported concentrations (USEPA 1989a).
Sludge concentration TCDF (ppt)	885	1,760	Mean and 90th percentile reported concentrations (USEPA 1989a).
Quantity of sludge received (DMT/yr)	28,653	91,250	Mean and highest reported quantities. (USEPA 1989a).

Table 5-13. Assumptions and Parameter Values - Surface Impoundments: Volatilization Pathway

Input parameter	Estimate for typical individual	Estimate for MEI individual	Notes/explanation/(reference)
Model used to estimate emissions	Two-phase resistance	Two-phase resistance	USEPA (1989b)
Model used to estimate wind transport and human exposure	ISCLT	ISCLT	Bowers et al. (1980)
Temperature (°C)	25	25	
Average windspeed (m/sec)	4.5	4.5	Single average windspeed used to estimate emissions (USEPA 1988b). Site-specific windspeeds and direction used for ISCLT calculations.

impoundment would diminish as a result of continued emissions. If sludge were instead deposited regularly in the impoundment, then the dissolved concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF might remain constant or increase. Unfortunately, sufficient data were not available for detailed, site-specific modeling of the time path of dissolved contaminant concentrations in existing pulp and paper sludge impoundments. This analysis therefore attempted to approximate concentration and emission estimates based on idealized scenarios.

It was assumed that the impoundment was well mixed, with regular additions of sludge containing 2,3,7,8-TCDD and 2,3,7,8-TCDF in known (dry weight) concentrations. It was assumed that 2,3,7,8-TCDD and 2,3,7,8-TCDF were partitioned at equilibrium between adsorbed and dissolved phases in the impoundment.

(b) Methods for estimating wind transport of volatile emissions. Once 2,3,7,8-TCDD or 2,3,7,8-TCDF vapor is emitted from a surface impoundment, it can be transported downwind to nearby residents, resulting in potential human exposure and health risks. This analysis used a Gaussian plume dispersion model to estimate the extent to which air concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF would be reduced in the process of wind transport. Calculations were performed by the area source version of ISCLT (Industrial Source Complex, Long Term) model maintained in the USEPA/OTS Graphical Exposure Modeling System, or GEMS.

Because specific information required to estimate emissions of 2,3,7,8-TCDD and 2,3,7,8-TCDF to air at each site was not available, all sites in the inventory were assumed to be of identical size, to contain sludge with the same concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF, to use the same management practices, and to emit contaminants at identical rates. These assumptions varied between "typical" and "MEI" estimates, but were applied consistently across all facilities.

(c) Estimates of human exposures and risks. Based on estimated concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF in ambient air, individual exposure and cancer risk were calculated using Equations 5-1 and 5-2, respectively.

ISCLT provided estimates of population-weighted average concentrations of the 2,3,7,8-TCDD or 2,3,7,8-TCDF in ambient air surrounding all surface impoundments. Aggregate exposures and cancer risks were calculated using Equations 5-3 and 5-4, respectively.

(d) <u>Estimates of populations exposed</u> The area source version of the model estimates ambient air concentrations at selected locations in a polar grid centered on an area source of emissions and extending 560 kilometers in all directions. It then maps those air concentrations onto actual human populations for the regions involved. Population data are

drawn from the 1980 Census, mapped to the level of Census block group and enumeration district.

(2) Estimates of exposures and risks from ingestion of ground water. If 2,3,7,8-TCDD and 2,3,7,8-TCDF are dissolved in water that seeps from the bottom of a surface impoundment, these contaminants may be transported to an aquifer beneath the facility. If those chemicals are then transported down-gradient by ground water, then they may reach drinking water wells near the site. This section outlines the methods used by this analysis to assess the extent of this potential route of exposure and risk. Table 5-14 lists key assumptions and input parameters used to estimate exposures and risks from ingestion of ground water.

(a) **Methods for estimating seepage beneath a surface impoundment.** Potential ground-water contamination from surface impoundments was estimated similarly to that from landfills, with one important difference. If a surface impoundment contains a significant volume of water, drainage from the pond may result in increased recharge and contaminant loading to the underlying aquifer. The extent to which the downward flux of water beneath a site is increased by water from the impoundment will depend on:

- the amount of water in the impoundment,
- whether a natural or synthetic liner is present,
- whether the water content of the impoundment is periodically restored by additional deposits of sludge,
- the extent to which the solids layer on the bottom of the impoundment inhibits water flux out of the impoundment, and
- the hydraulic conductivity of the medium between the impoundment and the water table.

Of course, if aquifer recharge beneath an impoundment significantly exceeds the sum of net precipitation and the annual influx of water from sludge, then the impoundment will soon dry out. A sustained and substantial increase in ground-water recharge beneath an impoundment therefore requires repeated additions of water to an active impoundment.

Movement of TCDD (and to a lesser extent TCDF) through soil is retarded by its high soil/water partition coefficient. Given a significant soil layer between the impoundment and the water table, steady-state loadings of these contaminants to groundwater beneath an impoundment may not be reached until many years after the sludge is last placed in the facility. If the active lifetime of the impoundment ends before 2,3,7,8-TCDD and 2,3,7,8-TCDF reach the underlying aquifer, then

Table 5-14. Assumptions and Parameter Values - Surface Impoundments: Ground-Water Pathway

Input parameter	Estimates for Typical individual	Estimate for MEI individual	Notes/explanation/(reference)
Model used for transport through unsaturated zone	SESOIL	SESOIL	Low and best estimates assume that long term average recharge to aquifer is not affected by water in impoundment. High estimate assumes recharge limited by hydraulic conductivity in unsaturated zone.
Result used for exposure estimates	Steady state	Steady state	
Vertical distance to aquifer (m)	3	3	Assumes 6-meter depth to water table (DRASTIC data base).
Aquifer recharge (cm/yr)	43	43	Estimate represents typical recharge rate for areas with surface impoundments. Assumes long-term recharge is not affected by water in impoundment.
Vadose zone medium	Sand & gravel, silt & clay	Sand & gravel silt & clay	Most common medium in counties containing surface impoundments (DRASTIC data base).
Hydraulic conductivity of unsaturated zone (m/sec)	1×10^{-7}	1×10^{-7}	Selected value is between ranges typical for sand and for clay. Would result in complete drainage of inactive lagoon within three years (Freeze and Cherry 1979).
Effective porosity of aquifer medium	0.25	0.25	Appropriate for sand and gravel (Freeze and Cherry 1979, Yeh 1981).
Hydraulic conductivity (m/hr)	10	10	Appropriate for productive sand and gravel aquifer (Freeze and Cherry 1979).
Hydraulic gradient	1%	1%	"Typical" value (USEPA 1988b).
Longitudinal dispersivity (m)	20	20	Within range of "typical" values reported for sand. Equals approx. 1/10 distance to receptor well (Yeh 1981).

Table 5-14. (Continued)

Input parameter	Estimates for Typical individual	Estimate for MEI individual	Notes/explanation/(reference)
Transverse dispersity (m)	7	7	Within range of "typical" values reported for sand. Equals 1/3 longitudinal dispersity (Yeh 1981).
Vertical dispersivity (m)	1	1	Within range of "typical" values reported for sand (Yeh 1981).
TCDD K_D in aquifer	10,000	10,000	Does not affect steady-state results.
TCDF K_D in aquifer	35	35	Does not affect steady-state results.
Distance to model wells (m)	200 1,200 3,000	200	Midpoints of ranges listed below.
Distances represented (m)	0-400 400-2,000 2,000-4,000	200	
Exposed populations: 0-400 m 400-2,000 m 2,000-4,000 m	63 1,500 4,670	--	Assumes density of 68 persons/sq. mile (USDOC 1987).

NA = Not applicable.

the water content of a surface impoundment may not result in appreciable increases in the peak or "steady state" loading of sludge contaminants to the aquifer.

Ideally, ground-water contamination beneath a surface impoundment could be modeled separately for the active phase of the impoundment (during which sludge is regularly added to the impoundment and the liquid content of the impoundment is maintained or increased), and the inactive phase of the impoundment (during which no further sludge is added, and the liquid content is decreasing or is at equilibrium with precipitation less evaporation). For lack of sufficient data, the present analysis did not attempt to model these two periods separately, or to quantify the exact extent to which water in an impoundment would increase recharge (and contaminant loading) to an underlying aquifer.

The analysis assumed that the impoundment would be inactive during the period in which maximum loading of 2,3,7,8-TCDD and 2,3,7,8-TCDF to the aquifer takes place. If so, then the resulting loading to ground water could be calculated with methods identical to those used to estimate ground-water contamination beneath sludge landfills. Estimates of exposure and risk were based on results from SESOIL simulations similar to those described in Section 5.2.1.

(b) Contaminant concentrations in seepage beneath an impoundment. Contaminant concentrations in a surface impoundment are likely to vary with depth and the age of the impoundment; dissolved concentrations may be highest at the bottom of the impoundment, where sludge has settled and the solids are concentrated. As an upper bound estimate of water concentrations at the bottom of the impoundment, this analysis assumed that concentrations were unlikely to exceed values suggested by the ratio: dry weight concentration of contaminant in sludge/soil-water partition coefficient for contaminant.

As an alternative approach, it was reasoned that 2,3,7,8-TCDD and 2,3,7,8-TCDF from a typical surface impoundment may not reach the aquifer during the active lifetime of the facility. If so, then long-term loadings of 2,3,7,8-TCDD and 2,3,7,8-TCDF may be better modeled based on the assumption that the surface impoundment had lost its excess moisture and behaved as a soil column similar to those modeled in Section 5.2.1 for landfills. The SESOIL model allows a more comprehensive approach to contaminant transport through soil layers. The model considers monthly climate data, and maintains a mass balance for contaminant transport through multiple soil layers. Estimates of potential human exposure and risks through the groundwater pathway were derived by using the SESOIL model to simulate 2,3,7,8-TCDD and 2,3,7,8-TCDF transport to the aquifer once steady state conditions had been reached in the unsaturated zone. Loadings to ground water were proportional to assumed recharge beneath

the landfill. A value of 43 centimeters recharge per year was selected based on GEMS data for a county in Wisconsin.

(c) Use of AT123D to predict contaminant transport through the aquifer. From estimated loadings of 2,3,7,8-TCDD and 2,3,7,8-TCDF to groundwater, the AT123D model was used to predict contaminant concentrations at wells down-gradient of each site. As with landfills, this analysis considered only the "steady state" concentrations predicted by the model, without regard to the amount of time required to reach steady state.

(d) Estimates of exposures and risks. Based on assumed rates of individual water ingestion per day (2 liters per day) and exposure over an entire lifetime, individual exposure and cancer risk were calculated using Equations 5-5 and 5-6, respectively.

Exposure and risk were estimated separately for persons taking drinking water at each of the three model distances from a surface landfill site. MEI exposure and risk were assumed to occur at the nearest well location.

(e) Estimates of exposed populations. To calculate the size of the exposed populations, the size of the affected area downgradient of each site (within 400, 2000, or 4000 meters) is multiplied by the average population density (68 persons per square mile) (USDOC 1987). The results are multiplied by 20 (i.e., the number of facilities reporting to use impoundments) (USEPA 1989a).

(3) Estimates of exposures and risks from ingestion of surface water. The extent of exposure and risks associated with surface water pathways for surface impoundments will depend on the characteristics of individual surface impoundment sites, and on management practices used to contain the sludge. If a facility is surrounded by a substantial berm, for example, runoff from the impoundment will be minimized. This analysis conservatively assumed that runoff from an inactive surface impoundment would result in the same amount of soil transport from the facility per unit area as estimated for landfills without cover. The methodology used for this analysis is thus nearly identical to the approach discussed in Section 5.2.1.

Table 5-15 presents the assumptions and input parameters used to calculate exposures and risks as a result of ingestion of surface water from disposal of TCDD- and TCDF-contaminated pulp and paper sludge in surface impoundments.

(a) Estimating 2,3,7,8-TCDD and 2,3,7,8-TCDF concentrations in surface water. In general, these calculations used the Universal Soil Loss Equation, together with estimates of sediment delivery ratios, to

Table 5-15. Assumptions and Parameter Values - Surface Impoundments: Surface Water Pathway

Input parameter	Estimate for Typical individual	Estimate for MEI individual	Notes/explanation/(reference)
Cover	None	None	
Vegetation	90%	None	USEPA (1988b).
Distance from site to surface water (meters)	152	30	USEPA (1988b).
"C" ratio	1.1:1	10:1	All scenarios assume that the surrounding drainage area is pasture land (USDA 1978).
"p" ratio	1:1	1:1	Assumes no support practices.
Drainage area (hectares)	1,295,000	4,047	The typical scenario assumes the sites drain into a major waterway. The MEI scenario assumes the sites drain into a smaller tributary.
Population density (people/square mile)	68	NA	USDOC (1987).
Percent of population served by surface water	49	NA	USGS (1985).
Fish consumption (g/day)	6.5	140	Average U.S. consumption; subsistence fisher.
Percent of water and fish consumed that is contaminated	100	100	
Fraction organic carbon in soil	0.01	0.001	USEPA (1988b)
Fish to sediment ratio: TCDD	1	10	USEPA (1988b)
TCDF	1	10	

NA = Not applicable

estimate the fraction of a lake or stream's sediment that originated from the landfill. By multiplying this fraction by the original concentration of 2,3,7,8-TCDD and 2,3,7,8-TCDF in sludge or soil particles on the landfill surface, the methodology derived estimates of the concentration of contaminants in the sediment. This contaminant load was then partitioned between adsorbed and dissolved phases, based on the assumption of equilibrium partitioning between the two phases.

(b) Estimates of exposures and risks. Individual exposure and cancer risks were calculated using Equations 5-5 and 5-6, respectively.

(c) Estimates of populations exposed. The population exposed to contaminated water is estimated by multiplying the area of the drainage basin by the estimated population density (68 persons/sq. mile). This estimated population was then multiplied by the fraction of the population that takes its drinking water from surface supplies (49 percent).

(4) Estimates of exposure and risks from ingestion of fish from surface water sources. Where pulp and paper sludge is deposited in uncovered landfills, particles of sludge or soil from the landfill surface can be transported by erosion to nearby lakes or streams. If the sludge contains 2,3,7,8-TCDD or 2,3,7,8-TCDF, then those particles can carry these contaminants to the surface water bodies. Fish living in the lakes or streams can take up sludge contaminants into their tissues; if humans then consume those fish, they can be exposed.

This section discusses methods that were used to estimate the extent of this potential exposure, and its associated risks to human health. The methodology was quite similar to that discussed in Section 5.2.3(3), in that both methodologies began by estimating sediment concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF in water bodies as a result of runoff from landfills. Once sediment concentrations were estimated, however, the methodology departed from that described in Section 5.2.3.(3), and used fish-to-sediment bioconcentration factors and estimates of human fish consumption contaminant doses to humans. The last step in the methodology involved estimating the sizes of exposed populations, and combining these results with estimates of individual dose and health risk to derive total health risks to the entire exposed population.

(a) Estimating the concentration of 2,3,7,8-TCDD and 2,3,7,8-TCDF in sediments and fish tissues. This analysis used the Universal Soil Loss Equation, together with estimates of sediment delivery ratios, to estimate the fraction of a lake or stream's sediment that originated from the landfill. By multiplying this fraction by the original concentration of 2,3,7,8-TCDD and 2,3,7,8-TCDF in sludge or soil particles on the surface of the landfill, the methodology derived estimates of the concentration of contaminants in the sediment. These

sediment concentrations were then used to estimate contaminant concentrations in the tissues of fish.

Based on the assumption that sediment concentrations were the best predictor of fish concentrations of hydrophobic compounds like 2,3,7,8-TCDD and 2,3,7,8-TCDF, the methodology used empirical fish-to-sediment bioconcentration factors to estimate concentrations of contaminant in freshwater fish as a function of concentrations in stream or lake sediment. The concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF in the muscle tissues of fish (consumed by humans) were considered to be fifty percent lower than the whole body concentrations of these contaminants.

(b) Estimates of exposures and risks. Individual exposure and cancer risk were calculated using Equations 5-7 and 5-8, respectively.

(c) Estimates of populations exposed. The population exposed to fish containing 2,3,7,8-TCDD and 2,3,7,8-TCDF was estimated by multiplying the area of the drainage basin containing each facility by the estimated population density (68 persons/sq. mile).

(5) Summary of results. Table 5-16 presents risks to humans from the four potential pathways of exposure considered for disposal of sludge in surface impoundments. Exposure through ingestion of fish from surface water sources showed the highest risk, followed by ingestion of drinking water. Highest total population risk was observed through the volatilization pathway.

5.2.4 Exposures and Risks from Land Application of Pulp and Paper Mill Sludge

Land application of sludge, while an alternative disposal method, also fertilizes and conditions soil and allows the sludge to be used as fill. According to the 104-Mill Study and follow-up conversations with state environmental offices, sludge from four mills in three states is used on forests; sludge from two mills in two states is applied to agricultural land; and sludge from two mills in two states is used to reclaim abandoned mine sites. Conversations with state environmental offices indicate that approximately 325,000 dry metric tons of sludge are being land applied per year with about 10 percent to mine reclamation, 10 percent to agriculture, and 80 percent to forests. This analysis examines the risks to human health from the land application of pulp and paper mill sludges.

In this assessment, two application rates are used - one for the agricultural application scenario and one for the mine and forest application scenario. To estimate reasonable values for these generic application rates, the rates of agricultural application obtained from states were averaged and the rates of forest and mine application

Table 5-16. Estimates of Health Risks to the General Population from Surface Impoundment of Pulp and Paper Sludges Contaminated with 2,3,7,8-TCDD and 2,3,7,8-TCDF

Exposure pathway	MEI risk[a] (per lifetime)	Typical risk[a] (per lifetime)	Exposed population	Total risk (cases/ year)[b]
Inhalation exposure from volatilization from surface impoundments in which sludge is disposed	$1x10^{-6}$	$5x10^{-8}$	7,100,000	$5x10^{-3}$
(Percent TCDD)[c]	(0.6)	(0.7)		(0.7)
Ingestion exposure from drinking ground water contaminated by leachate from surface impoundments in which sludge is disposed	$3x10^{-8}$	$5x10^{-10}$	6,000	$4x10^{-8}$
(Percent TCDD)[c]	(0.4)	(0.7)		(0.7)
Ingestion exposure from drinking surface water contaminated by surface runoff from surface impoundments in which sludge is disposed	$2x10^{-3}$	$6x10^{-8}$	2,330,000	$2x10^{-3}$
(Percent TCDD)[c]	(0.6)	(0.7)		(0.7)
Ingestion exposure from fish caught in surface water contaminated by runoff from surface impoundments in which sludge is disposed	$1x10^{-1}$	$1x10^{-7}$	4,760,000	$7x10^{-3}$
(Percent TCDD)[c]	(63)	(65)		(65)

[a] Calculated as: (Estimated Exposure) x $1.6x10^{-4}$ per (pg/kg)/day.

[b] Calculated as: (Typical Risk x Exposed Population) / (Life Expectancy)

[c] Calculated as: $100 \times \frac{\text{(Exposure to TCDD)}}{\text{(Exposure to TCDD) + (1/10) (Exposure to TCDF)}}$

obtained from states were averaged. Application rates that were estimated based on practices in other states were not included in the averages.

It is assumed that sludge that is applied agriculturally is incorporated to a depth of six inches. Sludge applied to reclaim mines is assumed to be top-dressed. Silvicultural application is analyzed as a unit with mine reclamation, and conservatively assumed not be be incorporated with the soil. Calculations of soil concentrations for mine reclamation and silvicultural applications assume a one-time application. Agricultural applications of sludge are assumed to continue for 70 years in the MEI analysis and for 20 years in the typical analysis. These assumptions are summarized in Table 5-17.

Since likely exposure pathways differ depending on the type of land receiving the sludge, exposure pathways considered in this analysis differed for agricultural and mine/forest application. In addition, farmers will be exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF from agricultural application through pathways that are not relevant to the population at large. The following pathways of exposure were considered in this analysis:

Risk estimates for forest application/mine reclamation considered two pathways for both a total population and a most exposed individual (MEI) risk:

- Contaminated soil erodes from the forest/mine site and contaminates surface water and stream sediment. The surface water serves as a drinking supply.

- Contaminated soil erodes from the forest/mine site and contaminates surface water and sediment. 2,3,7,8-TCDD and 2,3,7,8-TCDF is incorporated into fish tissue and fish are consumed by humans.

Risk estimates from agricultural application considered the two pathways above and added the following pathway for both a total population and a subsistence farmer's (MEI) risk:

- Small amounts of contaminant are taken up into the tissues of crops. These crops are then either consumed or fed to animals which bioconcentrate the contaminant and produce a meat or dairy product that is consumed.

Other exposure pathways particular to farming were:

- Children and adults in the farming household come into direct dermal contact with the sludge-amended soil in both outdoor and indoor settings. 2,3,7,8-TCDD and 2,3,7,8-TCDF from the sludge is

Table 5-17. Assumptions and Parameter Values - Land Application: All Exposure Pathways

Input parameter	Estimate for typical individual	Estimate for MEI individual	Notes/explanation/reference)
Application rate (DMT/HA)			
Forest/mine	776	776	Average of individual states (State Environmental Officials)
Agriculture	38	38	
Years land receives sludge			
Forest/mine	1	1	Estimates based on information from individual states
Agriculture	20	70	
Incorporation depth (cm)			
Forest/mine	0	0	Estimates based on information from individual states
Agriculture	15	15	
TCDD concentration in soil (ppt)			
Forest/mine	163	293	Average and 90th percentile values for all mills (USEPA 1989a).
Agriculture	35	107	See Section 5.2.4(1)(a).
TCDF concentration in soil (ppt)			
Forest/mine	885	1,760	Average and 90th percentile values for all mills (USEPA 1989a).
Agriculture	188	645	See Section 5.2.4(1)(a).

absorbed through the skin. Children ingest small amounts of the sludge/soil mixture through normal mouthing behavior. Adults also inadvertently ingest small quantities of sludge/soil.

- 2,3,7,8-TCDD and 2,3,7,8-TCDF applied to the farmland volatilizes from the sludge into the air. Residents of the farm inhale the volatilized 2,3,7,8-TCDD and 2,3,7,8-TCDF.

- Particles of the sludge/soil mixture become suspended in the air during application. Residents of the farm inhale the contaminated particles.

The following sections describe the methods and data used to estimate risks from land application of sludge through these pathways. Results are summarized in the final section.

(1) Estimates of exposures and risks from dermal contact with skin. Humans coming in direct contact with sludge contaminated soils may absorb 2,3,7,8-TCDD and 2,3,7,8-TCDF through their skin. The amount of 2,3,7,8-TCDD and 2,3,7,8-TCDF absorbed depends on the area of skin exposed and on the length of time that the contaminated soil is in contact with the skin. The following discussion summarizes the process used to estimate exposure through dermal contact. The values used for each model input are summarized in Table 5-18.

To estimate exposure through direct contact with soil containing 2,3,7,8-TCDD and 2,3,7,8-TCDF, various methodologies were used. The dermal exposure model used empirically-derived information on the amount of soil or dust that adheres to a square centimeter of skin, the area of skin exposed in various settings, and the absorption rate of 2,3,7,8-TCDD or 2,3,7,8-TCDF through skin to derive the dose of 2,3,7,8-TCDD or 2,3,7,8-TCDF from dermal contact with contaminated soil or dust.

The estimation of dermal exposure proceeded in two steps. First the average daily exposure from dermal contact was calculated as the product of area of skin affected, the contact rate, the dermal absorption rate and the duration of contact. Second, the risk from dermal contact was calculated using the estimate of daily exposure and the potency of 2,3,7,8-TCDD and 2,3,7,8-TCDF.

(a) **Methods for estimating soil concentrations.** The methods of sludge application considered were (1) application as a top dressing, and (2) incorporation into a layer of soil. Concentrations in the soil for all top dressing scenarios were assumed equal to concentrations in the land-applied sludge. For sludge that was incorporated into the soil, the concentrations were dependent on the depth of the sludge incorporated in the soil, frequency of application, and initial sludge concentration.

Table 5-18. Assumptions and Parameter Values - Land Application: Dermal Pathway

Input parameter	Estimate for Typical individual	Estimate for MEI individual	Notes/explanation/(reference)
CONCENTRATION			
Contact rate (mg/cm^2), outdoor, child	0.5	1.5	Schaum (1984).
Contact rate (mg/cm^2), outdoor, older child	0.5	1.5	Schaum (1984).
Contact rate (mg/cm^2), outdoor, work.	3.5	3.5	Estimate from Hawley (1985) based on adults doing yard
Contact rate (mg/cm^2), indoor, child	0.056	0.06	Estimate from Hawley (1985) assuming dust fall indoors = 20% outdoors, with cleaning every two weeks.
Contact rate (mg/cm^2), indoor, older child	0.056	0.06	Estimate from Hawley (1985) assuming dust fall indoors = 20% outdoors, with cleaning every two weeks.
Contact rate (mg/cm^2): - indoor, adult, living space - indoor, adult, attic	0.056 1.8 (110 mg/hr)	0.06	Contact rate for adults in living space same as for children; for work in attic value of 1.8 mg/cm^2 for direct contact; for indirect contact with dusty air, contact estimated as 110 mg for 1 hour of attic work (Hawley 1985).
Ratio of contaminant concentration indoors to soil concentration outdoors	0.80	0.85	Hawley (1985).
EXPOSURE			
Time spent outdoors, child (hr)	1,565	2,190	Typical estimate: assumes 5 days/wk, 6 months/yr, 12 hrs/day (12 hrs before soil is washed off). MEI: 7 days/wk, 6 months/yr, 12 hrs/day soil contact (Hawley 1985).

Table 5-18. (continued)

Input parameter	Estimate for Typical individual	Estimate for MEI individual	Notes/explanation/(reference)
EXPOSURE			
Time spent outdoors, older child (hr)	1,824	2,190	Typical: child outdoors from May to September every day (150 days), 12 hrs of soil contact before washing. MEI: child outdoors every day for 6 months, 12 hrs soil contact (Hawley 1985).
Time spent outdoors, adult (hr)	1,565	1,565	Assumes 12 hrs soil contact before washing, 5 days/wk, 6 months/yr (130 days) (Hawley 1985; Keenan et al. 1989).
Time spent indoors, child (hr)	4,380	6,570	Typical: 12 hrs/day of indoor dust contact all year. MEI: assumes 24 hrs/day of indoor dust contact for 6 winter months, plus 12 hrs/day for 6 summer months (Hawley 1985; Keenan et al. 1989).
Time spent indoors, older child (hr)	1,460	4,380	Typical: assumes 4 hrs/day of indoor dust contact all year. MEI: assumes 12 hrs/day of indoor dust contact all year (Hawley 1985).
Time spent indoors, adult (hr), living space, attic	4,380 48	6,570 72	Living space: low and best estimate assumes indoor dust contact 12 hrs/day, all year; high assumes indoor dust contact 24 hrs/day, 6 winter months, plus 12 hrs/day, 6 summer months. Attic: low assumes 1 day in attic with soil left on skin for 12 hrs while in attic plus 4 hrs before washing; best estimate assumes 12 days with soil on skin 4 hrs/day; high assumes 12 days with soil on skin 6 hrs/day (Hawley 1985).
Area of skin exposed outdoor, child (cm^2)	2,100 cm^2	2,800 cm^2	Low assumes only hands exposed (child wearing pants and long-sleeved shirt). Best estimate assumes both hands, legs, and feet exposed during play. High assumes both hands, arms, legs, and feet exposed (Hawley 1985).

Table 5-18. (continued)

Input parameter	Estimate for Typical individual	Estimate for MEI individual	Notes/explanation/(reference)
EXPOSURE			
Area of skin exposed outdoor, older child (cm^2)	1,600 cm^2	3,200 cm^2	Typical: estimate assumes both hands, forearms, legs from knees down exposed during play. MEI: assumes both hands, legs, and feet exposed during play (Hawley 1985; Keenan et al. 1989).
Area of skin exposed outdoor, adult (cm^2)	1,700 cm^2	2,940 cm^2	Typical: assumes both hands, most of forearms exposed. MEI: assumes adult is wearing short-sleeved shirt, with an open neck, pants, shoes, no gloves or hat (Hawley 1985; Schaum 1984).
Area of skin exposed indoor, child (cm^2)	500 cm^2	2,800 cm^2	Typical: estimate assumes area exposed equals one half of surface area of child's feet, hands, and forearms. MEI: assumes all of area of feet, hands, and forearms (Hawley 1985).
Area of skin exposed indoor, older child (cm^2)	400 cm^2	3,200 cm^2	Typical: estimate assume only hands are exposed. MEI: assumes area exposed indoors same as outdoors (Hawley 1985).
Area of skin exposed indoor, adult (cm^2)	1,700 cm^2 (attic) 900 cm^2 (living space)	2,940 cm^2 in both attic and living space	Typical: estimate assumes hands exposed in living space, while adult wears short-sleeved shirt, with an open neck, pants, shoes, no gloves or hat, to work in attic. MEI: assumes area exposed indoors same as outdoors (Hawley 1985).
Bioavailability through skin	0.012 h^{-1}	0.024 h^{-1} (children only)	Typical: data from Appendix A.1. MEI: assumed that skin of children has twice the absorption of adults (Hawley 1985).
Availability of dioxin from soil matrix	1%	15%	Recommended value: 1% (Appendix A.1).

The mass of the contaminant added was calculated by multiplying the application rate by the contaminant concentration in the sludge. The volume of soil with which the sludge was mixed was determined by multiplying the incorporation depth by the incorporation area. The volume was then multiplied by the soil density to obtain the mass of the soil with which the sludge was incorporated. Average soil concentrations for a given year were estimated by adding the mass of contaminant applied to the land for that specific year to the mass present from previous applications and then dividing by the mass of the receiving soil plus the mass of the applied sludge.

(b) Estimates of exposures and risks. The indoor dust and outdoor soil contaminant concentrations were used to estimate exposure and risk from dermal contact with these media. Daily doses were estimated for three age groups: young children (ages 1-6), older children (ages 7-11), and adults (ages 12 and older). The dose for each age group was calculated as:

$$DOSE_g = [[(C_o)(CR_{o,g})(SA_{out,g})(H_{o,g})(AB_d)(M)] + [(C_{in})(CR_{in,g})(SA_{in,g})(H_{i,g})(AB_d)(M)]] / BW_g \quad (5\text{-}10)$$

where:

- AB_d = systemic absorption rate through the skin
- BW_g = body weight = 70 kg for adult, 16 kg for young child, 35 kg for older child
- C_o = concentration of 2,3,7,8-TCDD or 2,3,7,8-TCDF in soil outdoors, mg/mg
- C_{in} = concentration of 2,3,7,8-TCDD or 2,3,7,8-TCDF in dust indoors, mg/mg
- $CR_{in,g}$ = contact rate of soil with skin for age group g, indoors, mg/cm^2
- $CR_{o,g}$ = contact rate of soil with skin for age group g, outdoors, mg/cm^2
- $DOSE_g$ = dose from outdoor exposure for age group g, mg/kg/day
- $H_{i,g}$ = hours spent indoors for age group g
- $H_{o,g}$ = hours spent outdoors for age group g
- $SA_{in,g}$ = surface area of skin exposed to soil for age group g, indoors, cm^2
- M = fraction ot 2,3,7,8-TCDD or 2,3,7,8-TCDF that migrates from the soil matrix and contacts skin
- $SA_{out,g}$ = surface area of skin exposed to soil for age group g, outdoors, cm^2

For each age group, the soil contaminant concentration (expressed in mg/mg for ease of calculation) was multiplied by the soil contact rate outdoors (mg/cm^2) and by the area of the skin exposed during outdoor

activity (cm^2) to obtain the total quantity of soil-bound 2,3,7,8-TCDD or 2,3,7,8-TCDF adhering to the skin (mg). The quantity of contaminant on the skin was then adjusted by two factors: the fraction of the contaminant that migrates from the soil matrix and comes into contact with the skin; and the fraction of 2,3,7,8-TCDD or 2,3,7,8-TCDF that absorbs through the skin. Since the dermal absorption rate was expressed as the fraction of 2,3,7,8-TCDD or 2,3,7,8-TCDF that is absorbed through the skin per hour of contact, it must be multiplied by the hours that the soil was assumed to be in contact with the skin. The same calculations were also performed for exposures in indoor settings, using the corresponding indoor values for the model input parameters. The total daily dermal absorption of 2,3,7,8-TCDD or 2,3,7,8-TCDF was the sum of indoor absorption and outdoor absorption. Dividing the total dermal absorption for each age group by the body weight for that age yielded a daily dose of 2,3,7,8-TCDD or 2,3,7,8-TCDF through dermal absorption in mg/kg/day.

To obtain the weighted average dose over the lifetime of an individual, the following calculation was used:

$$DOSE_{avg} = \sum (FRAC_g)(DOSE_g) \qquad (5\text{-}11)$$

where:

$DOSE_{avg}$ = weighted average daily dose for an individual, mg/kg/day
$DOSE_g$ = daily dose for individual in age group g
$FRAC_g$ = fraction of lifetime spent in age group g

Once the daily dose estimate was obtained, it was combined with information about the potency of 2,3,7,8-TCDD and 2,3,7,8-TCDF to obtain an estimate of lifetime risk from dermal exposure to these contaminants. Maximum and average individual risks were calculated as:

$$IC = (DOSE_{avg})(q_1^*) \qquad (5\text{-}12)$$

where:

$DOSE_{avg}$ = weighted average daily dose for an individual, pg/kg/day
IC = individual cancer risk over lifetime from $DOSE_{avg}$ of 2,3,7,8-TCDD or 2,3,7,8-TCDF
q_1^* = incremental lifetime risk per pg/kg/day dose of 2,3,7,8-TCDD or 2,3,7,8-TCDF

Individual cancer risk for an average exposed individual was converted to annual total population risk (in cases per year) by multiplying the number of persons exposed by the individual risk and dividing by the average person's lifespan, as described in the following equation:

$$PC = \frac{(IC)(POP)}{LS} \qquad (5\text{-}13)$$

where:

LS = average lifespan of an individual = 70 years
PC = population risk, cancer cases per year
POP = population exposed to $DOSE_{avg}$

(c) **Estimates of populations exposed.** The population exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF through dermal contact was limited to the population residing on the agricultural land application sites. The number of sites applying kraft mill sludge to land was equal to the total number of acres applied with sludge in the state divided by the average number of acres per site. Values for both the total acres and the acres per site were obtained through conversations with state officials in Mississippi and Pennsylvania, the states where agricultural land application is currently practiced. In Mississippi, 1,000 acres are applied with the sludge from one mill, with an estimated 100 acres per site, yielding an estimate of 10 sites in Mississippi. Pennsylvania has 50 acres covered with sludge from one mill, with an average of 12 acres per site, giving a total of 4 sites. The total number of sites in each state was then multiplied by the number of people living on each site to obtain the exposed population. According to the 1980 U.S. Census, the average number of persons per household is 2.7. In Pennsylvania, the exposed population is approximately 11 persons, while in Mississippi, the exposed population is approximately 27. The total population exposed was estimated to be 40 persons.

(2) Estimates of exposures and risks from ingestion of produce, meat, and dairy products grown on sludge-amended land. Sludge is applied to various types of land, including forest, abandoned mines, pasture, and land used for the production of animal feed or human food crops. This analysis evaluated the application of sludge to pasture and cropland, and examined the following potential pathways of exposure:

- Sludge is incorporated into the soil of farmland used for producing food crops. Contaminants in the sludge are drawn from the soil to the tissue of those crops, and are then ingested by humans who consume the crops directly.

- Sludge is incorporated into the soil of farmland used for producing animal feed or pasture. Contaminants in the sludge are absorbed into the tissues of these feeds or pasture grasses, which are then consumed by livestock. The meat and dairy products produced by these livestock are consumed by humans.

- Sludge is applied to the surface of pasture land, and adheres to the pasture grasses. Grazing cattle or sheep ingest the sludge directly as a fraction of their pasture consumption. Contaminated beef or dairy products are then consumed by humans.

The concentration of contaminants in food products from land application of paper and pulp mill sludge was estimated using a model that uses information regarding sludge application rates and sludge contaminant concentrations to calculate the uptake of contaminants by crops and by animals feeding on crops and pasture. Exposure was calculated using data on dietary consumption of these meats and crops. For each scenario, the model was given input data for sludge application rates, concentrations of individual contaminants in the sludge, uptake rates of soil contaminants to various crop tissues, uptake rates of contaminants in animal feed to meat or dairy products, the fraction of each type of feed in animal diets, the production yield of animal product per unit of food, human dietary data, the acreage of sludge-amended land devoted to each crop, the productivity of land for each crop, and the population of the distribution area. Values assumed for these parameters that were used to estimate MEI and typical risks are presented in Table 5-19. The model returned exposure estimates which were then used to estimate risk.

Exposure and risk were calculated for both a typical individual and a MEI. The MEI was assumed to be a subsistence farmer applying sludge to all crop types grown on his/her land. Each exposure calculation consisted of three steps. First, the model calculated tissue concentrations of contaminants in each crop as a result of the land application of sludge. Second, the model estimated concentrations of the contaminants in meat or dairy products. Third, the model summed the amount of each contaminant in all crops and animal products ingested to estimate typical exposure or MEI exposure. The estimated soil concentrations for each land application site are presented in Table 5-17.

(a) Methods for estimating soil concentrations. The concentration of 2,3,7,8-TCDD and 2,3,7,8-TCDF in the soil outdoors was derived as described in Section 5.2.4(1)(a).

(b) Methods for estimating tissue concentrations in crops. Each crop's uptake rate was applied to the soil contaminant concentrations to estimate the concentration of each contaminant per unit dry weight of crop tissue. Dry weight tissue concentrations were converted to fresh weight concentrations to match units of human consumption data.

(c) Methods for determining tissue concentrations in meat and dairy products. To calculate meat and dairy product contaminant concentrations, the model derived average concentrations of each contaminant in each animal's feed mix. Average concentrations were calculated by taking a weighted average of the contaminant concentrations

Table 5-19. Assumptions and Parameter Values - Agricultural Application: Dietary Pathway

Input parameter	Estimate for typical individual	Estimate for MEI individual	Notes/explanation (reference)
Percent of cow's consumption that is soil (dry weight)	8%	8%	USEPA (1988b)
Animal product yields:		N.A.	USDA (1987)
lb beef/lb feed	0.07		
lb milk/lb feed	0.97		
lb hog/lb feed	0.17		
lb chicken/lb feed	0.40		
lb catfish/lb feed	0.5		
Percent of product that is fat:			
beef	10	13	Kimbrough et al. (1984); NCASI (1987); Wisconsin DHSS (1989)
milk	4	4	
hog	7	8	
chicken	10	10	
Fish filet concentration as a percent of whole body concentration	50	50	See Section 2 of this report.
Animal consumption (% of total dry weight consumption)			USEPA (1989c); Delta Western (1989)
- corn			
beef	2	2	
dairy	8	8	
hogs	77	77	
chicken	59	59	
- soybeans			
catfish	50	50	
- pasture			
beef	38	38	
dairy	38	38	
Size of population over which produce is distributed	239,000,000	N.A.	Assumes national distribution

Table 5-19. (Continued)

Input parameter	Estimate for typical individual	Estimate for MEI individual	Notes/explanation (reference)
Uptake rates - (dry weight)			See Appendix A.4
wheat	0.02	0.02	
corn	0.02	0.02	
grass	0.02	0.02	
soybeans	0.02	0.02	
Fish BCF	1	10	USEPA (1988b)
Beef fat BCF (fresh weight)	4	6	Fries (1982)
Milk fat BCF (fresh weight)	4	5	Fries (1982)
Hog fat BCF (fresh weight)	4	6	Assumed to be equal to beef fat
Chicken fat BCF (fresh weight)	4	6	Assumed to be equal to beef fat
% dry matter			FDA "Reanalysis of FDA Revised Total Diet Food List" as cited in USEPA (1987)
grains	0.26	0.26	
soybeans	0.08	0.08	
grass	0.05	0.05	
% home grown	N.A.		USEPA (1987a)
grains		2	
legumes		17	
milk		40	
meat		44	
poultry, fish		34	
Consumption (mg/kg/day)	N.A.		Highest values for any age group are used for each food (USEPA 1987b)
wheat		3,154	
corn		1,013	
beef fat		669	
dairy fat		1,424	
hog fat		425	
chicken		796	
soybean oil		1,359	

Table 5-19. (Continued)

Input parameter	Estimate for typical individual	Estimate for MEI individual	Notes/explanation (reference)
Total sluidge area (ha)	435	N.A.	State contacts: Bureau of Waste Management, Wilkesbury, PA, Regional Office; and Bureau of Pollution Control, MS Dept. of Environmental Quality
Amount of beef produced from sludge amended pasture (kgs)	5,084	N.A.	State contacts listed above, County Extension Agent for Perry County, MS, and USDA (1987). Divided Perry and Forest Counties sludge-amended pasture by total pasture in the counties. Applied this percentage to marketed cows for the 2 counties. Multiplied by kilograms per cow.
Crop yields (bushels/ha):		N.A.	USDA (1987)
wheat	94		
corn	222		
soybean	59		
% of area producing:		N.A.	State contacts: Bureau of Pollution Control, Mississippi Dept. of Environmental Quality; County Extension Agent for Perry County, MS and Bureau of Waste Management, Wilkesbury, PA Regional Office
wheat	22%		
corn	33%		
soybeans	36%		
pasture	9%		
% wheat to animals	0	N.A.	County Extension Agency for Perry County, MS and Bureau of Waste Mangement, Wilkesbury, PA Regional Office
% corn to animals	92%	N.A.	
% soybeans to animals	40%	N.A.	
% of animal corn fed to:		N.A.	County Extension Agent for Perry County, MS: Harrisburg, PA Dept. of Agriculture; and USDA (1987). MS: State contact said there are not commercial dairy cows though farmers may have them for own use. From agriculture statistics, multiplied beefs, hogs, and chickens in the state by the anount they each consumed. Summed to get total consumption and then took %'s consumed by beefs, hogs, chickens. PA: 100% of corn is assumed to be fed to dairy cows.
beef cows	74%		
hogs	3%		
chickens	1%		
dairy cows	22%		

Table 5-19. (Continued)

Input parameter	Estimate for typical individual	Estimate for MEI individual	Notes/explanation (reference)
% of animal soybeans fed to catfish	100	N.A.	The MS Perry County extension agent reports that approximately 50% of soybeans fed to animals serve as catfish feed. The remainder are fed to hogs, broilers and laying hens. In the absence of information on quantities fed to each animal and on the animal yield per unit soybean feed, all soybeans fed to animals are assumed to be fed to catfish.

N.A. = Not applicable.

in each animal's food sources. An additional source of contaminant, direct soil ingestion, was added to the dose derived from food. The feed contaminant concentrations were multiplied by animal bioconcentration factors to determine fresh weight concentrations of contaminants in each meat or dairy product.

(d) Estimates of exposures and risks (MEI). Methods of calculating MEI exposure and population exposure differed. The calculation for MEI exposure proceeded in three steps. First, exposure through direct consumption of crops was calculated. To obtain the exposure from direct crop consumption, the daily dietary consumption of each crop was multiplied by the fraction of that crop produced in sludge amended soil, by the fresh weight contaminant concentration of the crop, and by the bioavailability of the pollutant when consumed with the crop:

$$D_c = \sum_i [(CW_{ij})(FC_i)(DC_i)(B_i)(10^{-6})] \quad (5\text{-}14)$$

where:

- B_i = Bioavailability of pollutant when consumed in crop i (unitless); assumed to be 1.
- CW_{ij} = Tissue concentration of pollutant j in crop i (mg/kg fresh weight),
- D_c = Dose of pollutant j from crops produced with sludge-amended soil (mg/kg/day),
- DC_i = Daily dietary consumption of crop i (mg/kg/day fresh weight),
- FC_i = Fraction of dietary consumption of crop i grown in sludge-amended soil (unitless)

In the second step, the MEI dose of the contaminant through consumption of animals raised on contaminated feed was determined. The equations were similar to those discussed for dose from crop consumption:

$$D_a = \sum_k [(CW_{jk})(FC_k)(DC_k)(B_k)(10^{-6})] \quad (5\text{-}15)$$

where:

- B_k = Bioavailability of pollutant when consumed in meat or dairy product k (unitless); assumed to be 1.
- CW_{jk} = Tissue concentration of pollutant j in meat or dairy product k (mg/kg fresh weight),
- D_a = Exposure to pollutant j from animals produced with sludge-amended soil (mg/kg/day),
- DC_k = Daily dietary consumption of meat or dairy product k (mg/kg/day fresh weight),
- FC_k = Fraction of dietary consumption of animal product k produced from sludge-amended soil (unitless).

In the third step, doses from crop and animal consumption were summed:

$$D_j = D_c + D_a \quad (5\text{-}16)$$

where:

D_j = Total exposure to pollutant j from crops, meat and dairy products produced with sludge-amended soil (mg/kg/day).

Once the daily dose estimate to the MEI was obtained, it was combined with information about the potency of 2,3,7,8-TCDD and 2,3,7,8-TCDF to obtain an estimate of lifetime risk from dietary exposure to these contaminants. The calculation of maximum and average individual risks was:

$$IC = (DOSE_{avg})(q_1^*) \quad (5\text{-}17)$$

where:

$DOSE_{avg}$ = weighted average daily dose for an individual, pg/kg/day
IC = individual cancer risk over lifetime from $DOSE_{avg}$ of 2,3,7,8-TCDD or 2,3,7,8-TCDF
q_1^* = incremental lifetime risk per pg/kg/day dose of 2,3,7,8-TCDD or 2,3,7,8-TCDF

(e) Estimates of exposures and risks (population). Population contaminant dose was calculated for each state in which sludge was applied agriculturally and then summed over these states. The dose from the dietary pathway was calculated by summing three inputs: dose of contaminant bioavailable in crops for direct human consumption; dose of contaminant bioavailable in animal products contaminated by crop consumption; and dose of contaminant bioavailable in animal products contaminated by grazing. The average population dose was calculated as:

$$TD_j = (D_j + A_j + G_j)/BW/DP/DY \quad (5\text{-}18)$$

where:

A_j = Dose of pollutant j in animal products contaminated by crop consumption (mg/year)
DP = Population over which the crops and animal products are distributed
BW = Body weight (kg)
DY = Days per year
D_j = Dose of pollutant j in crops for direct human consumption (mg/year)

G_j = Dose of pollutant j in animal products contaminated by grazing (includes soil adherence and grass uptake) (mg/year)

TD_j = Total exposure to pollutant j from crops, meat and dairy products produced with sludge-amended soil (mg/kg/day)

The following discussion describes the methods used to obtain each of the three components of the estimation of total dose.

(i) Method for determining population contaminant dose from ingestion of crops. The dose of contaminant bioavailable in crops for direct human consumption was calculated by multiplying the mass of each crop grown on sludge-amended land that was directly consumed by the tissue concentration of the crop. The result was multiplied by a bioavailability factor:

$$D_j = (MH_i)(CW_{ij})(Bi) \quad (5\text{-}19)$$

where:

B_i = Bioavailability of pollutant when consumed in crop i (unitless); assumed to be 1.

CW_{ij} = Tissue concentration of pollutant j in crop i (mg/kg fresh weight),

MH_i = Mass of crop i grown on sludge-amended land that is consumed directly by humans (kg fresh weight/year).

The mass of each crop grown on sludge-amended land that was consumed directly was calculated by multiplying total acres of the crop receiving sludge by the crop yield. This mass was then multiplied by the percent of the crop consumed directly:

$$M_i = (A_i)(Y_i) \quad (5\text{-}20)$$

where:

A_i = Sludge-amended land area on which crop i is grown (hectares/year),

Y_i = Yield per area of crop i (kg/hectare),

$$MH_i = (M_i)(PH_i) \quad (5\text{-}21)$$

where:

M_i = Mass of crop i grown on sludge-amended land (kg/year),

PH_i = Percent of crop i consumed directly by humans (unitless).

(ii) Method for determining population contaminant dose from ingestion of animals fed on contaminated crops. To determine the dose of contaminant available through consumption of animal products produced from sludge-amended land, the mass of each contaminated crop fed to animals was multiplied by an animal product yield from each kilogram of contaminated feed adjusted by dividing by the fraction of animal diet that the crop constitutes. To obtain a dose to humans who ingest the animal products, this yield was multiplied by contaminant concentration in the animal tissue, percent of the animal that is fat, and a bioavailability factor:

$$A_j = \sum_{ik}[(MA_{ik})(YA_k/FA_{ki})(CW_{jk})(B_k)(PF_k)] \quad (5\text{-}22)$$

where:

B_k = Bioavailability of pollutant when consumed in meat or dairy product k (unitless); assumed to be 1.
CW_{jk} = Tissue concentration of pollutant j in meat or dairy product k (mg/kg fresh weight),
FA_{ki} = Fraction of dietary consumption of crop i for animal k (unitless),
MA_{ik} = Mass of crop i grown on sludge-amended land that is fed to animal k (kg/year),
PF_k = Percent fat in animal product k (unitless),
YA_k = Yield of animal k per unit of corn-equivalent feed (kg/kg).

To obtain the mass of each crop fed to each animal the mass of each crop fed to all animals was divided between the animals according to each animal's percentage of total consumption:

$$MA_i = M_i - MH_i \quad (5\text{-}23)$$

where:

MA_i = Mass of crop i grown on sludge-amended land that is fed to animals (kg fresh weight/year)

$$MA_{ik} = MA_i\ [(N_k)(C_k) / \sum_k[(N_k)(C_k)]] \quad (5\text{-}24)$$

where:

C_k = Food consumed per animal k (kg/year)
MA_i = Mass of crop i grown on sludge-amended land that is fed to animals (kg fresh weight/year)
N_k = Number of animal k in the state

(iii) Method for determining population contaminant dose from ingestion of animals grazing on contaminated pasture land. To obtain the dose of contaminant from animal products raised on sludge-amended pasture land, the number of contaminated animals marketed was multiplied by an average production weight to yield a total mass of meat available for consumption. This mass was then multiplied by the tissue contaminant concentration, the percent of the animal that is fat, and a bioavailability factor:

$$G_j = \sum_g [(NS_g)(WH_g)(CW_{jk})(B_k)(PF_k)] \quad (5\text{-}25)$$

where:

NS_g = Number of each animal, g, grown on sludge-amended land and marketed (animals/year),

WH_g = Average U.S. production of grazing animal g per head marketed (kg/animal).

The number of each type of animal raised on sludge-amended land was the product of the percentage of pasture land that receives contaminated sludge and the total number of animals grazed on pasture land. The resulting number of animals was then multiplied by the percent of grazed animals that are marketed each year:

$$NS_g = (SP_c / P_c)(N_g)(PM_g) \quad (5\text{-}26)$$

where:

N_g = Number of grazing animals (g) in the counties with sludge-amended pasture,

P_c = Total pasture land in all counties with sludge-amended pasture land in state (hectares),

PM_g = Percent of grazing animals (g) marketed per year on a national basis,

SP_c = Sludge-amended pasture land in all counties in state applying sludge agriculturally (hectares).

Once the daily dose estimate was obtained, it was combined with information about the potency of 2,3,7,8-TCDD and 2,3,7,8-TCDF to obtain an estimate of lifetime risk from dietary exposure to these contaminants. The maximum and average individual risks were calculated as:

$$IC = (DOSE_{avg})(q_1^*) \quad (5\text{-}27)$$

where:

$DOSE_{avg}$ = weighted average daily dose for an individual, pg/kg/day
IC = individual cancer risk over lifetime from $DOSE_{avg}$ of 2,3,7,8-TCDD or 2,3,7,8-TCDF
q_1^* = incremental lifetime risk per pg/kg/day dose of 2,3,7,8-TCDD or 2,3,7,8-TCDF

Individual cancer risk for an average exposed individual was converted to annual total population risk (in cases per year) by multiplying the number of persons exposed to the individual risk and dividing by the average person's lifespan, as described in the following equation:

$$PC = [(IC)(POP)] / LS \quad (5\text{-}28)$$

where:

LS = average lifespan of an individual = 70 years
PC = population risk, cancer cases per year
POP = population exposed to $DOSE_{avg}$

(f) Estimates of populations exposed. To determine typical exposures, total available quantity of contaminated food was assumed to be distributed nationally. This assumption does not affect population risk since the quantity of contaminated food is determined independently of the population exposed and risk is assumed to be a linear function of exposure.

(3) Estimates of exposures and risks from direct ingestion of sludge. Direct ingestion of soil can occur when sludge is applied to sites where people may live and work, such as a family farm. This analysis assumes that both the typical and MEI individual consume an average of 0.1 grams/day of soil over a lifetime. It is assumed that all ingested soil originates from outdoor sources. The values used for each model input are summarized in Table 5-20. Average soil concentrations are presented in Table 5-17.

(a) Methods for estimating soil and indoor dust concentrations. The concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF in outdoor soil were estimated as described in Section 5.2.4(1)(a).

(b) Estimates of human exposures and risks. Exposures and risks were calculated as follows. The daily dose was calculated as:

$$DOSE_g = [(C_o)(DC_g)] (AB_{gi}) / BW_g \quad (5\text{-}29)$$

Table 5-20. Assumptions and Parameter Values - Land Application: Soil Ingestion Pathway

Input parameter	Estimate for Typical individual	Estimate for MEI individual	Notes/explanation/(reference)
EXPOSURE			
Amount soil ingested (g/day)	0.1	0.1	
Percent of daily ingestion from outdoors	100%	100%	
Fraction of ingested soil that is from sludge-amended land	0.10	1.0	

where:

AB_{gi} = systemic absorption rate from gastrointestinal tract; assumed to be 1
BW_g = body weight of individual
C_g = concentration of contaminant in soil, mg/kg
DC_g = daily soil ingestion rate
$DOSE_g$ = daily dose to individual (mg/kg/day)

Individual and population risks were calculated using Equations 5-12 and 5-13, respectively.

(c) Estimates of populations exposed. The population exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF through ingestion was limited to the population residing on the agricultural land application sites as discussed in Section 5.4.2.(1)(d).

(4) Estimates of exposures and risks from inhalation of sludge-contaminated particulates. 2,3,7,8-TCDD and 2,3,7,8-TCDF adhering to soil particles can become suspended in the air near a land application site. Transport downwind will dilute the concentration of particles from a land application site; these particles will also re-deposit on surfaces. Residents living on or near the land application sites may be exposed to 2,3,7,8-TCDD or 2,3,7,8-TCDF by inhaling these particles. This section describes the methods used to estimate the emissions of particles from a land application site and the subsequent human exposure to these emissions. This analysis only considered exposure to inhaled particulates for residents onsite. The data inputs for the particle inhalation model are presented in Table 5-21 except for respiration rate. This parameter is discussed below in Section 5.2.4(4)(c). The estimated soil concentrations are presented in Table 5-17.

The estimation of risks from inhalation of particles required several steps. First, the emissions of particles from the treated area was estimated. Next, the indoor and outdoor concentrations of particles onsite were calculated. The concentrations were combined with information about the length of time spent indoors and outdoors, respiratory rate, and the slope factor of 2,3,7,8-TCDD and 2,3,7,8-TCDF to yield the estimated cancer risks.

(a) Methods for estimating soil and indoor dust concentrations. The concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF in outdoor soil were estimated as described in Section 5.2.4(1)(a).

(b) Methods for estimating emissions and particulate concentrations. The method used for estimating emissions due to wind erosion assumed that the uncrusted contaminated surface is exposed to the wind and consists of finely divided particles. This created a condition that

Table 5-21. Assumptions and Parameter Values - Land Application: Vapor and Particulate Inhalation Pathways

Input parameter	Estimate for Typical individual	Estimate for MEI individual	Notes/explanation/(reference)
CONCENTRATION			
Method for calculating particulate emissions	Assume 50% vegetative cover	Assume 50% of total TSP from local soil	Typical: calculated assuming 50% vegetation. MEI: assume 50% of total TSP from local soil; local TSP = 64 ug/m^3 (Hawley 1985).
Indoor TSP concentration as function of outdoor TSP concentration	0.75	0.85	Hawley (1985).
EXPOSURE			
Percent TSP respirable	N/A	0.90	Typical: estimate for emissions is from information that gives estimates of small (<10 um) particle emissions. All of these particles are assumed to be respired. The range given here applies only to TSP, which consists of large and small particles (i.e., this range applies only to the MEI estimate of emissions).
Fraction of TSP inhaled to lung	1/3 of respired TSP	0.90 (100% of respired TSP)	Typical: one-third of total TSP is initially found in lungs; MEI: 100% (Schaum 1984).
Fraction of TSP inhaled to GI tract	2/3 of respired TSP	0%	
Absorption rate, lung	1.0	1.0	Value from bioavailability is applied to the particles that reach the alveoli.
Hours per day and number of days spent outdoors, adult	12 hrs/day 130 days	12 hrs/day 130 days	Assumes outdoor work for 6 months, 5 days/wk, 12 hrs/day (Hawley 1985).

Table 5-21. (continued)

Input parameter	Estimate for Typical individual	Estimate for MEI individual	Notes/explanation/(reference)
EXPOSURE			
Hours per day and number of days spent outdoors, child	8 hrs/day 130 days	12 hrs/day 182 days	Typical: assumes child plays outdoors 8 hrs/day, May-October, 5 days per wk. MEI: assumes child plays outdoors 12 hrs/day, May-October, 7 days per wk.
Hours per day and number of days spent outdoors, older child	5 hrs/day 152 days	12 hrs/day 152 days	Typical: assumes child plays outdoors an average of 5 hrs/day, May-September. MEI: assumes 12 hrs/day, May-September (Hawley 1985).
Hours per day and number of days spent indoors, adult, attic, living space	12 hrs/yr in attic; 12 hrs/ day, 130 days plus 24 hrs/day, 235 days, farmer	12 hrs/yr in attic; 12 hrs/ day, 130 days plus 24 hrs/ day 235 days	Attic: assumed that adult spends 12 hrs/yr (either 1 day for 12 hrs or 1 hr for 12 days) in attic. Living space: 12 hrs/day in living space for 130 days in summer months plus 24 hrs/day in living space for rest of year (Hawley 1985).
Hours per day and number of days spent indoors, child	16 hrs/day, 130 days, plus 24 hrs/day, 235 days	12 hrs/day, 182 days, plus 24 hrs/day, 182 days	MEI: assumes child plays indoors 12 hrs per day, 7 days per wk during May-October, rest of year indoors 24 hrs per day. Typical: assumes indoors 16 hrs/day, 5 days/wk during May-October, indoors 24 hrs/day for remainder of days.
Hours per day and number of days spent indoors, older child	16 hrs/day for 365 days	12 hrs/day, 152 days plus 24 hrs/day for 213 days	Typical: assumes child is indoors 16 hrs/day. MEI: assumes child is indoors an average 12 hrs/day from May-September, and is indoors for the rest of the year 24 hrs/day (Hawley 1985).
CONCENTRAITON			
Indoor contaminant concentration as function of outdoor contaminant concentration	indoor = 80% outdoor	indoor = 100% outdoor	Typical: from Hawley (1985). MEI: estimate is maximum value possible.

results in maximum wind-caused dust emissions. The model for estimating particulate concentration incorporated information on wind speed and percent vegetation cover to estimate the flux of small particles (i.e., less that 10 μm) from an area of land. Soil amended with paper mill sludge may not have the characteristics assumed by the model; to the extent that the surface of a sludge-amended site consists of coarser particles, the model was likely to overestimate emissions.

To estimate particulate concentration, the calculated emission rate was used as input to a box model of atmospheric mixing. The box model ignored any atmospheric dispersion downwind, and was only appropriate for estimating onsite concentrations. The model used wind speed, size of the site and the mixing height to yield an onsite particulate concentration. As an alternative approach to estimating onsite particulate concentration, another model, which used measured values of total suspended particles adjusted by the fraction of particles assumed to be derived from local soils, was applied.

The indoor suspended particle concentration was derived by applying the ratio of suspended particulate concentration indoors to the suspended particulate concentration outdoors. Since only a portion of indoor dust was assumed to originate from outdoor sources, the contaminant concentration in indoor dust was adjusted by a fraction representing the ratio of indoor dust contaminant concentration to the outdoor soil contaminant concentration.

(c) Estimates of exposures and risks. Once the concentration of contaminants in particulates was estimated, exposure to contaminated particulates was calculated. In the MEI estimate of risk scenario, the particulate concentration was estimated based on total particulates. When calculating exposure from this estimate of particulate concentration, the first step was to determine the concentration of particles that were respirable. In the typical estimate scenarios, all of the emissions were assumed to be respirable.

The next step in the estimation of human exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF through the inhalation of particulates was the estimation of the daily dose. The daily dose was calculated for three age groups: young children (ages 1-6), older children (ages 7-11), and adults (ages 12 and older).

$$DOSE_{o,g} = \frac{[[(RC_o)(D_l)(AB_l)(H_{o,g})] + [(RC_g)(D_{gi})(AB_{gi})(H_{o,g})]]\,V_g}{BW_g} \quad (5\text{-}30)$$

where:

AB_l = systemic absorption rate through the lung; assumed to be 1.0.
AB_{gi} = systemic absorption rate through the gastrointestinal tract; assumed to be 1.0.
BW_g = body weight of individual in age group g
D_l = fraction of respired particles retained by the lung
D_{gi} = fraction of respired particles swallowed (fraction of particles to gastrointestinal tract)
$DOSE_{o,g}$ = dose to individual in age group g, outdoors, mg/kg/day
$H_{o,g}$ = hours spent outdoors for individual in age group g
RC_o = respirable particulate concentration outdoors, mg/m^3
V_g = weighted average ventilation rate for individual in age group g, m^3/day

(5-31)

$$DOSE_{i,g} = \frac{[[(RC_{in})(D_l)(AB_l)(H_{i,g})] + [(RC_{in})(D_{gi})(AB_{gi})(H_{i,g})]]\,V_g}{BW_g}$$

where:

$DOSE_{i,g}$ = dose to individual in age group g, indoors, mg/kg/day
$H_{i,g}$ = hours spent indoors for individual in age group g
RC_{in} = respirable particulate concentration indoors, mg/m^3

In this equation, the concentration of the contaminant adhering to particles was multiplied by the volume of air inhaled each day and by the fraction of the day spent outdoors. Similarly, the quantity of particulates inhaled indoors each day was the product of the indoor respirable concentration, the volume of air inhaled each day, and the fraction of the day spent indoors. The total quantity of particles inhaled each day was then partitioned between the lung and the gastrointestinal tract. A gastrointestinal absorption fraction was then applied to the portion swallowed, while a respiratory absorption fraction was applied to the portion remaining in the lung.

A weighted average dose for an individual over the entire lifetime was derived by weighting the daily dose received during each age interval by the fraction of the individual's lifespan spent in that age group. This calculation is described in the following equation:

$$DOSE_{avg} = \sum (DOSE_{o,g} + DOSE_{i,g})(F_g) \qquad (5\text{-}32)$$

where:

$DOSE_{avg}$ = weighted average daily dose over lifetime, mg/kg/day
$DOSE_{o,g}$ = dose to individual in age group g, outdoors, mg/kg/day
$DOSE_{i,g}$ = dose to individual in age group g, indoors, mg/kg/day
F_g = fraction of lifespan spent in age group g

Individual and population risks were calculated using Equations 5-12 and 5-13, reespectively.

(d) Data sources and model inputs for respiration rate. Respiration rate was used in the model to assess the total daily volume of particles inhaled. For adults, the average respiration rate was calculated to be 23 m^3 per day. This value was calculated using data on the ventilation rates during different levels of activity, and the amount of time spent per day engaging in these levels of activity, to obtain a daily total. The ventilation rate of young children engaged in light activity is 7.6 l/min, while the ventilation rate during rest is 2.8 l/min; assuming children spend roughly one-third of their day engaged in light activity and two-thirds at rest, the total ventilation rate is 6.3 m^3 per day. For older children, the ventilation rate is 11.6 l/min during light activity and 4.3 l/min at rest, with a total ventilation rate of 8.4 m^3 per day.

(e) Estimates of populations exposed. The population exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF through inhalation of particulates was limited to the population residing on the agricultural land application sites as discussed in Section 5.4.2(1)(d).

(5) Estimates of exposures and risks from inhalation of vapors. Residents of land application sites may incur risk from the inhalation of volatilized 2,3,7,8-TCDD and 2,3,7,8-TCDF. Because actual locations of the land application sites were not known, the ISCLT model was not used to estimate concentrations downwind from such sites. As a result, this analysis estimated only exposures to onsite residents, using an atmospheric box model to obtain the onsite concentrations from the estimated emissions.

The calculation of risks from the inhalation of vaporized 2,3,7,8-TCDD or 2,3,7,8-TCDF required the estimation of emissions, and the calculation of indoor and outdoor onsite concentrations. The concentrations were combined with data on time spent indoors and outdoors, respiration rate and slope factor of 2,3,7,8-TCDD and 2,3,7,8-TCDF to obtain the estimated cancer risk from this pathway of exposure. Table 5-21 summarizes key assumptions and input parameters for estimating exposure through the vapor inhalation pathway.

(a) Methods for estimating vapor emissions. Information on the partitioning of 2,3,7,8-TCDD and 2,3,7,8-TCDF between soil and air

and between water and soil was used to estimate emissions of 2,3,7,8-TCDD and 2,3,7,8-TCDF vapor per m^2 area. The emissions estimate was then multiplied by the area of the site, in m^2, to obtain the total emissions of vapor from the land application site. The emission rate was then coupled with a box model to obtain the onsite concentrations of vapor.

(b) Methods for estimating vapor concentrations. The indoor vapor concentration was derived by applying the ratio of vapor concentration indoors to the vapor concentration outdoors. It was assumed that the relationship between vapor concentrations indoors and outdoors was similar to the ratio between indoor and outdoor particulate concentrations (that is, indoor concentrations are approximately 75 percent of outdoor concentrations with a range from 70 to 85 percent).

(c) Estimates of exposures and risks. Once the concentration of contaminant in the air was estimated, the calculation of exposure and risks from the inhalation of vapor then proceeded in the same manner as the exposure and risk from the inhalation of particles, described in Section 5.2.4(4). In some cases, the data inputs used for the estimation of exposure and risk were different than those used in Section 5.2.4(4).

The emissions model required the soil/water partition coefficient as one input. This partition coefficient was, in turn, based on the fraction of organic carbon (f_c) in the soil. For land application sites where sludge is soil-incorporated, it was assumed that the fraction of organic carbon in the sludge-soil mixture was approximately equal to the fraction of organic carbon in the soil alone. Estimates of f_c for soils of 1 percent and 4 percent were assumed for typical and MEI estimates of exposure, respectively. The organic carbon content of sludge has been reported to range from 14 to 40 percent. Estimates of f_c for sludge of 25 percent and 40 percent were assumed for typical and MEI estimates of exposure, respectively.

The data inputs and model sources for the vapor exposure estimate were the same as those described in Section 5.2.4(4), with two exceptions. The first exception is that 100 percent of vapor emissions were assumed to be respired. The second exception is that all of the vapor was absorbed through the lung; none was absorbed through the GI tract. This analysis also assumed that all volatilized 2,3,7,8-TCDD and 2,3,7,8-TCDF was completely absorbed into the system when inhaled, due to its high lipophilicity.

(d) Estimates of populations exposed. The population exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF through inhalation of vapors was limited to the population residing on the agricultural land sites as discussed in Section 5.4.2(1)(d).

(6) Estimates of exposures and risks from ingestion of ground water. Land application of sludge is not expected to present significant risk to human health through contamination of ground water. Conservative, high risk estimates of ground-water contamination from sludge in industrial landfills yielded risk estimates on the order of 10^{-7} for a most exposed individual; typical estimate assumptions yielded lower risk estimates. Consideration of some major differences between landfilling and land application of pulp and paper sludge suggests that risks of ground-water contamination and health risks from land application would be lower than those estimated for landfills and are therefore too low to justify more detailed evaluation.

Land application and landfill sites differ in at least four important respects: (1) land application sites may be larger than landfills; (2) local geo-hydrological or weather conditions may differ between landfill and land application sites; (3) sludge may be placed in landfills to a significant depth below ground level (land-applied sludge is generally applied to the ground surface, or incorporated into a relatively shallow surface soil layer); and (4) the quantities of sludge applied to a hectare of treated land tend to be much lower than the quantities deposited in a hectare of landfill.

(7) Estimates for exposures and risks from ingestion of drinking water from surface water sources. Where pulp and paper sludge is applied to land, particles of sludge or soil from the surface can be transported by erosion to nearby lakes or streams. If humans consume water or fish from these lakes or streams, they may be exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF from the land applied sludge.

The methodology to estimate exposure consisted of three general steps: (1) based on sludge concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF, local topography, land use and other factors, it estimated contaminant concentrations in sediments, surface water, and fish; (2) it used these estimated concentrations, assumptions about individual ingestion of drinking water, and assumptions about the bioavailability and the slope factor of 2,3,7,8-TCDD and 2,3,7,8-TCDF, to estimate individual health risks for humans potentially exposed; and (3) it combined these results with estimates of the size of exposed populations to derive estimates of total human health risks in the U.S. population. Table 5-22 summarizes the values used for each model input parameter. The application rates and soil incorporation depths for the sites receiving the sludge are displayed in Table 5-17, along with soil and sludge concentrations.

(a) **Methods for estimating surface water concentrations.** The Universal Soil Loss Equation was used along with estimates of sediment delivery ratios, to estimate the fraction of a lake or stream's sediment that originates from the landfill. By multiplying this fraction by the

Table 5-22. Assumption and Parameter Values - Land Application: Surface Water Pathways

Input parameter	Estimate for Typical individual	Estimate for MEI individual	Notes/explanation/(reference)
CONCENTRATION			
"C" ratio			USDA (1978)
Agriculture	4	6	
Forest/mine reclamation	1	1	
"P" ratio			
Agriculture	0.75	1	Typical: estimate assumes contouring; MEI: assumes no support practices.
Forest/mine reclamation	1	1	
Drainage area (ha)	1,295,000	4,047	
Percent organic carbon in soil	0.01	0.001	USEPA (1986b)
Fish to sediment ratio - dioxin	1	10	USEPA (1988b)
Fish to sediment ratio - foran	1	10	USEPA (1988b)
Concentration in filet vs. concentration in whole fish	0.5	0.5	
Sludge-amended area (hectares per yr)			
Agriculture	435	405	From each state's permit data or best estimate.
Forest/mine reclamation	1,805	1,012	
Distance to stream (meters)			
Agriculture	30	30	
Forest/mine reclamation	1,355	46	
EXPOSURE			
Water consumption (liters/day)	2	2	
Population density (per square mile)	68	N/A	USDOC (1987).

Table 5-22. (continued)

Input parameter	Estimate for Typical individual	Estimate for MEI individual	Notes/explanation/(reference)
EXPOSURE (continued)			
Fish consumption (grams/day)	6.5	140	U.S. average; subsistence fisher consumption.
Percent of fish ingested that is contaminated	100%	100%	Assumes that for the population in the drainage area, 100% of freshwater and estuarine fish consumption is from the contaminated source.
Percent of population served by surface water	49%	N/A	USGS (1985)
Percent of water ingested that is contaminated	100%	100%	Assumes that the affected population that is served by surface water consumes 100% contaminated water.

original concentration of 2,3,7,8-TCDD and 2,3,7,8-TCDF in sludge or soil particles on the landfill surface, the methodology derived estimates of the concentration of contaminants in the sediment. This contaminant load was then partitioned between adsorbed and dissolved phases, based on the assumption of equilibrium partitioning between the two phases.

(b) **Estimates of exposures and risks.** Exposures and risks were calculated using Equations 5-5 and 5-6, respectively.

(c) **Estimates of populations exposed.** The population exposed to contaminated water was estimated by multiplying the area of the drainage basin above each sludge management area (SMA) drainage point by the national population density (68 persons/sq. mile) (USDOC 1987). This estimated population was then multiplied by the fraction of the population that takes its drinking water from surface supplies:

$$PE_W = (A_B)(PD)(PSW) \qquad (5\text{-}33)$$

where:

PE_W = Population exposed to contaminated water
A_B = Area of the drainage basin (ha)
PD = Population density (persons/ha)
PSW = Percent of population served by surface water

Only a portion of this population will rely on surface water for their drinking water. Therefore, the estimated population exposed was reduced by multiplying by the average percentage of population served by surface water. Forty-nine percent of the population was assumed to be served by surface water.

(8) <u>Estimates of exposures and risks from ingestion of fish from surface water sources</u>. Where pulp and paper sludge is applied to land, particles of sludge or soil from the surface can be transported by erosion to nearby lakes or streams. If the sludge contains 2,3,7,8-TCDD or 2,3,7,8-TCDF, then those particles can carry these contaminants to the surface water bodies. Fish living in the lakes or streams can take up sludge contaminants into their tissues; if humans then consume those fish, they can be exposed.

The methodology used to estimate exposure and its associated risks began by estimating sediment concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF in water bodies as a result of runoff. The methodology also used fish to sediment bioconcentration factors and estimates of fish consumption contaminant doses. In addition, the methodology involved estimating the sizes of exposed populations, combining these results with estimates of individual dose and health risk to derive total health risks to the entire exposed population. The values used for each model input are summarized in Table 5-22.

(a) **Methods for estimating sediment concentrations.** The method for estimating concentrations used the Universal Soil Loss Equation, together with estimates of sediment delivery ratios, to estimate the fraction of a lake or stream's sediment that originates from the landfill. Estimates of the concentration of contaminants in the sediment were determined by multiplying this fraction by the original concentration of 2,3,7,8-TCDD and 2,3,7,8-TCDF in sludge or soil particles on the surface of the landfill. These sediment concentrations were then used to estimate contaminant concentrations in the tissues of fish.

(b) **Methods for estimating fish tissue concentrations.** The methodology used empirical fish to sediment bioconcentration factors to estimated concentrations of contaminant in freshwater fish as a function of concentrations in stream or lake sediment. This was based on the assumption that sediment concentrations were the best predictor of fish concentrations of hydrophobic compounds like 2,3,7,8-TCDD and 2,3,7,8-TCDF. The concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF in the muscle tissues of fish (consumed by humans) were considered to be fifty percent lower than the whole body concentrations of these contaminants.

(c) **Estimates of exposures and risks.** Exposures and risks were calculated using Equations 5-7 and 5-8, respectively.

(d) **Estimates of exposed populations.** The population exposed to fish containing 2,3,7,8-TCDD and 2,3,7,8-TCDF was estimated by multiplying the area of the drainage basin containing each facility by an estimated population density of the regions containing the SMA's. This estimated population was then multiplied by the fraction of the population that takes its drinking water from surface supplies:

$$PE_W = (A_B)(PD)(PSW) \qquad (5\text{-}34)$$

where:

PE_W = Population exposed to contaminated water
A_B = Area of the drainage basin (ha)
PSW = Percent of population served by surface water
PD = Population density

This analysis assumed that all fish were consumed regionally. An alternative approach would be the assumption that the fish were distributed nationally. Therefore, the percent of the freshwater fish each person consumed from the contaminated stream would be calculated by using the ratio of the drainage area of the contaminated stream to the drainage area of the entire United States. This percentage could be used as the percent of contaminated freshwater fish consumed by the entire U.S. population. However, the current methodology used drainage area to determine the proportion of U.S. citizens who were exposed to contaminated water

and fish. This population was assumed to consume 100 percent of their freshwater fish from the contaminated stream.

The receiving stream for each SMA fill was assumed to be a major stream with a 5,000 square mile watershed area. Another assumption was that the population exposed was positively correlated with stream size and that stream size was positively correlated with drainage area i.e., the larger the drainage area, the more people were likely to receive their drinking water supply from the stream. To quantify this relationship, population exposed was modeled as a function of drainage area. Each unit area of the watershed was multiplied by the average population density for the regions through which the waterways flow to yield population exposed.

The entire exposed population was assumed to ingest fish at concentrations appropriate for the "point" of entry of the SMA runoff into the stream. Since the population exposed inhabited an area of approximately 70 by 70 miles, this assumption was conservative, and tends to overstate exposure and risk. Dilution and dispersion of the contaminant would have occurred before much of the population was exposed.

(9) Summary of results. The risks resulting from exposures associated with the land applications of sludge are summarized in Table 5-23. Typical risk to individuals are low in all pathways analyzed for this disposal/re-use practice. Highest typical risks are estimated for persons living on the land application site, exposed through direct ingestion vapor inhalation, and dermal contact with contaminated soil. The highest typical risk (i.e., 1×10^{-5}) is associated with the vapor inhalation pathway. However, because the size of the population exposed through this pathway is small, the total annual cancer risk resulting from this exposure is estimated to be only 7×10^{-6} cases per year.

The highest MEI risks are from the ingestion of fish caught in contaminated surface water bodies, and from the ingestion of produce, meat, and dairy products grown on sludge-amended land. The MEI is assumed to take fish from a relatively small stream from the location in the stream with the maximum dissolved concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF. For the produce and meat ingestion pathway, the MEI is assumed to be a subsistence farmer who raises his/her own meat and dairy products on sludge-amended agricultural land. The MEI risk associated with the fish ingestion pathway is 1×10^{-1}, while the MEI risk from the ingestion of produce and meat is 1×10^{-2}.

5.2.5 Exposures and Risks from Distribution and Marketing of Pulp and Paper Sludge

Sludge that is composted and marketed can be used as a soil amendment in residential settings as well as for agricultural and commercial

Table 5-23. Estimates of Health Risks to the General Population from Land Application of Pulp and Paper Sludges Contaminated with 2,3,7,8-TCDD and 2,3,7,8-TCDF

Exposure pathway	MEI risk[a] (per lifetime)	Typical risk[a] (per lifetime)	Exposed population	Total risk (cases/year)[b]
Dermal exposure from contact with soil contaminated by land application of sludge:	$5x10^{-5}$	$3x10^{-7}$	40	$2x10^{-7}$
(Percent TCDD)[c]	(62)	(65)		(65)
Exposure from direct ingestion of soil contaminated by land application of sludge:	$5x10^{-5}$	$1x10^{-6}$	40	$6x10^{-7}$
(Percent TCDD)[c]	(62)	(65)		(65)
Inhalation exposure to air contaminated by volatilization from soil contaminated by land application of sludge:	$2x10^{-4}$	$1x10^{-5}$	40	$6x10^{-6}$
(Percent TCDD)[c]	(4)	(4)		(4)
Inhalation exposure to particulates from soil contaminated by land application of sludge:	$5x10^{-6}$	$8x10^{-7}$	40	$5x10^{-7}$
(Percent TCDD)[c]	(62)	(65)		(65)
Ingestion exposure from drinking surface water contaminated by surface runoff from soil contaminated by agricultural land application of sludge:	$2x10^{-3}$	$3x10^{-7}$	333,000	$1x10^{-3}$
(Percent TCDD)[c]	(0.6)	(0.6)		(0.6)
Ingestion exposure from drinking surface water contaminated by surface runoff from soil contaminated by land application of sludge to mines/forests	$3x10^{-3}$	$3x10^{-7}$	833,000	$4x10^{-3}$
(Percent TCDD)[c]	(0.6)	(0.6)		(0.6)
Ingestion exposure from drinking ground water contaminated by leaching from soil contaminated by land application of sludge	$<3x10^{-7}$	$<3x10^{-7}$	NA[d]	NA[d]
(Percent TCDD)[c]	(0.2)	(0.2)		
Ingestion exposure to fish contaminated by surface runoff from areas contaminated by agricultural land application of sludge	$1x10^{-1}$	$5x10^{-7}$	679,000	$5x10^{-3}$
(Percent TCDD)[c]	(63)	(65)		(65)
Ingestion exposure to fish contaminated by surface runoff from areas contaminated by land application of sludge to mines/forests	$2x10^{-1}$	$5x10^{-7}$	1,700,000	$1x10^{-2}$
(Percent TCDD)[c]	(63)	(65)		(65)

Table 5-23. (Continued)

Exposure pathway	MEI risk[a] (per lifetime)	Typical risk[a] (per lifetime)	Exposed population	Total risk (cases/ year)[b]
Dietary exposure from produce grown in soil contaminated by land application of sludge	$1x10^{-2}$	$2x10^{-10}$	240,000,000	$7x10^{-4}$
(Percent TCDD)[c]	(62)	(65)		(65)

[a] Calculated as: [Estimated Dose] x 1.6 x 10^{-4} per (pg/kg)/day.

[b] Calculated as: [Typical Risk x Exposed Population] / [Life Expectancy].

[c] Calculated as: $100 \times \frac{[\text{Exposure to TCDD}]}{[\text{Exposure to TCDD}] + (1/10)[\text{Exposure to TCDF}]}$

[d] Not applicable.

purposes. According to the 104-Mill Study, seven mills in five states distribute and market at least a portion of their sludge. Based on data from the 104-Mill Study, the total volume of sludge distributed and marketed by these plants is estimated to be 208,000 dry metric tons per year. In some cases, the plants in this study reported two methods of sludge disposal, but did not provide a break-down of the quantities of sludge disposed by each method. In these cases, it was assumed the entire quantity of sludge by the plant produced is distributed and marketed. To the extent that this assumption overestimates the quantity of sludge distributed and marketed every year, the population risk estimates derived from this analysis overestimate the true population risk.

This analysis estimates risks to members of households using composted sludge from the following routes of exposure:

- Home gardeners incorporate distributed and marketed sludge into home gardens. The home-grown crops incorporate small amounts of contaminant into their tissues. Household residents then consume the home-grown crops.

- Home gardeners incorporate distributed and marketed sludge into their home gardens, or use it for other home gardening purposes, such as lawns or flower beds. Children and adults in the gardening household come into direct dermal contact with the sludge. 2,3,7,8-TCDD and 2,3,7,8-TCDF from the sludge is absorbed through the skin. Children ingest small amounts of the sludge/soil mixture through normal mouthing behavior. Adults also inadvertently ingest small quantities of sludge/soil.

- 2,3,7,8-TCDD and 2,3,7,8-TCDF in distributed and marketed sludge volatilizes from the sludge into the air. Residents of the household inhale the volatilized 2,3,7,8-TCDD and 2,3,7,8-TCDF.

- Distributed and marketed sludge is applied to home gardens or other home uses. Particles of the sludge/soil mixture become suspended in the air. Members of the household inhale the contaminated particles.

Since the actual users of distributed and marketed sludge are not known, a generic scenario was used to estimate risks from the distribution and marketing of sludge. In this scenario, a household was assumed to use composted sludge as a soil amendment for ornamental or vegetable gardening. The following discussion briefly describes the scenario considered. The estimated soil concentrations used in the scenarios are shown in Table 5-24. Other parameters that describe the generic scenario used in this assessment are presented in Table 5-25.

Table 5-24. Distribution and Marketing Sludge and Soil Contaminant Concentration*

Contaminant	Mean sludge concentration	90th percentile sludge concentration	Estimate for Typical Individual: Soil concentration using mean conc., and assuming 6 inches (15 cm) depth of incorporation	Estimate for MEI: Soil concentration using 90th percentile conc. assuming no soil incorporation
TCDD	163	293	10	293
TCDF	885	1760	55	1760

*Average concentrations over 70 years of exposure, assuming sludge is applied to land for 20 years.

Table 5-25. Parameters and Model Inputs Used to Model: Distribution and Marketing

Input parameter	Estimate for typical individual	Estimate for MEI individual	Notes/explanation/reference)
Percent of sludge to home garden vs. other uses	50%	100%	Typical estimate: based on analysis of National Garden Survey (1987). MEI: maximum value possible.
Soil incorporation assumptions	6 inches	0 inches	USDA (1979)
Garden size (hectares)	0.016	0.022	National Garden Survey (1987)
Application rate (DMT/year)	10	20	USDA (1979)

According to recent data, 34 million of the 69 million households involved in gardening activity in 1986 grew vegetables (National Gardening Survey 1987). Based on these data, this analysis assumed that approximately one-half of the distributed and marketed sludge was used at households with vegetable gardens, while the other half was used at households that use the sludge for ornamental gardening. Furthermore, the analysis assumed that households using sludge for ornamental gardening apply sludge at the same rate as households with vegetable gardens. In the MEI scenario, it was assumed that all of the distributed and marketed sludge was used at households with vegetable gardens.

The concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF in sludge from plants that distribute and market sludge were obtained from the 104-Mill Study. The methods for calculating the soil concentration of 2,3,7,8-TCDD and 2,3,7,8-TCDF in soils amended with the composted sludge were described in Section 5.2.4. The soil concentration model requires inputs for the initial 2,3,7,8-TCDD and 2,3,7,8-TCDF concentrations, length of the application period and the depth of incorporation with background soils. Decay of 2,3,7,8-TCDD and 2,3,7,8-TCDF during the composting process was assumed to be negligible. The analysis assumed that composted sludge is applied to a home garden for 20 years. The home gardener was assumed to continue to use the garden for an additional fifty years.

In the typical scenarios, the analysis assumed that sludge is soil incorporated. For the MEI scenario, it was assumed that sludge is applied only to the top layer of soil. Recommended depths of incorporation for the maintenance of crops range from approximately 6 to 10 inches (USDA 1979). In the typical scenario, this analysis used 6 inches of incorporation. The top-dressing scenario was used to model risks to the MEI.

This analysis assumed that the home gardener applies 10 dry metric tons of sludge per hectare per year to his or her home garden. The recommended application rates for home uses ranges from 5 to 20 dry metric tons per hectare (USDA 1979). The MEI was assumed to apply 20 metric tons per year to his/her home garden.

The size of the garden assumed in the generic scenario affects the individual risk estimates and influences the estimate of the size of the exposed population. The average garden size for combined rural and urban vegetable gardens is 0.016 hectares. For the MEI scenario, the average rural garden size (0.022 hectares) was assumed.

Using the assumptions described above, this analysis estimated exposures from dermal contact, vapor and particulate inhalation, and direct ingestion for young children (ages 1-6), older children (ages 7-12) and for adults, while dietary exposures were estimated for

young children (1-6) and adults. The average daily dose over a lifetime from each of these pathways was the weighted average of the daily doses during these stages of life. These doses were combined with cancer potency estimates to obtain incremental lifetime risk from 2,3,7,8-TCDD and 2,3,7,8-TCDF exposure. The following sections describe the methods and data used to estimate risks from home uses of distributed and marketed sludge through each these pathways. Results are summarized in the final section.

(1) Estimates of exposures and risks from dermal contact with skin. Humans coming in direct contact with sludge contaminated soils may absorb 2,3,7,8-TCDD and 2,3,7,8-TCDF through their skin. The amount of 2,3,7,8-TCDD and 2,3,7,8-TCDF absorbed will depend on the area of skin exposed and on the length of time that the contaminated soil is in contact with the skin. The following discussion summarizes the model used to estimate dose through dermal contact with composted pulp and paper mill sludges used in residential settings.

The model used to estimate dose through direct contact with soil containing 2,3,7,8-TCDD and 2,3,7,8-TCDF used empirically derived information on the amount of soil or dust that adheres to a square centimeter of skin, the area of skin exposed in various settings and the absorption rate of 2,3,7,8-TCDD or 2,3,7,8-TCDF through skin to derive the dose of 2,3,7,8-TCDD or 2,3,7,8-TCDF from dermal contact with contaminated soil or dust. The values used for each model input are presented in Table 5-26.

The calculation of dermal exposure proceeded in two steps. First the average daily exposure from dermal contact was calculated as the product of the area of skin affected, the contact rate, the dermal absorption rate and the duration of contact. Second, the risk from dermal contact was calculated using the estimate of daily dose and the slope factor of 2,3,7,8-TCDD and 2,3,7,8-TCDF.

(a) **Methods for estimating soil concentrations.** The methods of sludge application considered were (1) application as a top dressing, and (2) incorporation into a layer of soil. Concentrations in the soil for all top dressing scenarios were assumed equal to concentrations in the land-applied sludge. For sludge that was incorporated into the soil, the concentrations were dependent on the depth of the sludge incorporated in the soil, frequency of application, and initial sludge concentration.

The mass of the contaminant added was calculated by multiplying the application rate by the contaminant concentration in the sludge. The volume of soil with which the sludge was mixed was determined by multiplying the incorporation depth by the incorporation area. The volume was then multiplied by the soil density to obtain the mass of the soil with which the sludge was incorporated. Average soil concentrations

Table 5-26. Assumptions and Parameter Values - Distribution and Marketing: Dermal Pathway

Input parameter	Estimate for typical individual	Estimate for MEI individual	Notes/explanation/(reference)
Contact rate mg/cm^2, outdoor, child	0.5	1.5	Schaum (1984).
Contact rate mg/cm^2, outdoor, older child	0.5	1.5	Schaum (1984).
Contact rate mg/cm^2, outdoor, adult	3.5	3.5	Based on adults doing yard work (Hawley 1985).
Contact rate mg/cm^2, indoor, child	0.056	0.06	Typical: assuming dust fall indoors = 20% outdoors, with cleaning every two weeks (Hawley 1985).
Contact rate mg/cm^2, indoor, older child	0.056	0.06	Typical: assuming dust fall indoors = 20% outdoors, with cleaning every two weeks (Hawley 1985).
Contact rate mg/cm^2, indoor, adult living space indoor, adult, attic	0.056 1.8 (110 mg/hr)	0.06	Contact rate for adults in living space same as for children; for work in attic, a value of 1.8 mg/cm^2 used for direct contact; for indirect contact with dusty air, estimated contact is 110 mg for 1 hour of attic work (Hawley 1985).
Ratio of contaminant concentration indoors to soil concentration outdoors	0.80	0.85	Hawley (1985).
Time spent outdoors, child (hr)	1,565	2,190	Typical: estimate assumes 5 days/wk, 6 months/yr, 12 hrs/day (12 hrs before soil is washed off). MEI: 7 days/wk, 6 months/yr, 12 hrs/day soil contact (Hawley 1985).
Time spent outdoors, older child (hr)	1,824	2,190	Typical: child outdoors from May to September every day (150 days), 12 hrs of soil contact before washing. MEI: child outdoors every day for 6 months, 12 hrs soil contact (Hawley 1985).
Time spent outdoors, adult (hr)	348	1,565	Typical: assumes 8 hrs of soil contact before washing, 2 days/wk for 5 months. MEI: assumes 12 hrs soil contact before washing, 5 days/wk, 6 months/yr (130 days) (Hawley 1985; Kamman 1990).

Table 5-26. (continued)

Input parameter	Estimate for typical individual	Estimate for MEI individual	Notes/explanation/(reference)
Time spent indoors, child (hr)	4,380	6,570	Typical: 12 hrs/day of indoor dust contact all year. MEI: assumes 24 hrs/day of indoor dust contact for 6 winter months, plus 12 hrs/day for 6 summer months (Hawley 1985; Keenan 1989).
Time spent indoors, older child (hr)	1,460	4,380	Typical: assumes 4 hrs/day of indoor dust contact all year. MEI: assumes 12 hrs/day of indoor dust contact all year (Hawley 1985).
Time spent indoors, adult (hr) living space attic	4,380 48	6,570 72	Living space: Typical: assumes indoor dust contact 12 hrs/day, all year; high assumes indoor dust contact 24 hrs/day, 6 winter months, plus 12 hrs/day, 6 summer months. Attic: Typical: assumes 12 days with soil on skin 4 hrs/day. MEI: assumes 12 days with soil on skin 6 hrs/day (Hawley 1985).
Area of skin exposed outdoor, child (cm^2)	2,100 cm^2	2,800 cm^2	Typical: estimate assumes both hands, legs, and feet exposed during play. MEI: assumes both hands, arms, legs, and feet exposed (Hawley 1985).
Area of skin exposed outdoor, older child (cm^2)	1,600 cm^2	3,200 cm^2	Typical: estimate assumes both hands, forearms, legs from knees down exposed during play. MEI: assumes both hands, legs, and feet exposed during play (Hawley 1985; Keenan 1989).
Area of skin exposed outdoor, adult (cm^2)	1,700 cm^2	2,940 cm^2	Typical: assumes both hands, most of forearms exposed. MEI: assumes adult is wearing shortsleeved shirt, with an open neck, pants, shoes, no gloves or hat (Hawley 1985; Schaum 1984).

Table 5-26. (continued)

Input parameter	Estimate for typical individual	Estimate for MEI individual	Notes/explanation/(reference)
Area of skin exposed indoor, child (cm^2)	500 cm^2	2,800 cm^2	Typical: estimate assumes area exposed equals one half of surface area of child's feet, hands and forearms. MEI: assumes area exposed indoors same as outdoors (Hawley 1985).
Area of skin exposed indoor, older child (cm^2)	400 cm^2	3,200 cm^2	Typical: estimate assumes only hands are exposed. MEI: assumes area exposed indoors same as outdoors.
Area of skin exposed indoor, adult (cm^2)	1,700 cm^2 (attic) 900 cm^2 (living space)	2,940 cm^2 in both attic and living space (living space)	Typical: estimate assumes hands exposed in living space, while adult wears short-sleeved shirt, with an open neck, pants, shoes, no gloves or hat, to work in attic. MEI: assumes area exposed indoors same as outdoors.
Bioavailability through skin	0.012 h^{-1}	0.024 h^{-1} (children only)	Typical: see Appendix A.1. MEI: assumed that skin of children has twice the absorption of adults.
Availability of dioxin from soil matrix	1%	15%	Recommended value: 1% (see Appendix A.1).

for a given year were estimated by adding the mass of contaminant applied to the land for that specific year to the mass present from previous applications and then dividing by the mass of the receiving soil plus the mass of the applied sludge.

(b) Estimates of exposure and risks. The indoor dust and outdoor soil contaminant concentrations were used to estimate human exposure and risk from dermal contact with these media. Daily doses were estimated for three age groups: young children (ages 1-6), older children (ages 7-11) and adults (ages 12 and older). The dose for each age group was calculated using Equation 5-10.

To obtain the weighted average dose over the lifetime of an individual, Equation 5-11 was used. Individual and population risks were calculated using Equations 5-12 and 5-13, respectively.

(c) Model inputs for calculating size of the exposed population. The size of the potentially exposed population was calculated in the following manner. First, the total tons of sludge to distribution and marketing from each plant engaged in this practice were obtained from the 104-Mill Study. This analysis assumed that sludge was applied at a rate of 10 DMT per hectare. Dividing tons by the application rate yielded the number of acres covered by the distributed and marketed sludge. Next, the size of the average garden was used to determine the number of households using distributed and marketed sludge. The average garden size for combined rural and urban vegetable gardens was assumed to be 0.016 hectares. Dividing acres covered by sludge by the number of acres per household gave the number of households affected. Finally, the number of households was multiplied by the average number of persons per household to obtain the total number of persons affected by distributed and marketed sludge.

(2) Estimates of exposures and risks from ingestion of home-grown produce. To model the risks associated with ingestion of home-grown crops, a computer model, executed on an IBM personal computer, was used. Risks through the dietary pathway were calculated by estimating the contaminant concentration in homegrown crops, and then multiplying this concentration by the daily consumption of home-grown vegetables. This analysis assumed that only the residents of the household using the composted sludge were exposed to sludge contaminants. Furthermore, the analysis assumed that home gardeners do not produce meat or dairy products with the distributed and marketed sludge. The total number of households using distributed and marketed sludge was determined based on the quantity of compost going to residential uses, the average application rate in a residential setting, and size of the average garden; the number of persons potentially exposed was then derived by multiplying the number of households by the average number of persons per household.

For households with vegetable gardens, the calculations proceeded in three steps. The first calculation estimated the sludge 2,3,7,8-TCDD and 2,3,7,8-TCDF concentrations in the tissues of crops grown in sludge-amended home gardens. Next, individual risks were estimated based on dietary ingestion of each crop. Finally, risks from all crops were summed to estimate the total cancer risk from 2,3,7,8-TCDD and 2,3,7,8-TCDF through dietary exposures. The calculations for each of these three components are described below.

(a) Determining tissue concentrations of contaminants for produce grown in sludge-amended gardens.

where:

$$CD_i = (C_S)(U_i) \quad (5\text{-}35)$$

CD_i = tissue concentration (dry weight) of 2,3,7,8-TCDD or 2,3,7,8-TCDF in crop i, ug/g dry
C_S = soil concentration of 2,3,7,8-TCDD or 2,3,7,8-TCDF, adjusted for additional mass from added sludge (mg/kg)
U_i = rate of uptake of 2,3,7,8-TCDD or 2,3,7,8-TCDF into tissue of crop i (ug/g dry weight per mg/kg in soil).

$$CW_i = (CD_i)(KDW_i) \quad (5\text{-}36)$$

where:

CW_i = tissue concentration (wet weight) of 2,3,7,8-TCDD or 2,3,7,8-TCDF in crop i, ug/g wet
KDW_i = constant for converting dry weight concentration to fresh weight concentration for crop i

The calculations for determining the soil concentration of 2,3,7,8-TCDD or 2,3,7,8-TCDF are described in Section 5.2.4. Once the contaminant soil concentration had been determined, each crop's uptake rate was applied to contaminant amounts to estimate the concentration of 2,3,7,8-TCDD or 2,3,7,8-TCDF per unit of dry-weight of crop tissue. Finally, dry weight tissue concentrations were converted to wet weight concentrations.

(b) Determining exposures and risks from contaminant ingestion through foods grown in sludge-amended gardens. Exposures were calculated using Equation 5-14. Daily doses were estimated for children ages 1-6 and for individuals over age 7. Doses of 2,3,7,8-TCDD or 2,3,7,8-TCDF from each food were estimated by multiplying fresh weight contaminant concentrations by the amount of that food crop consumed in the average diet for each age group and by the fraction of the daily

quantity consumed that came from the sludge-amended garden. The dose for each food was combined with the dose of 2,3,7,8-TCDD or 2,3,7,8-TCDF from other garden produce to yield a total dietary ingestion of 2,3,7,8-TCDD and 2,3,7,8-TCDF.

The weighted average daily dose of contaminant over an individual's lifetime was calculated as the sum of the daily doses for each age group weighted by the fraction of the individual's lifespan spent as a member of that age group, as described in the following calculation:

$$DOSE_{avg} = \sum (F_g)\ (DOSE_g) \qquad (5\text{-}37)$$

where:

$DOSE_{avg}$ = average daily dose over lifetime, pg/kg/day
$DOSE_g$ = daily dose for individual in age group g, pg/kg/day
F_g = fraction of an individual's lifetime spent in age group g.

The calculation of individual and population risks were performed using Equations 5-12 and 5-13, respectively.

The input parameters used to estimate dose through ingestion of contaminated home-grown produce are summarized in Table 5-27.

(c) Model inputs for calculating size of the exposed population. The size of the potentially exposed population was calculated in the following manner. First, the total tons of sludge to distribution and marketing from each plant engaged in this practice were obtained from the 104-Mill Study. This analysis assumed that sludge was applied at a rate of 10 DMT per hectare. Dividing tons by the application rate yielded the number of acres covered by the distributed and marketed sludge. Next, the size of the average garden was used to determine the number of households using distributed and marketed sludge. The average garden size for combined rural and urban vegetable gardens was assumed to be 0.016 hectares. Dividing acres covered by sludge by the number of acres per household gave the number of households affected. Finally, the number of households was multiplied by the average number of persons per household to obtain the total number of persons affected by distributed and marketed sludge.

(3) Estimates of exposures and risks from direct ingestion of sludge. Direct ingestion of soil can occur when sludge is applied to sites where people may live and work, such as a family farm. To model the risks from the direct ingestion of sludge contaminated with 2,3,7,8-TCDD and 2,3,7,8-TCDF, this analysis adapted a model which accounted for differences in dose from exposure to indoor and outdoor concentrations of soil contaminants. Children ingest far more soil on average than adults; however, adults may also inadvertently ingest soil that adheres to food or cigarettes.

Table 5-27. Assumptions and Parameter Values - Distribution and Marketing: Dietary Pathway

Input parameter	Estimate for typical individual	Estimate for MEI individual	Notes/explanation/(reference)
Plant uptake rates	Above-ground: 0.02; root: 0.5	Above-ground: 0.02; root: 0.5	See Appendix A.4.
Fraction of vegetables from home garden	0.27 for all vegetables but legumes; 0.15 potatoes; 0.07 dried legumes.	0.6 for all vegetables but legumes; 0.45 potatoes; 0.17 dried legumes.	Typical: estimate values represent rural, nonfarm households; MEI: represent rural, farm households (USEPA 1988e).
Adult and child consumption rates	Values based on TAS Dietary Data Base	Values based on TAS Dietary Data Base	

The calculation of risks from direct ingestion of sludge was straightforward. First, the soil concentrations outdoors and the dust concentration indoors were estimated. The outdoor contaminant concentration was multiplied by the quantity of dirt consumed outdoors, while the indoor contaminant concentration was multiplied by the quantity of indoor dust ingested daily. Risk was estimated based on the daily quantity of soil and dust ingested, the gastrointestinal absorption of 2,3,7,8-TCDD and 2,3,7,8-TCDF from soil, and the potency of 2,3,7,8-TCDD and 2,3,7,8-TCDF.

(a) **Methods for estimating soil concentrations.** The concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF in outdoor soil were estimated as described in Section 5.2.4.

(b) **Exposure was estimated using Equation 5-29.** Individual and population risks were calculated using Equations 5-12 and 5-13, respectively. The values used for each model input are summarized in Table 5-28.

(c) **Model inputs for calculating size of the exposed population.** The size of the potentially exposed population was estimated in the following manner. First, the total tons of sludge to distribution and marketing from each plant engaged in this practice were obtained from the 104-Mill Study. This analysis assumes that sludge was applied at a rate of 10 DMT per hectare. Dividing tons by the application rate yielded the number of acres covered by the distributed and marketed sludge. Next, the size of the average garden was used to determine the number of households using distributed and marketed sludge. The average garden size for combined rural and urban vegetable gardens was assumed to be 0.016 hectares. Dividing acres covered by sludge by the number of acres per household gave the number of households affected. Finally, the number of households was multiplied by the average number of persons per household to obtain the total number of persons affected by distributed and marketed sludge.

(4) Estimates of exposures and risks to the general population from inhalation of sludge-contaminated particulates. 2,3,7,8-TCDD and 2,3,7,8-TCDF adhering to soil particles can become suspended in the air near a site treated with sludge. Transport downwind will dilute the concentration of particles from a treated area; these particles will also redeposit on surfaces. Residents using composted pulp and paper mill sludge on their home gardens may be exposed to 2,3,7,8-TCDD or 2,3,7,8-TCDF by inhaling these particles. This section describes the methods used to estimate the emissions of particles from a treated site and the subsequent human exposure to these emissions. This analysis only considered exposure to inhaled particulates for residents onsite.

Table 5-28. Assumptions and Parameter Values - Distribution and Marketing: Soil Ingestion Pathway

Input parameter	Estimate for typical individual	Estimate for MEI individual	Notes/explanation/(reference)
Amount soil ingested, g/day.	0.1	0.1	Single value used to represent average daily consumption over lifetime. (OSWER Directive 9850.4 "Interim Final Guidance for Soil Ingestion Rates," J. Winston Porter, January 27, 1989).
Percent of daily ingestion from outdoors, child	100%	100%	Analysis assumes 100% of soil from outdoor sources.
Amount of soil ingested from sludge-amended land	0.10	1.0	Typical value of 10% was used in evaluation of reuse of municipal sewage sludge. MEI values arbitrary.

To estimate the suspended particulate concentration at treated sites, a model for emissions from wind erosion was chosen since the analysis focuses on average exposures over the long-term. The assumptions underlying the model are described as follows: "This method assumes that the uncrusted contaminated surface is exposed to the wind and consists of finely divided particles. This creates a condition defined ... as an "unlimited reservoir" and results in maximum wind-caused dust emissions." The model incorporated information on wind speed and percent vegetation cover to estimate the flux of small particles (i.e., less than 10 μm) from an area of land. Soil amended with paper mill sludge may not have the characteristics assumed by the model; to the extent that the surface of a sludge-amended site consists of coarser particles, the model was likely to overestimate emissions.

To obtain particulate concentration, the calculated emission rate was used as input to a box model of atmospheric mixing. The box model ignored any atmospheric dispersion downwind, and was only appropriate for estimating onsite concentrations. The model used wind speed, size of the site and the mixing height to yield an onsite particulate concentration.

As an alternative approach to estimating onsite particulate concentration, an additional model was applied that uses measured values of total suspended particles adjusted by the fraction of particles assumed to be derived from local soils.

The calculation of risks from inhalation of particles required several steps. First, the emissions of particles from the treated area were estimated. Next, the indoor and outdoor concentrations of particles onsite were calculated. The concentrations were combined with information about the length of time spent indoors and outdoors, respiratory rate, and the slope factor of 2,3,7,8-TCDD and 2,3,7,8-TCDF to yield the estimated cancer risks.

(a) Method used to estimate the concentration of 2,3,7,8-TCDD and 2,3,7,8-TCDF in particulates. The first step in the calculation of concentration of 2,3,7,8-TCDD and 2,3,7,8-TCDF in particulates was to estimate the emissions of particulates from the treated areas. The method used estimated the flux of dust particles from the surface as a function of (1) the vegetative covering of the surface, and (2) the cube of the ratio of the windspeed to the threshold wind velocity (the velocity required to initiate erosion). The dust flux was then converted to a contaminant emission rate.

The next step was to estimate the concentrations of particulates on the land-treatment site. Both outdoor concentrations and indoor concentrations were calculated. The outdoor concentrations were derived by dividing the emissions by the product of the length of one side of the treated area by mixing height and by windspeed.

An alternative method of calculating outdoor contaminant particulate concentrations was to adjust the measured total suspended particulate concentration at the site by the fraction believed to originate from local (contaminated) soils.

The indoor suspended particle concentration was derived by applying the ratio of suspended particulate concentration indoors to the suspended particulate concentration outdoors. Next, since only a portion of indoor dust was assumed to originate from outdoor sources (the rest is derived from smoking, cooking, etc.) the contaminant concentration in indoor dust was adjusted by a fraction representing the ratio of indoor dust contaminant concentration to the outdoor soil contaminant concentration.

(b) Estimates of exposures and risks. Once the concentration of contaminants in particulates was estimated, human exposure to contaminated particulates can be estimated. In the MEI scenario, the particulate concentration was estimated based on total particulates. When calculating exposure from this estimate of particulate concentration, the first step was to determine the concentration of particles that are respirable. The respirable concentration was estimated as:

$$RC_o = (C_o)(FR) \quad (5\text{-}38)$$

and

$$RC_{in} = (C_{in})(FR) \quad (5\text{-}39)$$

where:

C_o = concentration of 2,3,7,8-TCDD or 2,3,7,8-TCDF in suspended particles outdoors, mg/m^3
FR = fraction of suspended particles that are respirable
RC_o = respirable particulate concentration outdoors, mg/m^3
C_{in} = concentration of 2,3,7,8-TCDD or 2,3,7,8-TCDF in suspended particles indoors, mg/m^3
RC_{in} = respirable particulate concentration indoors, mg/m^3.

In the typical scenarios, the estimation of contaminant emissions adhering to respirable particles assumed that all of the emissions were respirable.

The next step in the calculation of human exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF through the inhalation of particulates was the estimation of the daily dose. The daily dose was calculated for three age groups: young children (ages 1-6), older children (ages 7-11), and adults (ages 12 and older) using Equation 5-30.

A weighted average dose for an individual over the entire lifetime was derived by weighting the daily dose received during each age interval by the fraction of the individual's lifespan spent in that age group. This calculation is described in Equation 5-32. Individual and population risks were calculated using Equations 5-12 and 5-13, respectively.

The data inputs for the particle inhalation model are presented in Table 5-29.

(c) Model inputs for calculating size of the exposed population. The size of the potentially exposed population was estimated in the following manner. First, the total tons of sludge to distribution and marketing from each plant engaged in this practice were obtained from the 104-Mill Study. This analysis assumes that sludge was applied at a rate of 10 DMT per hectare. Dividing tons by the application rate yielded the number of acres covered by the distributed and marketed sludge. Next, the size of the average garden was used to determine the number of households using distributed and marketed sludge. The average garden size for combined rural and urban vegetable gardens was assumed to be 0.016 hectares. Dividing acres covered by sludge by the number of acres per household gave the number of households affected. Finally, the number of households was multiplied by the average number of persons per household to obtain the total number of persons affected by distributed and marketed sludge.

(5) Estimates of exposures and risks from inhalation of vapors. Residents using distributed and marketed sludge may incur risk from the inhalation of volatilized 2,3,7,8-TCDD and 2,3,7,8-TCDF. Because actual locations of the homes using composted sludge are not known, the ISCLT model could not be used to estimate downwind concentrations. As a result, this analysis estimated only exposures to onsite residents, using a box model to obtain the onsite concentrations from the emissions estimates.

The calculation of risks from the inhalation of vaporized 2,3,7,8-TCDD and 2,3,7,8-TCDF required first the estimation of emissions, then the calculation of indoor and outdoor onsite concentrations. The concentrations were combined with data on time spent indoors and outdoors, respiration rate and potency of 2,3,7,8-TCDD and 2,3,7,8-TCDF to obtain the estimated cancer risk from this pathway of exposure.

(a) Methods for estimating vapor emissions. This analysis assumed that emissions from home gardens were a function of the partitioning of 2,3,7,8-TCDD and 2,3,7,8-TCDF between soil and air and between water and soil. The emissions estimate was then multiplied by the area of the home garden, in m^2, to obtain the total emissions of vapor from the residential site. The emission rate was then coupled with a box model to obtain the onsite concentrations of vapor.

Table 5-29. Assumptions and Parameter Values - Distribution and Marketing: Vapor and Particulate Inhalation Pathways

Input parameter	Estimate for Typical individual	Estimate for MEI individual	Notes/explanation/(reference)
CONCENTRATION			
Method for calculating particulate emissions	Assume 50% vegetative cover	Assume 50% of total TSP from local soil	Typical: calculated assuming 50% vegetation. MEI: assume 50% of total TSP from local soil; local TSP = 64 ug/m^3 (Hawley 1985).
Indoor TSP concentration as function of outdoor TSP concentration	0.75	0.85	Hawley (1985).
EXPOSURE			
Percent TSP respirable	N/A	0.90	Typical: estimate for emissions is from information that gives estimates of small (<10 um) particle emissions. All of these particles are assumed to be respired. The range given here applies only to TSP, which consists of large and small particles (i.e., this range applies only to the MEI estimate of emissions).
Fraction of TSP inhaled to lung	1/3 of respired TSP	0.90 (100% of respired TSP)	Typical: one-third of total TSP is initially found in lungs; MEI: 100% (Schaum 1984).
Fraction of TSP inhaled to GI tract	2/3 of respired TSP	0%	
Absorption rate, lung	1.0	1.0	Value from bioavailability is applied to the particles that reach the alveoli.
Hours per day and number of days spent outdoors, adult	8 hrs/day 40 days	12 hrs/day 130 days	Typical: assumes adult works 2 days per wk. MEI: 6 months, 5 days/wk, 12 hrs/day (Hawley 1985).

Table 5-29. (continued)

Input parameter	Estimate for Typical individual	Estimate for MEI individual	Notes/explanation/(reference)
EXPOSURE			
Hours per day and number of days spent outdoors, child	8 hrs/day 130 days	12 hrs/day 182 days	Typical: assumes child plays outdoors 8 hrs/day, May-October, 5 days per wk. MEI: assumes child plays outdoors 12 hrs/day, May-October, 7 days per wk.
Hours per day and number of days spent outdoors, older child	5 hrs/day 152 days	12 hrs/day 152 days	Typical: assumes child plays outdoors an average of 5 hrs/day, May-September. MEI: assumes 12 hrs/day, May-September (Hawley 1985).
Hours per day and number of days spent indoors, adult, attic, living space	12 hrs/yr in attic; 12 hrs/ day, 130 days plus 24 hrs/day, 235 days, farmer	12 hrs/yr in attic; 12 hrs/ day, 130 days plus 24 hrs/ day 235 days	Attic: assumed that adult spends 12 hrs/yr (either 1 day for 12 hrs or 1 hr for 12 days) in attic. Living space: 12 hrs/day in living space for 130 days in summer months plus 24 hrs/day in living space for rest of year (Hawley 1985).
Hours per day and number of days spent indoors, child	16 hrs/day, 130 days, plus 24 hrs/day, 235 days	12 hrs/day, 182 days, plus 24 hrs/day, 182 days	MEI: assumes child plays indoors 12 hrs per day, 7 days per wk during May-October, rest of year indoors 24 hrs per day. Typical: assumes indoors 16 hrs/day, 5 days/wk during May-October, indoors 24 hrs/day for remainder of days.
Hours per day and number of days spent indoors, older child	16 hrs/day for 365 days	12 hrs/day, 152 days plus 24 hrs/day for 213 days	Typical: assumes child is indoors 16 hrs/day. MEI: assumes child is indoors an average 12 hrs/day from May-September, and is indoors for the rest of the year 24 hrs/day (Hawley 1985).
CONCENTRAITON			
Indoor contaminant concentration as function of outdoor contaminant concentration	indoor = 80% outdoor	indoor = 100% outdoor	Typical: from Hawley (1985). MEI: estimate is maximum value possible.

The indoor vapor concentration was derived by applying the ratio of vapor concentration indoors to the vapor concentration outdoors. It was assumed that the relationship between vapor concentrations indoors and outdoors was similar to the ratio between indoor and outdoor particulate concentrations (that is, indoor concentrations are approximately 75 percent of outdoor concentrations with a range from 70 to 85 percent).

(b) Estimates of exposure and risk. Once the concentration of contaminant in the air was estimated, the calculation of exposure and risks from the inhalation of vapor then proceeds in the same manner as the exposure and risk from the inhalation of particles, described in Section 5.2.5(4). In some cases, however, the data inputs used for the estimation of exposure and risk were different than those used in Section 5.2.5(4).

(6) Summary of results. Health risks from the distribution and marketing of paper mill sludge containing 2,3,7,8-TCDD and 2,3,7,8-TCDF are summarized in Table 5-30. In general, risks for the "maximum exposed individual" (MEI) were two to three orders of magnitude higher than the risks for a typical individual. Estimated risks for the MEI were lowest for the particle inhalation and dietary pathways, and were highest for the pathways involving direct human contact with contaminated soil (i.e., the dermal and direct ingestion pathways).

5.3 Analysis of Uncertainty

Total risk estimates for ingestion of contaminated ground water were prepared for the disposal of sludge in landfills, disposal of sludge in surface impoundments, and the land application of sludge. For landfills and surface impoundments, risk estimates varied by one to two orders of magnitude. Results varied significantly between calculations involving the SESOIL model and those based on simpler, but less data intensive, assumptions about the equilibrium partitioning of contaminants between dissolved and adsorbed phase. Low estimates of risk were not prepared, but estimates of zero risk through this pathway could be easily derived if landfills are assumed to be located in areas without productive aquifers.

Total risks from inhalation of particulates were estimated for persons living on a land application site, or applying pulp and paper sludge to home gardens. The estimated range of uncertainty was relatively low for these estimates (one to two orders of magnitude), and reflected differences in results based on different mathematical models for estimating particulate suspension. Other key variables were the ratio of indoor to outdoor concentrations, and assumed absorption rates in the lungs and gastrointestinal tract.

Table 5-30. Estimates of Health Risks to the General Population from Distribution and Marketing of Pulp and Paper Sludges Contaminated with 2,3,7,8-TCDD and 2,3,7,8-TCDF

Exposure pathway	MEI risk[a] (per lifetime)	Typical risk[a] (per lifetime)	Exposed population	Total risk (cases/year)[b]
Dermal exposure from contact with soil contaminated by distributed and marketed sludge	$1x10^{-4}$	$3x10^{-8}$	3,500,000	$2x10^{-3}$
(Percent TCDD)[c]	(63)	(59)		(59)
Exposure from direct ingestion of soil contaminated by distributed and marketed sludge	$1x10^{-4}$	$3x10^{-7}$	3,500,000	$2x10^{-2}$
(Percent TCDD)[c]	(57)	(56)		(56)
Inhalation exposure from air contaminated by volatilization from soil contaminated by distributed and marketed sludge	$6x10^{-7}$	$5x10^{-8}$	3,500,000	$3x10^{-3}$
(Percent TCDD)[c]	(5)	(6)		(6)
Inhalation exposure from particulates from soil contaminated by distributed and marketed sludge	$2x10^{-7}$	$5x10^{-9}$	3,500,000	$3x10^{-4}$
(Percent TCDD)[c]	(64)	(71)		(71)
Dietary exposure from produce grown in gardens contaminated by distributed and marketed sludge	$2x10^{-8}$	$5x10^{-11}$	3,500,000	$3x10^{-6}$
(Percent TCDD)[c]	(62)	(71)		(71)

[a] Calculated as: [Estimated Dose] x $1.6x10^{-4}$ per (pg/kg)/day.

[b] Calculated as: [Typical Risk x Exposed Population] / [Life Expectancy].

[c] Calculated as: $\frac{\text{[TCDD Dose]}}{\text{[TCDD Dose]} + (1/10)\text{[TCDF Dose]}}$

Risk estimates for inhalation of volatilized 2,3,7,8-TCDD and 2,3,7,8-TCDF were also relatively stable with respect to the assumptions tested for land application and distribution and marketing scenarios, and represented variation in assumptions about time spent indoors and outdoors, ratios between indoor and outdoor concentrations, and the fraction of organic carbon in the soil. For landfills, the wider range of estimates for exposure and risks from volatilized contaminants reflected the testing of two different models for estimating volatile emissions from soil. Both typical estimates of risk and MEI estimates of risk from emissions from surface impoundments were based on a two film resistance model; differences in risk estimates were therefore attributable to differences in selected parameter values.

Precise estimation of total risks from ingestion of surface water contaminated by runoff from sludge re-use or disposal would require extensive, site-specific data for each sludge management site. Without detailed data about the location of each site, distances to surface water, site and surrounding topography, hydrology of nearby surface water, locations (if any) of withdrawal of surface water for drinking water sources, and numerous other key data, one must rely on simple screening models to derive rough estimates of potential risk. The wide range of risk estimates reported for this exposure pathway highlights the fact that without such site-specific data, precise quantification was impossible. The possibility of significant risks through this pathway cannot be ruled out on the basis of existing data. Risk estimates are sensitive to the size of the drainage area assumed for the stream receiving runoff from each site.

Similarly, precise estimation of total risk from fish ingestion was impossible without detailed, site-specific information. Depending on the assumptions chosen for modeling exposure and risks through this pathway, resulting estimates can vary by four orders of magnitude or more. As with risks from the ingestion of drinking water from surface water sources contaminated by surface runoff, significant risks from ingestion of fish cannot be dismissed on the basis of this analysis.

Total risks from dietary consumption of vegetables, meat or dairy products grown from sludge amended land (or from feeds grown on sludge-amended land) varied by about two orders of magnitude for land application sites, depending on the assumptions chosen. Key assumptions included the percentage of animal diet consisting of sludge, and bioconcentration factors. For distribution and marketing, dietary risk estimates varied by four orders of magnitude, depending on which set of assumptions was used. Results were greatly influenced by whether we assume soil incorporation, and by the fraction of each household's diet assumed to be homegrown.

Estimated total risks from dermal absorption of 2,3,7,8-TCDD and 2,3,7,8-TCDF in sludge or soil were also subject to the selection of parameter values. This range was attributable to numerous differences in the two scenarios, including whether or not the sludge was assumed to be soil incorporated, the area of skin exposed, contact rates and absorption rates for skin, and numerous other factors. Estimated total risks from direct ingestion of soil varied by two to three orders of magnitude, depending on whether the sludge was assumed to be soil incorporated, assumed rates of soil ingestion for children and adults, and the fraction of daily soil ingestion originating from the treated area.

Considerable uncertainty is implicit in the risk estimates derived in this chapter. In the absence of detailed site-specific data for the numerous sites at which sludge is re-used or disposed, such broad ranges of uncertainty in exposure and risk estimates are unavoidable.

5.4 Conclusions

Table 5-31 compares human health risk estimates for each waste disposal method analyzed. For each waste management practice, it reports results for the exposure pathway found to result in highest risks to the "most exposed individual" and to the total population. The possibility of simultaneous exposure through multiple pathways was ignored. By comparing maximum estimated risks for each management practice, Table 5-31 allows a simple comparison of risks associated with each practice.

As can be seen from the table, estimated risks to the typical exposed individuals were typically three to six orders of magnitude lower than risks to the MEI, except for the landfilling of paper wastes, for which separate population risk estimates were not performed. As shown by the table, none of the waste disposal methods and exposure pathways analyzed was expected to result in a total population cancer risk of more than one expected incremental cancer case per twenty years of sludge or paper disposal.

In summary, this analysis found that typical risks to human populations exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF from pulp and paper mill sludge are generally low. Risks to most exposed individuals, however, could be significant, depending on site-specific circumstances and individual human behavior; it is not certain that the hypothetical circumstances depicted in estimating these risks actually occur. As discussed in Section 5.5, the best estimates of human exposure and risk provided in this report should not be interpreted as precise quantification of exposure and risks, but rather as general indicators of the magnitude of those risks.

Table 5-31. Maximum General Population Health Risks from Exposure to TCDD and TCDF, by Waste Management Method

Exposure pathway	MEI risk[a,b] (per lifetime)	Typical risk[a,c] (per lifetime)	Exposed population[d]	Total risk (cases/ year)[e]
Potential human exposure from disposal of pulp and paper sludge in landfills	$5x10^{-2}$	$8x10^{-8}$	14,200,000	$2x10^{-2}$
(Percent TCDD)[f]	(63)	(65)		(65)
Potential human exposure from disposal of paper wastes in landfills	$<3x10^{-7}$	$<3x10^{-7}$	Not estimated	Not estimated
(Percent TCDD)[f]	(17)	(17)		
Potential human exposure from disposal of pulp and paper sludge in surface impoundments	$1x10^{-1}$	$1x10^{-7}$	4,760,000	$7x10^{-3}$
(Percent TCDD)	(63)	(65)		(65)
Human exposure from land application of pulp and paper sludge	$2x10^{-1}$	$1x10^{-5}$	1,700,000	$1x10^{-2}$
(Percent TCDD)[f]	(63)	(4)		(65)
Human exposure from distribution and marketing of pulp and paper sludge	$1x10^{-4}$	$3x10^{-7}$	3,500,000	$1x10^{-3}$
(Percent TCDD)[f]	(63)	(56)		(56)

[a] Calculated as: (Estimated Exposure) x $1.6x10^{-4}$ per (pg/kg)/day.

[b] Represents highest risk to most exposed individual through any single exposure pathway associated with this waste disposal or re-use method. All risk estimates based on best estimate input parameters.

[c] Represents risk to typical exposed individual for single exposure pathway associated with highest total risk for this waste disposal or re-use method. All risk estimates based on best estimate input parameters.

[d] Total exposed population for single exposure pathway associated with highest typical risks for this sludge re-use or disposal method.

[e] Calculated as: (Average Risk x Exposed Population) / (Life Expectancy).

[f] Calculated as: $100 \times \frac{\text{(Exposure to TCDD)}}{\text{(Exposure to TCDD) + (1/10) (Exposure to TCDF)}}$

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6. ASSESSMENT OF RISKS TO THE GENERAL POPULATION FROM THE DISCHARGE OF EFFLUENTS FROM THE PULP AND PAPER INDUSTRY

6.1 Introduction

6.1.1 Purpose

The purpose of this assessment was to develop reasonable worst-case estimates of exposures and risks to humans from 2,3,7,8-TCDD and 2,3,7,8-TCDF discharges from chlorine-bleaching pulp and paper mills. The assessment focused on the highest estimated in-stream contaminant concentrations immediately downstream of each mill discharge point (assuming steady-state, fully mixed conditions) and the potential human health impacts resulting from the consumption of 2,3,7,8-TCDD and 2,3,7,8-TCDF contaminated fish and drinking water associated with these exposures. Because no comprehensive studies on 2,3,7,8-TCDD and 2,3,7,8-TCDF build-up in sediments and bioaccumulation up the food chain exist, only the water column was investigated as a potential route of exposure and uptake of 2,3,7,8-TCDD and 2,3,7,8-TCDF by exposed fish. Carcinogenic and non-carcinogenic effects in humans are considered.

This chapter is a condensed version of the following report prepared by the U.S. EPA Office of Water Regulations and Standards (OWRS) as part of the Interagency Dioxin-in-Paper Workgroup:

> USEPA. 1990. U.S. Environmental Protection Agency. Risk assessment for 2,3,7,8-TCDD and 2,3,7,8-TCDF contaminated receiving waters from U.S. chlorine bleaching pulp and paper mills. Washington, D.C.: Office of Water Regulation and Standard, U.S. Environmental Protection Agency. August 1990.

6.1.2 Scope

Two approaches were used to estimate and compare exposures to 2,3,7,8-TCDD and 2,3,7,8-TCDF resulting from surface water effluent discharges from pulp and paper mills. In the first approach, a simple dilution calculation was conducted to estimate the in-stream contaminant concentration after the effluent is mixed in the receiving water. This calculation assumed 100 percent of the in-stream contaminants (both dissolved and adsorbed to suspended solids) are bioavailable to fish.

The second approach used the Exposure Assessment Modeling System (EXAMS II) (Burns et al., 1982; Burns and Cline, 1985; Harrigan and Battin, 1989) to partition in-stream steady-state water column contaminant concentrations between dissolved and particulate forms. However, only the dissolved contaminant concentration predicted by EXAMS II was considered in determining exposure and risk. Both the simple dilution and EXAMS II in-stream exposure methods were used to estimate

the potential human health risks associated with ingestion of contaminated fish and drinking water. No attempt was made in the EXAMS II approach to estimate fish exposure to contaminants associated with suspended particulates, bed sediments, or the food chain due to lack of sufficient and appropriate data.

The EXAMS II method, on the other hand, provides a more reasonable estimate of the direct exposure of fish to the contaminants from water only. One result of this analysis is an upper bound estimate of the potential risk of cancer over the lifetime of a hypothetically exposed individual. No attempt has been made to characterize the human population potentially at risk. For these risk estimations, reasonable worst-case ambient and effluent characterizations were used, along with best estimates of physical and chemical properties of 2,3,7,8-TCDD and 2,3,7,8-TCDF. Because not all of the parameter values used in this assessment are "worst-case," the hypothetically exposed individual is not considered the "most exposed individual."

The probability of an individual developing cancer in a lifetime due to the ingestion of contaminated fish or drinking water was calculated based on exposure estimates and the EPA carcinogenic potency factor. Also, the data for exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF from bleached paper mills were screened for exposure scenarios exceeding an average of 100 pg/kg/day, an estimated one-day Health Advisory (HA) for 2,3,7,8-TCDD for protection against human liver effects (see Section 3.3.2 for details on this HA). Exposure scenarios exceeding this level were examined in more detail to determine whether the cancer or non-cancer endpoint is the most sensitive indicator of risk.

6.2 Exposure and Risk Assessment Methodology Requirements

The approach taken was designed to incorporate an appropriate balance between the difficulty (detail) of the analysis and the accuracy of the results. The critical factors considered in the development of the analytical approach were: 1) in-stream chemical transformation processes, 2) applicability of calculation methods, 3) availability of environmental data, and 4) model sensitivity.

The chemical/physical processes thought to most significantly influence the fate of 2,3,7,8-TCDD and 2,3,7,8-TCDF in the aquatic environment are dilution and adsorption of the contaminants to particulates. Other processes, such as volatilization, hydrolysis, photolysis, and biotransformation do not appear to significantly affect the fate of the contaminants.

A simple dilution calculation method for estimating water column concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF provides total in-stream contaminant concentration without consideration of the effects of adsorption to particulates and eventual sedimentation or other fate

processes. This method of predicting exposure results in worst-case water column exposure estimates. Because 2,3,7,8-TCDD and 2,3,7,8-TCDF appear to have a high affinity for adsorption to particulates, other methods of estimating contaminant fate and transport are necessary to consider partitioning between the dissolved and solid forms of the contaminants. The Exposure Assessment Modeling System (EXAMS II), a state-of-the-art surface water contaminant modeling system, is capable of estimating the partitioning of a contaminant between its dissolved form in the water column and that portion that associates with suspended and benthic solids.

Many parameters describing the physical/chemical properties of 2,3,7,8-TCDD and 2,3,7,8-TCDF and mill-specific effluent and receiving water characteristics are required to operate EXAMS II. STORET (a water quality data base maintained by EPA's Office of Water that can access water quality sampling data from monitoring stations around the country) was used to access data on flow, total suspended solids (TSS), pH, and other parameters required to operate EXAMS II for the receiving waters of most mills.

A model sensitivity analysis was conducted to determine which environmental data parameter variations had the greatest influence on EXAMS II contaminant concentration estimation results under steady-state conditions and given known 2,3,7,8-TCDD and 2,3,7,8-TCDF physical/chemical properties. The analysis indicated that variations in receiving water suspended solids levels produced the greatest variations in resulting in-stream dissolved contaminant concentrations. Therefore, mill-specific values were obtained and used in all subsequent EXAMS II analyses. All other environmental parameters, except for mill-specific contaminant loadings, and receiving water flow rates, were assigned default values.

6.3 Exposure Assessment Methodology

(1) In-stream contaminant concentrations. In this investigation, two approaches are used to estimate and compare exposures to 2,3,7,8-TCDD and 2,3,7,8-TCDF resulting from surface water effluent discharges from pulp and paper mills. The first approach consists of a simple dilution calculation conducted to estimate the total, steady-state in-stream concentration of the contaminants after the effluent is mixed in the receiving water. This calculation assumes 100 percent of the in-stream contaminants (both dissolved and adsorbed to suspended solids) are bioavailable to fish. In the second approach, the Exposure Assessment Modeling System (EXAMS II) is used to partition in-stream steady-state concentrations of the contaminants between dissolved and particulate forms.

EXAMS II accounts for the high affinity of 2,3,7,8-TCDD and 2,3,7,8-TCDF for solids and, therefore, the likelihood that a percentage of the contaminants will be associated with suspended and benthic

solids. Both the simple dilution and EXAMS II approaches are used to estimate and compare the potential human health risks associated with ingestion of contaminated fish tissue and drinking water.

The following simple dilution equation was used to provide an estimate of the concentration of a contaminant downstream from a point source release into a flowing water body after dilution of the substance by the receiving water (USEPA 1988b):

$$C = \frac{C_e Q_e}{Q_t} \qquad (6\text{-}1)$$

where:

C = concentration of substance in stream (mass/volume),
C_e = concentration of substance in effluent (mass/volume),
Q_e = effluent flow rate (volume/time), and
Q_t = combined effluent and stream flow rate (volume/time).

Although this calculation is easily executed and provides a quantitative estimate of in-stream contaminant concentration which is limited in precision only by the precision of the input parameters, this calculation provides only the total in-stream contaminant concentration attributable to the point source. It does not provide a distribution of the contaminant between the dissolved and adsorbed states.

EXAMS II, on the other hand, is a sophisticated computer modeling system capable of computing parameters of exposure, fate, and persistence. Once input parameters describing the environment (temperature, stream compartment geometry, receiving water flow, solids, organic carbon fraction, etc.), the chemical contaminant characteristics (molecular weight, vapor pressure, Henry's Law constant, K_{ow}, K_{oc}, solubility, etc.), and the loadings are entered, the model produces a report detailing the three sets of computations described above (i.e., exposure, fate, and persistence).

For each mill, the calculated water column concentrations were used as the basis for further calculations. The estimated concentrations were considered 100 percent available to the aquatic organisms living in the receiving waters, 100 percent available to humans using the water as a drinking water source, and 95 percent available to humans through fish tissue consumption.

For this assessment, the mills were grouped into one of three categories: direct dischargers to free flowing streams, direct dischargers to open waters (e.g, oceans, lakes, reservoirs), and indirect dischargers (dischargers to POTWs) to either free-flowing streams or open

waters. Contaminant concentrations resulting from direct discharges to free-flowing streams were calculated directly using the simple dilution and EXAMS II water column methods when adequate environmental data were available for the site. Contaminant concentrations resulting from direct discharges to open water bodies were calculated using the simple dilution method, based on zone of initial dilution factors for the mills that were provided by EPA Regions (Albright 1990; Davis 1989; Derose 1989; Fisher 1989; Greenburg 1989; Greenfield 1990; Hall 1989; Hangarden 1989; Henry 1989; Hyatt 1989; Keefler 1989; Loster 1989; Menzardo 1989; Tingperg 1989; and Weeks 1989). The zone of initial dilution is the region of initial mixing surrounding or adjacent to the end of the outfall pipe in which aquatic inhabitants may be chronically exposed to concentrations of pollutants in excess of water quality standards. Initial dilution is defined by USEPA (1982) as the flux-averaged dilution (averaged over the cross-sectional area of the plume) achieved during the period when dilution is primarily a result of plume entrainment, and is not dominated by ambient conditions.

EXAMS II requires stream flow data as input to calculate in-stream contaminant concentrations. Because flows for open water bodies were not available, it was necessary to back-calculate "surrogate" water body flows for direct dischargers to open water bodies based on known mill plant flows and the dilution factors for the mills. The following calculation was used to determine surrogate water body flows for direct open water discharges:

$$F_o = (D * F_p) - F_p \qquad (6\text{-}2)$$

where:

F_o = surrogate open water body flow
F_p = mill plant flow
D^p = dilution factor

Contaminant concentrations resulting from indirect discharges to either free-flowing streams or open water bodies were calculated using the same methods described above, except that loadings were decreased to 2 and 25 percent of the total to account for the effects of treatment on the discharge effluent stream.

In-stream contaminant concentrations were calculated using the harmonic mean flow for the receiving water. The harmonic mean flow is defined as the reciprocal of the mean value of the reciprocal of individual values.

(2) Whole-body and fish filet contaminant concentrations. Tissue residue levels for fish exposed to the in-stream contaminant concentrations estimated above were calculated by multiplying the

contaminant concentration by estimated bioconcentration factors (BCFs) for 2,3,7,8-TCDD and 2,3,7,8-TCDF. Estimated fish tissue residue levels resulting from exposure to these contaminants in the water column were based on BCFs of 5,000 (2,3,7,8-TCDD, filet only), 100,000 (2,3,7,8-TCDD, whole body) and 3,900 (2,3,7,8-TCDF, whole body). For example, assuming a 2,3,7,8-TCDD water column concentration of 2.2×10^{-8} ng/l, and a BCF of 5,000, whole body fish tissue concentration would be calculated as follows:

(water column concentration) (BCF) = filet concentration

$$(2.2 \times 10^{-8}\text{ng/l})(5{,}000) = 1.1 \times 10^{-4}\text{ng/kg}$$

The BCF for 2,3,7,8-TCDD of 5,000 (USEPA 1984) is based on fish filet residue levels, not whole body levels. This BCF, in combination with a moderate fish tissue consumption rate (6.5 g/day), was the basis for estimating human health impacts from consumption of contaminated fish in EPA's ambient water quality criteria for 2,3,7,8-TCDD.

A second BCF of 100,000 was developed primarily from the results of the EPA Duluth Laboratory's most recent studies on the bioconcentration of 2,3,7,8-TCDD by fish (Cook et al. Unpublished). During these investigations, BCFs for carp and fathead minnows were determined through laboratory studies with exposures of up to 71 days in duration. The BCF levels presented in the Cook et al. study ranged from 65,900 ± 9,300 to 159,000 ± 40,000. These BCF values were developed from whole body tissue residue levels. The Cook et al. study is preliminary and has not been peer-reviewed. Based on this information, the present study selected a reasonably conservative BCF value of 100,000 to represent a more extreme worst-case bioaccumulation potential that, in combination with higher fish tissue consumption rates, result in higher estimated human health risks.

Results of a recent literature review by Nabholz et al. (Unpublished) were used as the basis for selecting BCF values to determine whole body contaminant concentrations in fish exposed to 2,3,7,8-TCDF in the water column. Only three measured fish BCF values for 2,3,7,8-TCDF were identified, two from water exposures and one from a dietary source. The geometric mean of the measured BCF values for water exposure (3,900) was used in the present study.

Contaminants are generally distributed unequally among the tissues in the fish after their ingestion of the contaminants. For instance, high concentrations of many contaminants accumulate in the fish liver, generally an inedible portion of the fish. Also, whole body residue levels include the viscera, which contain significant quantities of sediments ingested during feeding. Because of the affinity of 2,3,7,8-TCDD and 2,3,7,8-TCDF for sediment, high concentrations of

contaminants would be found in this inedible portion. Using the estimated whole-body concentration of a contaminant would therefore not accurately reflect human exposure as a result of consumption of the edible portion of the fish (the filet). In general, the concentration of 2,3,7,8-TCDD in fish muscle is about 50 percent of whole fish concentration (Branson et al. 1985). To compensate for the unequal partitioning of contaminants between the edible and inedible fish tissues, the estimated whole-body BCFs of 100,000 (for 2,3,7,8-TCDD) and 3,900 (for 2,3,7,8-TCDF) were multiplied by 0.5 to estimate the concentration in the edible portion of the fish. Thus, the effective BCFs are 50,000 for 2,3,7,8-TCDD and 1,950 for 2,3,7,8-TCDF.

The 5,000 BCF for 2,3,7,8-TCDD is based on fish filet residue levels, and, consequently, no adjustments in the fish tissue 2,3,7,8-TCDD concentration estimates are necessary. It should be noted that for some species of shellfish (e.g., mollusks) the whole body (minus the shell) is consumed by humans, and the whole-body contaminant concentration would more accurately reflect human exposure.

(3) Drinking water concentration. Drinking water contaminant concentrations are assumed to be the same as the in-stream receiving water concentrations which were calculated using the simple dilution and EXAMS II water column (i.e., dissolved) approaches. It is assumed that (1) water that is ingested is taken from the point of highest in-stream contaminant concentration after the effluent is fully mixed in the receiving stream and (2) the untreated water is ingested as raw stream water with no removal of contaminants.

(4) Human exposures from ingestion of contaminated fish tissue and drinking water. Human exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF from the consumption of contaminated fish tissue is estimated based on fish tissue consumption rates of 6.5, 30, and 140 g/day. The 6.5 g/day consumption rate is equivalent to less than two 1/4 lb meals per month and is cited by USEPA (1980) as the average level of fish and shellfish consumption in the United States. The 30 g/day consumption rate is equivalent to approximately eight 1/4 lb meals per month and is considered applicable for typical recreational fishers. The 140 g/day consumption rate is equivalent to approximately thirty-eight 1/4 lb meals per month and is considered a high consumption rate applicable for subsistence fishers. The 30 g/day and 140 g/day consumption rates are the values used in this report to represent consumption rates for recreational fishers in any area with a large water body and where widespread contamination is evident (USEPA 1989a).

Estimates of human exposure to 2,3,7,8-TCDF through the consumption of contaminated fish tissue are based on the three consumption rates (6.5, 30, and 140 g/day) in combination with fish tissue 2,3,7,8-TCDF

concentration estimates based on a single BCF (1,950) for fish exposed to the contaminant in the water column.

The average daily lifetime exposure (mg of contaminant/kg of body weight/day over a 70-year lifetime) is calculated by multiplying the chemical concentration in the edible fish tissue by the ingestion rate and dividing by an average adult body weight of 70 kg. The equation is represented as follows:

$$LADE = \frac{C \times R}{\text{Body weight (kg)} \times \text{Lifetime (70 yrs)} \times (365 \text{ days/yr})} \quad (6\text{-}3)$$

where:

LADE = Lifetime average daily exposure
C = Concentration in tissue (pg/kg)
R = Consumption rate (kg/day)

The average daily lifetime human exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF from the ingestion of contaminated drinking water is based on a 2 liter/day average lifetime ingestion rate (NAS 1977). The average daily lifetime exposure for a 70 kg adult is determined by multiplying in-stream chemical concentrations by a 2 liter/day average lifetime ingestion rate and then dividing by 70 kg, as follows:

$$LADE = \frac{C \times R}{\text{Body weight (kg)} \times \text{Lifetime (70 yrs)} \times (365 \text{ days/yr})} \quad (6\text{-}4)$$

where:

C = Concentration of chemical in stream (pg/l)
R = Consumption rate (l/day)

6.4 Risk Assessment Methodology

6.4.1 Bioavailable Dose From Ingestion Of Contaminated Fish Tissue And Drinking Water

Not all contaminants that are ingested with fish tissue are available for uptake by humans. Results of a recent review conducted by Boyer (1989) suggest that 85 to 95 percent absorption is a reasonable estimate of 2,3,7,8-TCDD bioavailability in humans from the ingestion of fatty or oily foods, especially milk, fish, and meats. For the present study, the conservative upper limit of this range of bioavailability (95 percent) was used. Therefore, the estimated exposure of humans to 2,3,7,8-TCDD and 2,3,7,8-TCDF from the consumption of contaminated fish is multiplied

by 0.95 to arrive at an estimated human dose. Further characterizations of doses to specific target organs, for example, via a pharmacokinetic analysis, were not conducted.

Boyer (1989) also investigated bioavailability of 2,3,7,8-TCDD from water. Although the author could find no data that specifically addressed the bioavailability of 2,3,7,8-TCDD from drinking water, he assumed that the contaminant would be present at its maximum solubility in water and, therefore, would be 100 percent bioavailable for absorption to the gastrointestinal tract. The present study also assumes that contaminants in drinking water are 100 percent bioavailable.

(1) Estimated cancer risk from ingestion of contaminated fish tissue and drinking water. The average daily lifetime bioavailable dose (for both fish tissue and drinking water contamination) is multiplied by the EPA carcinogenic potency factor for 2,3,7,8-TCDD to calculate a conservative (upper bound) estimate of the hypothetically exposed individual's cancer incidence rate above background incidence rates due to 2,3,7,8-TCDD. The probability of developing cancer in a lifetime due to a given dose of contaminant is represented by the following formula, which estimates a plausible upper limit to excess lifetime risk of cancer at low doses:

$$R = (b)(d) \qquad (6\text{-}5)$$

where:

R = cancer risk
b = the EPA carcinogenic potency factor, $(1.6 \times 10^{-4}$ $(pg/kg/day)^{-1})$
d = lifetime average daily bioavailable dose.

To estimate the combined 2,3,7,8-TCDD/2,3,7,8-TCDF cancer risk, 2,3,7,8-TCDF doses are converted to 2,3,7,8-TCDD toxicity equivalences (TEQs) using a multiplier of 0.1. The TEQ value is then multiplied by the carcinogenic potency factor for 2,3,7,8-TCDD to obtain the combined 2,3,7,8-TCDD/ 2,3,7,8-TCDF risk. The TEQ is generated by using the toxicity equivalency factor (TEF) recommended in Barnes et al. (1989).

In this study, TEQ represents only the contribution of 2,3,7,8-TCDD and 2,3,7,8-TCDF to risk. There are likely to be additional risk contributions from other chlorinated dibenzo-p-dioxins and furans associated with discharges from chlorine-bleaching pulp and paper mills that are not addressed here. However, 2,3,7,8-TCDD and 2,3,7,8-TCDF generally account for more than 90 percent of the TEQ in effluents from chlorine-bleaching pulp and paper mills.

(2) Non-cancer health risks from ingestion of contaminated fish tissue. For risk associated with 2,3,7,8-TCDD and 2,3,7,8-TCDF exposures, cancer is generally considered the most sensitive endpoint.

It is assumed that if individuals are protected from significant concern for cancer, they will also be protected from other endpoint risks such as developmental toxicity, reproductive effects, liver toxicity, immunotoxicity, etc. However, cancer risks are computed assuming an average daily dose over a lifetime of exposure. If individuals were exposed infrequently to relatively high doses over a short period of time, the risks associated with that level of exposure, when averaged over a lifetime, might not be significant in terms of carcinogenic risk. However, the individual could be at risk for other health effects from the short-term exposure.

EPA issues Health Advisories (HAs) for such short-term exposures. HAs are considered doses likely to be without appreciable risk for deleterious effects. The HAs are appropriate for comparison with a single dose or single-day intakes, or short-term exposure. For the purpose of this assessment, one-day and ten-day HAs have been developed for exposure to 2,3,7,8-TCDD. They were derived from animal toxicity data and incorporate uncertainty factors intended to take into account differences in sensitivity between animals and humans, variability in susceptibility within human populations, and other factors. The HAs for exposures to 2,3,7,8-TCDD are as follows: 1 day - 300 pg/kg/day for protection against developmental toxicity effects; 1 day - 100 pg/kg/day for protection against liver effects; and 10 day - 10 pg/kg/day for protection against liver effects (USEPA 1989b; Farmon 1989).

For this assessment, the data for exposures to 2,3,7,8-TCDD and 2,3,7,8-TCDF from the paper mills were screened for exposure scenarios exceeding an average of 100 pg/kg/day. Exposure scenarios exceeding this level were examined in more detail to determine whether the cancer or non-cancer endpoint was the more sensitive indicator of risk.

6.5 Results of the Assessment

The results of this investigation are presented in two parts. The first part addresses exposure estimates and compares the results of the in-stream contaminant concentration calculations that were performed using the two exposure assessment approaches (simple dilution and EXAMS II water column). The second part of the results presents estimated human health risks (individual cancer risks as well as non-cancer health effects) associated with the ingestion of 2,3,7,8-TCDD and 2,3,7,8-TCDF contaminated fish tissue and drinking water.

Sufficient data were not available for all mills investigated to allow a complete evaluation and comparison of results for each of the 104 facilities. For example, for several mills discharging to open waters (i.e., lakes, open ocean), information was not available on the zone of initial dilution in the receiving water. This parameter is necessary for

calculating effluent dilution. For other mills, the accuracy of the data was questionable and new samples were being taken. However, the results of the new sample evaluations were not available for inclusion in this study. In addition, for some mills there was sufficient information to predict risks based on the simple dilution method, but insufficient information to predict risk based on the EXAMS II method. Also, harmonic mean flow data were not available for several facilities. Actual fish tissue concentration data from the National Bioaccumulation Study (NBS) were also evaluated as part of this assessment. The number of facilities evaluated was dependent on the number and location of samples taken as part of the study.

6.5.1 Exposure Assessment Results

(1) <u>In-stream contaminant concentrations</u>. The estimated in-stream 2,3,7,8-TCDD and 2,3,7,8-TCDF concentrations for each of the samples from the 104 mill sites are based on harmonic mean flow (in pg/l). In some instances, more than one sample result is presented for a given mill. Results are ranked by mill in decreasing 2,3,7,8-TCDD and 2,3,7,8-TCDF concentration order (based on the highest sample value per mill).

For each of the samples, estimated in-stream 2,3,7,8-TCDD and 2,3,7,8-TCDF concentrations based on harmonic mean flow were highest when calculated using the simple dilution exposure assessment method. In-stream 2,3,7,8-TCDD concentrations estimated using the simple dilution method ranged from a high of 3.2×10^{2} pg/l to a low of 4.1×10^{-5} pg/l. In-stream 2,3,7,8-TCDF concentrations ranged from a high of 8.0×10^{2} pg/l to a low of 1.0×10^{-4} pg/l. Using the EXAMS II water column method, estimated 2,3,7,8-TCDD concentrations ranged from a high of 8.3×10^{1} pg/l to a low of 3.4×10^{-5} pg/l. Estimated 2,3,7,8-TCDF concentrations ranged from 7.1×10^{2} pg/l to 1.1×10^{-3} pg/l.

The estimated distribution of mills for which discharges result in 2,3,7,8-TCDD and 2,3,7,8-TCDF concentrations falling within specific concentration ranges (based on harmonic mean flow) are presented using the simple dilution method (Figure 6-1) and EXAMS II water column method (Figure 6-2).

(2) <u>Fish tissue contaminant concentrations</u>. The mill-specific fish tissue concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF were also estimated using the two exposure assessment methods. The actual fish tissue concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF were measured during the National Bioaccumulation Study.

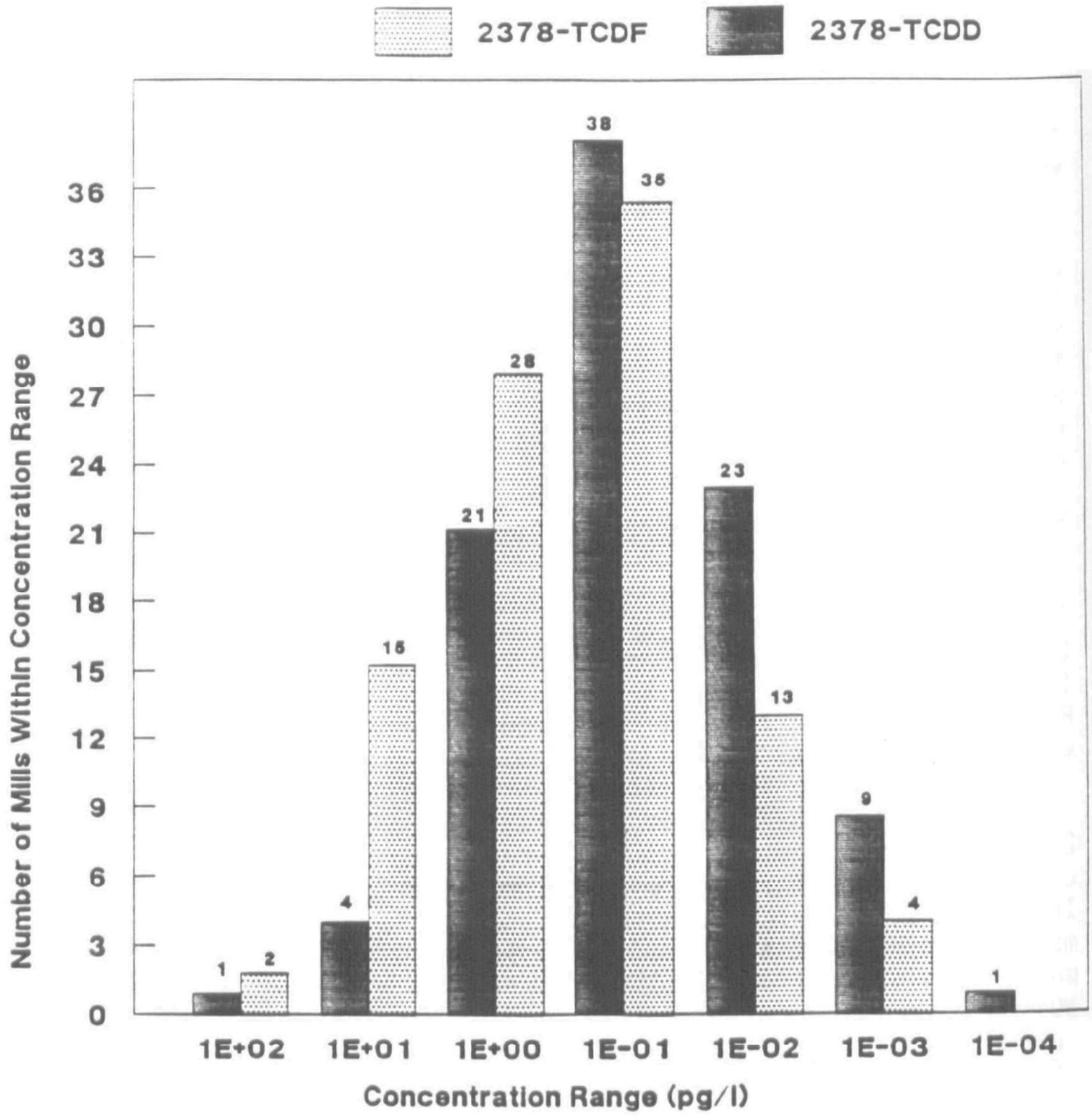

FIGURE 6-1. **Distribution of the number of mills for which discharges would result in a given range of water column contaminant concentrations as estimated by the simple dilution method.**

Notes:

Total number of mills evaluated = 97.
Estimates based on Harmonic Mean Flow of receiving waters.

Number of mills within concentration ranges for which 2378-TCDD and/or 2378-TCDF were not detected in the effluent and therefore water column concentration estimates are based on effluent concentrations of 1/2 the detection limit:

	1E+0	1E-1	1E-2	1E-3
TCDD	1	9	7	4
TCDF		3	3	1

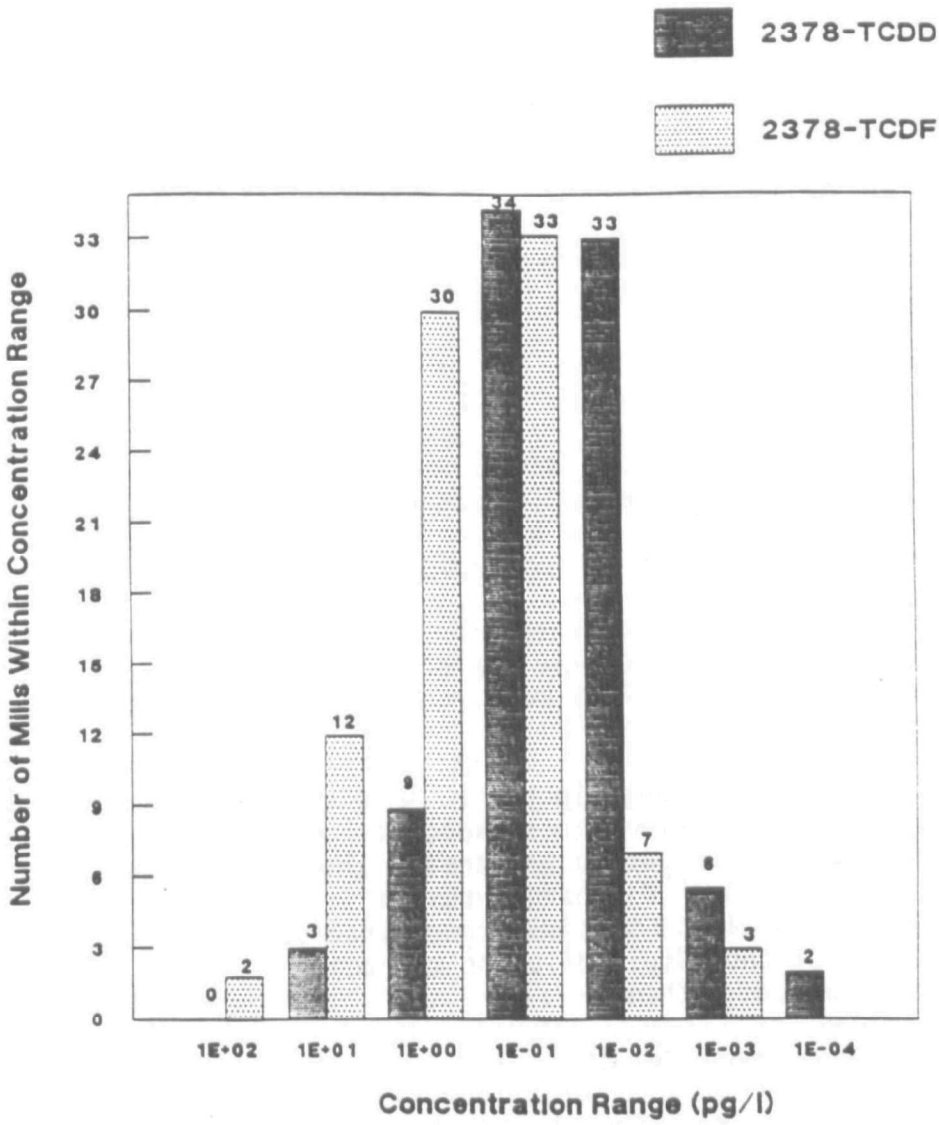

Figure 6-2. **Distribution of the number of mills for which discharges would result in a given range of water column contaminant concentrations as estimated by the EXAMS II water column method.**

Notes:

Total number of mills evaluated = 87.

Estimates based on Harmonic Mean Flow of receiving waters.

Number of mills within concentration ranges for which 2378-TCDD and/or 2378-TCDF were not detected in the effluent and therefore water column concentration estimates are based on effluent concentrations of 1/2 the detection limit.

	1E+0	1E-1	1E-2	1E-3	1E-4
TCDD	1	7	6	4	2
TCDF	1	2	4	1	

The highest fish tissue concentrations due to in-stream exposure to the contaminants were estimated by the simple dilution method. The 2,3,7,8-TCDD fish tissue concentrations estimated using the BCF of 5,000 ranged from a high of 1.6×10^{3} ng/kg to a low of 2.05×10^{-4} ng/kg. Using the BCF of 50,000, 2,3,7,8-TCDD fish tissue concentrations ranged from a high of 1.6×10^{4} ng/kg to a low of 2.0×10^{-3} ng/kg. Use of the simple dilution method estimated 2,3,7,8-TCDF concentrations in fish tissue (using the single BCF of 1,950) ranging from 1.6×10^{3} ng/kg to 2.0×10^{-4} ng/kg.

The EXAMS II water column method resulted in fish tissue concentrations of 2,3,7,8-TCDD ranging from a high of 4.2×10^{2} ng/kg to a low of 1.71×10^{-4} ng/kg using the 5,000 BCF and from 4.2×10^{3} ng/kg to 1.2×10^{-3} ng/kg using the 50,000 BCF. The 2,3,7,8-TCDF fish tissue concentrations estimated by the EXAMS II water column method ranged from 1.4×10^{3} ng/kg to 1.5×10^{-3} ng/kg.

Measured 2,3,7,8-TCDD concentrations in fish tissue from the National Bioaccumulation Study ranged from a high of 7.2×10^{1} ng/kg to a low of 2.0×10^{-1} ng/kg. 2,3,7,8-TCDF measured values ranged from 2.1×10^{2} ng/kg to 1.3×10^{-1} ng/kg.

(3) Drinking water contamination. This study assumes that the concentrations of the contaminants expected to be found in drinking water are the same as those predicted in-stream. The distribution of the number of mills for which discharges result in in-stream concentrations of the contaminants within specific concentration ranges are illustrated in Figure 6-1 (for the simple dilution method) and Figure 6-2 (for the EXAMS II water column method).

6.5.2 Risk Assessment Results

(1) Bioavailable dose from ingestion of fish tissue and drinking water. The bioavailable dose to humans from consumption of contaminated fish tissue was calculated based on 95 percent bioavailability and three fish tissue consumption rates: 6.5 g/day in combination with fish tissue concentrations based on fish bioconcentration factors of 5,000 for 2,3,7,8-TCDD and 1,950 for 2,3,7,8-TCDF; and 30 and 140 g/day in combination with fish tissue concentrations based on fish bioconcentration factors of 50,000 for 2,3,7,8-TCDD and 3,900 for 2,3,7,8-TCDF. The bioavailable dose from drinking water was calculated based on a drinking water ingestion rate of 2 L/day and a 100 percent oral dose bioavailability. The mill-specific bioavailable doses of 2,3,7,8-TCDD and 2,3,7,8-TCDF from consumption of contaminated fish tissue are estimated based on both the simple dilution and EXAMS II methods. Mill-specific bioavailable doses from ingestion of contaminated drinking water are also estimated. These values were used to predict the hypothetically exposed individual's upper bound cancer risks associated with discharges from each mill.

(2) Estimated cancer risk from ingestion of contaminated fish tissue and drinking water. The mill-specific upper bound lifetime risks of cancer to the hypothetically exposed individual from consumption of contaminated fish tissue are predicted based on the simple dilution and EXAMS II methods. The mill-specific upper bound risks of cancer from ingestion of contaminated drinking water are also predicted . The values presented are in toxicity equivalents (TEQs), representing the combined impacts of 2,3,7,8-TCDD and 2,3,7,8-TCDF. The cancer risks associated with contaminated fish consumption are presented for 6.5 g/day, 30 g/day, and 140 g/day consumption rates. The percent 2,3,7,8-TCDD contributing to TEQ is also estimated for contaminated fish tissue consumption and for contaminated drinking water ingestion.

(a) **Contaminated fish tissue.** Figures 6-3 through 6-6 present the estimated distribution of the number of mills for which discharges would result in a given range of estimated lifetime cancer risks for the hypothetically exposed individual due to the consumption of contaminated fish tissue based on the simple dilution exposure assessment method and the EXAMS II water column exposure assessment method. Estimated values are in TEQs.

The results of calculations using the 6.5 g/day fish tissue consumption rate in combination with the BCF of 5,000 are considered to be reasonable worst-case scenarios. The results of these calculations are therefore presented separately from the results of calculations using the 30 and 140 g/day consumption rates in combination with the BCF of 100,000, which are considered more extreme worst-case scenarios.

Using the simple dilution exposure assessment estimates, the 6.5 g/day fish tissue consumption rate, and the fish tissue contaminant concentrations based on a BCF of 5,000, the upper bound mill-specific cancer rates for the hypothetically exposed individual range from the 10^{-7} to 10^{-8} risk levels (Figure 6-3). Risk levels associated with discharges from 80 of the 97 mills evaluated (82 percent) fell within the 10^{-4} to 10^{-6} risk levels, with 35 mills within the 10^{-5} risk level.

Mill-specific cancer rate estimates using the 30 g/day fish tissue consumption rate and fish tissue contaminant concentrations based on a BCF of 50,000 range from the $\geq 10^{-1}$ to 10^{-6} risk levels (Figure 6-4). Seventy of the 97 mills (71 percent) were associated with risk levels between 10^{-3} to 10^{-4}, and 40 of these 70 fell within the 10^{-3} range. Using the 140 g/day fish tissue consumption rate and fish tissue contaminant concentrations based on the 50,000 BCF, risk levels range from $\geq 10^{-1}$ to 10^{-6} (Figure 6-4). Sixty-six out of the 97 mills (68 percent) were associated with risk levels between 10^{-2} to 10^{-3} with 39 within the 10^{-3} range.

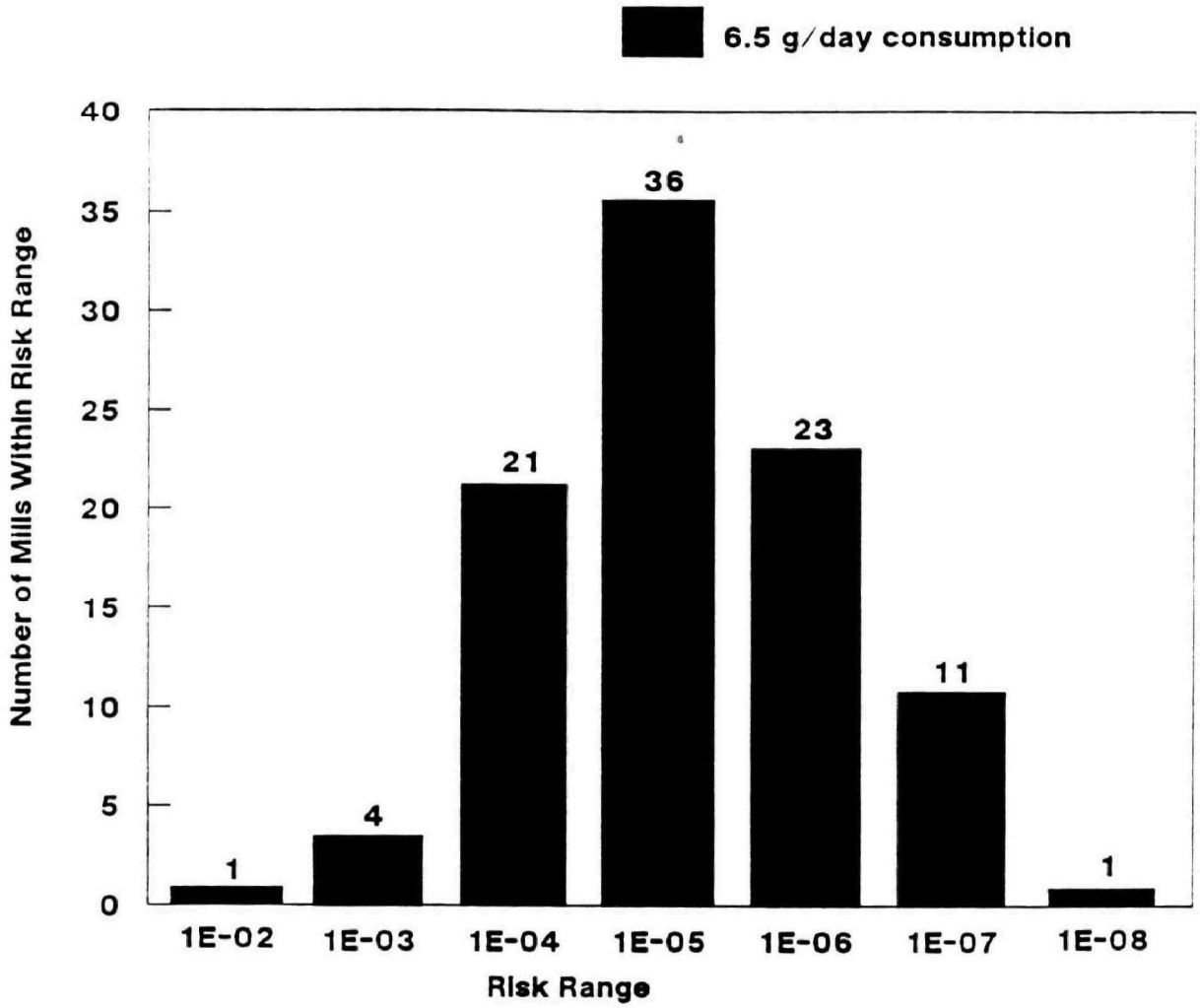

FIGURE 6-3. Distribution of the number of mills for which discharges would result in a given range of lifetime cancer risk due to the consumption of contaminated fish tissue as estimated by the simple dilution method (6.5 g/day consumption rate and BCF of 5,000 for 2378-TCDD*).

Notes:

Total number of mills evaluated = 97.

Combined 2378 -TCDD/-TCDF risk predicted using TEQ.

Number of mills within risk ranges for which 2378-TCDD and/or 2378-TCDF were not detected in the effluent and therefore risk estimates are based on effluent concentrations of 1/2 the detection limit:

	1E-4	1E-5	1E-6	1E-7
TCDD	2	7	4	3
TCDF		1		1
TCDD & TCDF		2	2	1

*** Recent laboratory evidence indicates that a BCF higher than 5,000 for 2378-TCDD (e.g., 50,000) more accurately reflects uptake of 2378-TCDD by fish. Use of a BCF of 50,000 for 2378-TCDD would increase risk by an order of magnitude.**

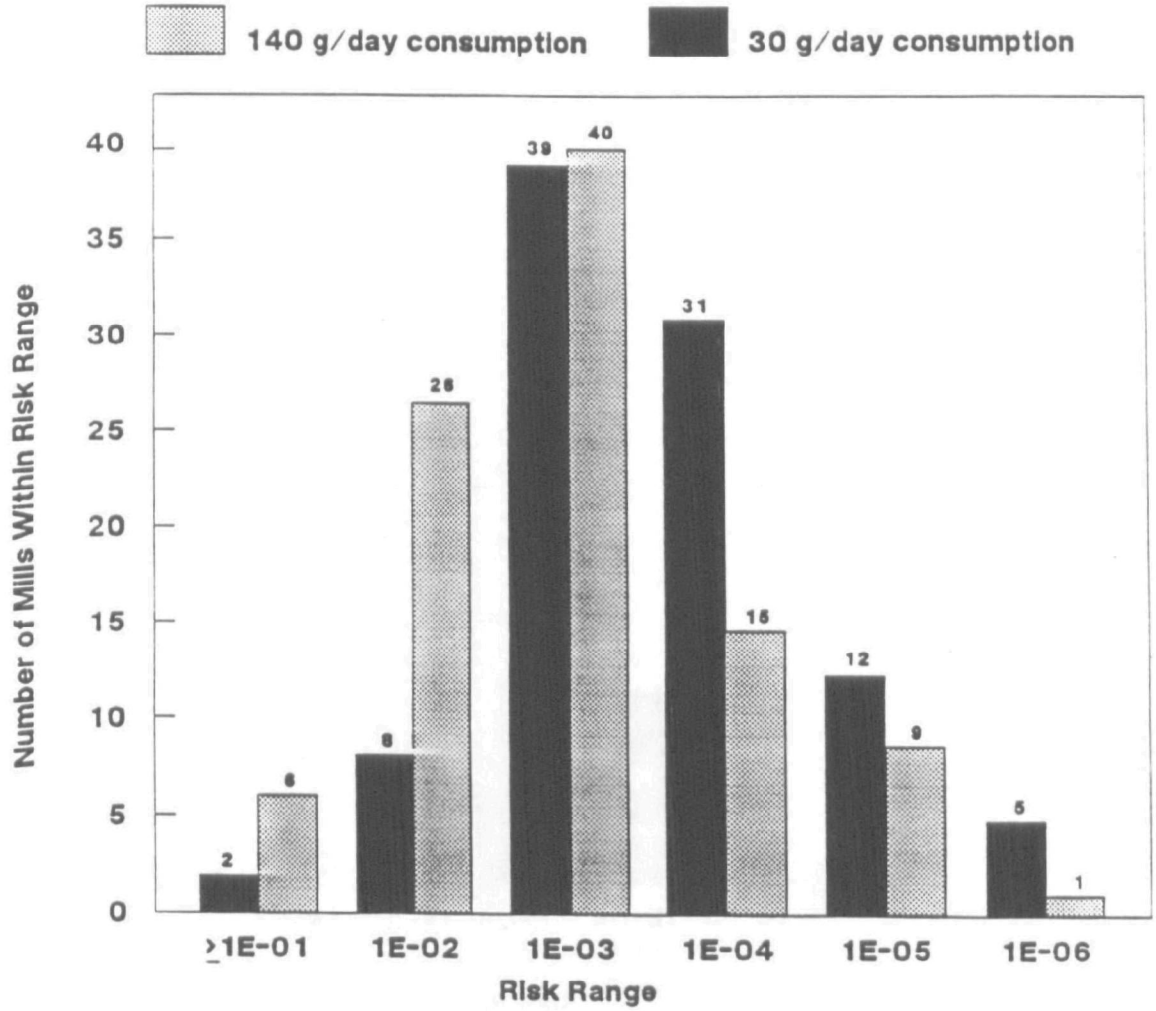

FIGURE 6-4. **Distribution of the number of mills for which discharges would result in a given range of lifetime cancer risk due to the consumption of contaminated fish tissue as estimated by the simple dilution method (30 and 140 g/day consumption rates and BCF of 50,000 for 2378-TCDD).**

Notes:

Total number of mills evaluated = 97.

Combined 2378 -TCDD/-TCDF risk predicted using TEQ.

Number of mills within risk ranges for which 2378-TCDD and/or 2378-TCDF were not detected in the effluent and therefore risk estimates are based on effluent concentrations of 1/2 the detection limit:

	1E-2	1E-3	1E-4	1E-5	1E-6
30g/day					
TCDD		7	4	3	2
TCDF		1		1	
TCDD & TCDF		1	3		1
140g/day					
TCDD	4	4	5	3	
TCDF		1	1		
TCDD & TCDF		3	1	1	

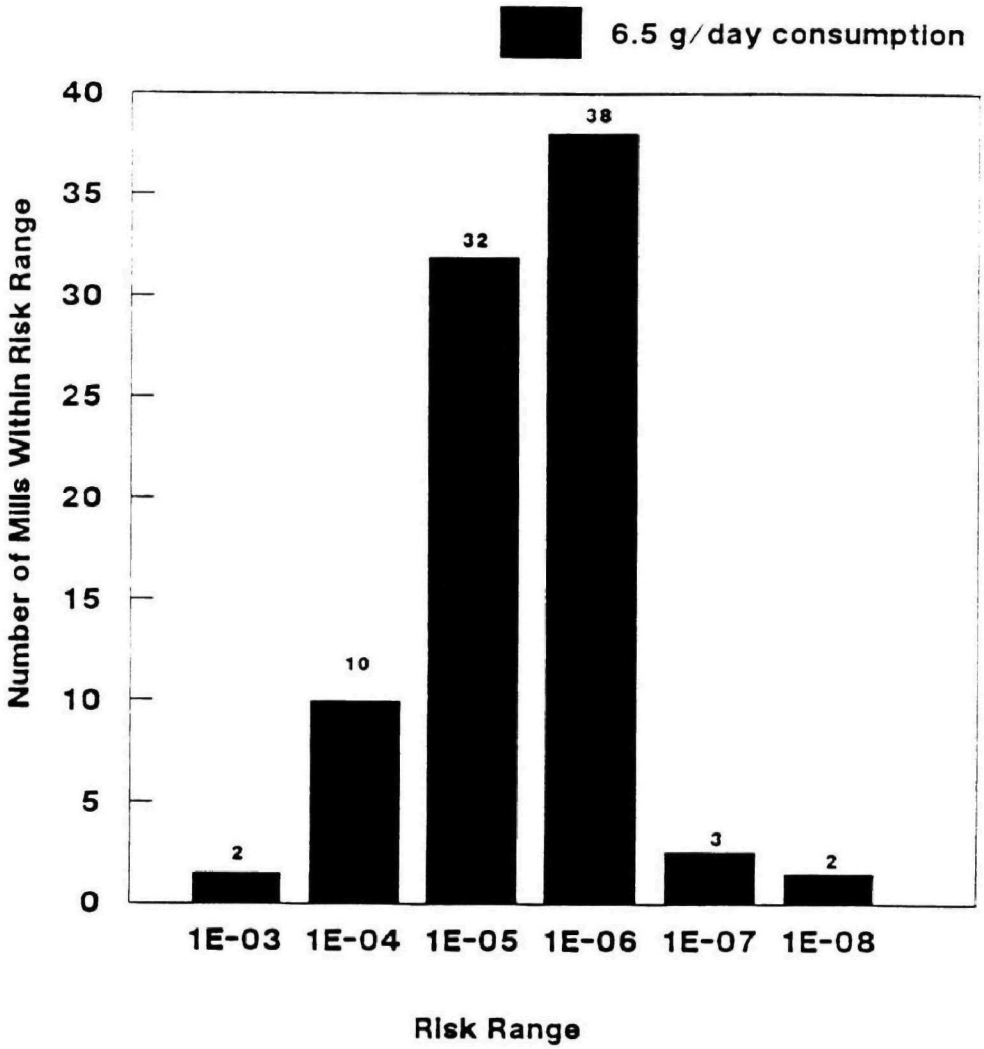

FIGURE 6-5. **Distribution of the number of mills for which discharges would result in a given range of lifetime cancer risk due to the consumption of contaminated fish tissue as estimated by the EXAMS II method (6.5 g/day consumption rate and BCF of 5,000 for 2378-TCDD*).**

Notes:

Total number of mills evaluated = 87.

Combined 2378 -TCDD/-TCDF risk predicted using TEQ.

Number of mills within risk ranges for which 2378-TCDD and/or 2378-TCDF were not detected in the effluent and therefore risk estimates are based on effluent concentrations of 1/2 the detection limit:

	1E-4	1E-5	1E-6	1E-7	1E-8
TCDD	1	6	5	2	1
TCDF		1		1	
TCDD & TCDF		1	3		1

*** Recent laboratory evidence indicates that a BCF higher than 5,000 for 2378-TCDD (e.g., 50,000) more accurately reflects uptake of 2378-TCDD by fish. Use of a BCF of 50,000 for 2378-TCDD would increase risk by an order of magnitude.**

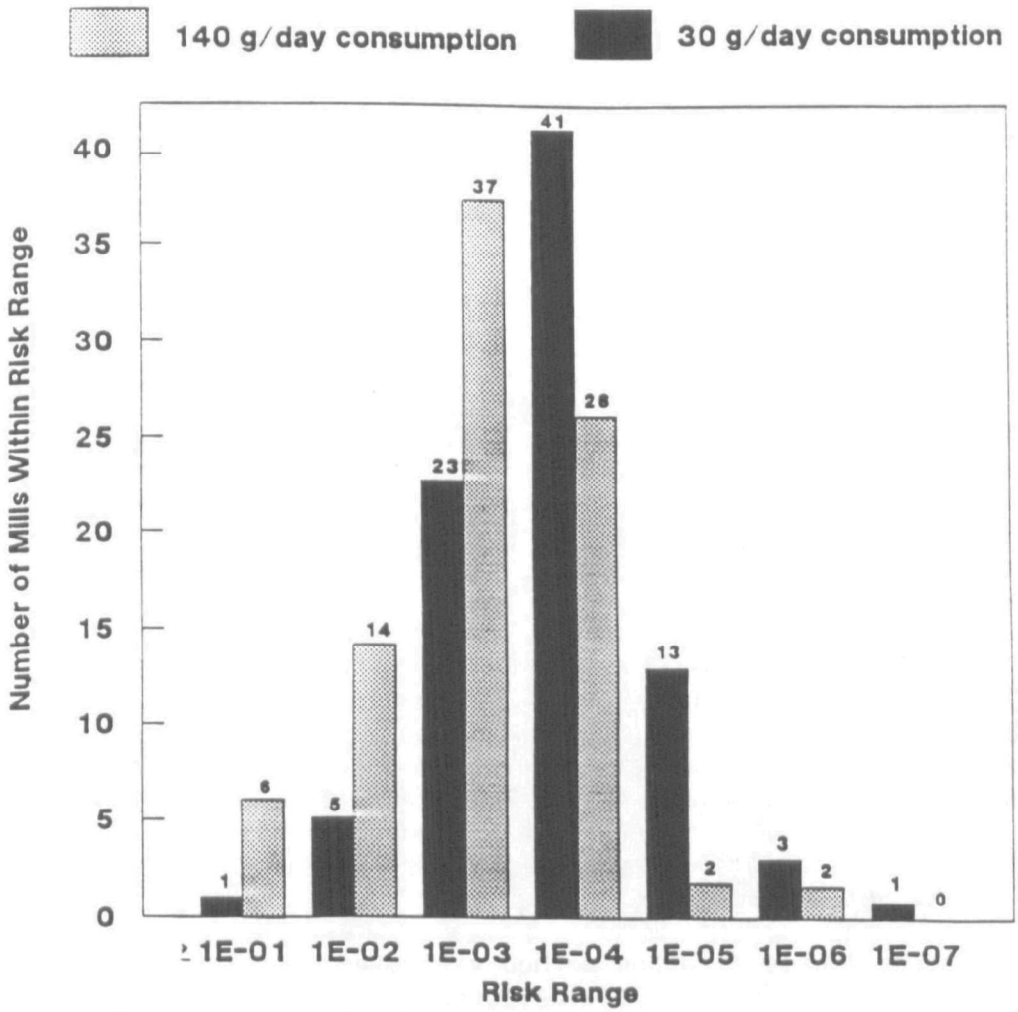

FIGURE 6-6. Distribution of the number of mills for which discharges would result in a given range of lifetime cancer risk due to the consumption of contaminated fish tissue as estimated by the EXAMS II method (30 and 140 g/day consumption rates and BCF of 50,000 for 2378-TCDD).

Notes:
Total number of mills evaluated = 87.
Combined 2378 -TCDD/-TCDF risk predicted using TEQ.

Number of mills within risk ranges for which 2378-TCDD and/or 2378-TCDF were not detected in the effluent and therefore risk estimates are based on effluent concentrations of 1/2 the detection limit:

	1E-2	1E-3	1E-4	1E-5	1E-6	1E-7
30g/day						
TCDD		4	6	2	2	
TCDF			1		1	
TCDD & TCDF			2	2		1
140g/day						
TCDD	1	7	4	1	1	
TCDF		1		1		
TCDD & TCDF		1	3		1	

Mill-specific upper bound cancer rate estimates for the hypothetically exposed individual using the EXAMS II water column exposure assessment method, the 6.5 g/day fish tissue consumption rates, and the fish tissue contaminant concentrations based on a BCF of 5,000 range from the 10^{-3} to 10^{-8} risk levels (Figure 6-5). Seventy of the 87 mills evaluated (80 percent) were associated with risk levels between 10^{-5} (33 mills) to 10^{-6} (37 mills).

Using the 30 g/day consumption rate and fish tissue contaminant concentrations based on the BCF of 50,000, mill-specific cancer rates range from the $\geq 10^{-1}$ to 10^{-7} risk levels (Figure 6-6). Sixty-four of the 87 mills (74 percent) were associated with risk levels within the 10^{-3} to 10^{-4} range, and 40 of these fell within the 10^{-4} range. Cancer risk estimates using the 140 g/day fish tissue consumption rate and the BCF of 50,000, range from the $\geq 10^{-1}$ to 10^{-6} risk levels (Figure 6-6). Sixty-two of the 87 mills (71 percent) were associated with risk levels between the 10^{-3} and 10^{-4} range, and 37 of these fall within the 10^{-3} range.

(b) Contaminated drinking water. Figures 6-7 and 6-8 present the distribution of the number of mills for which discharges are estimated to result in a given range of upper bound lifetime cancer risks to the hypothetically exposed individual due to the ingestion of contaminated drinking water. Only those facilities discharging to fresh water lakes, rivers, and streams are included in this analysis. No discharges to marine or estuarine waters are included, since these water bodies would not be used as drinking water sources.

Use of the simple dilution method estimated that the cancer risks associated with the 69 mills evaluated range from the 10^{-4} to 10^{-9} risk levels (Figure 6-7). The greatest percentage of these mills (44, or 64 percent) were associated with risk levels within the 10^{-6} (23 mills) to 10^{-7} (21 mills) range. Use of the EXAMS II water column method estimated that the risk levels associated with the 64 mills evaluated range from the 10^{-5} to 10^{-9} levels (Figure 6-8). Fifty of these mills (78 percent) were associated with risk levels between the 10^{-6} (18 mills) to 10^{-7} (32 mills) range.

(3) Non-cancer health effects from ingestion of contaminated fish tissue. The mill-specific human doses from the consumption of a single 115 gram (1/4 pound) portion of contaminated fish tissue (using a BCF of 50,000 for 2,3,7,8-TCDD and a BCF of 1,950 for 2,3,7,8-TCDF) were estimated based on the simple dilution and EXAMS II water column exposure assessment methods. The values are presented in 2,3,7,8-TCDD and TEQ doses. Results are reported in pg/kg/day for comparison to the estimated one-day Health Advisory for protection against liver effects (100 pg/kg/day).

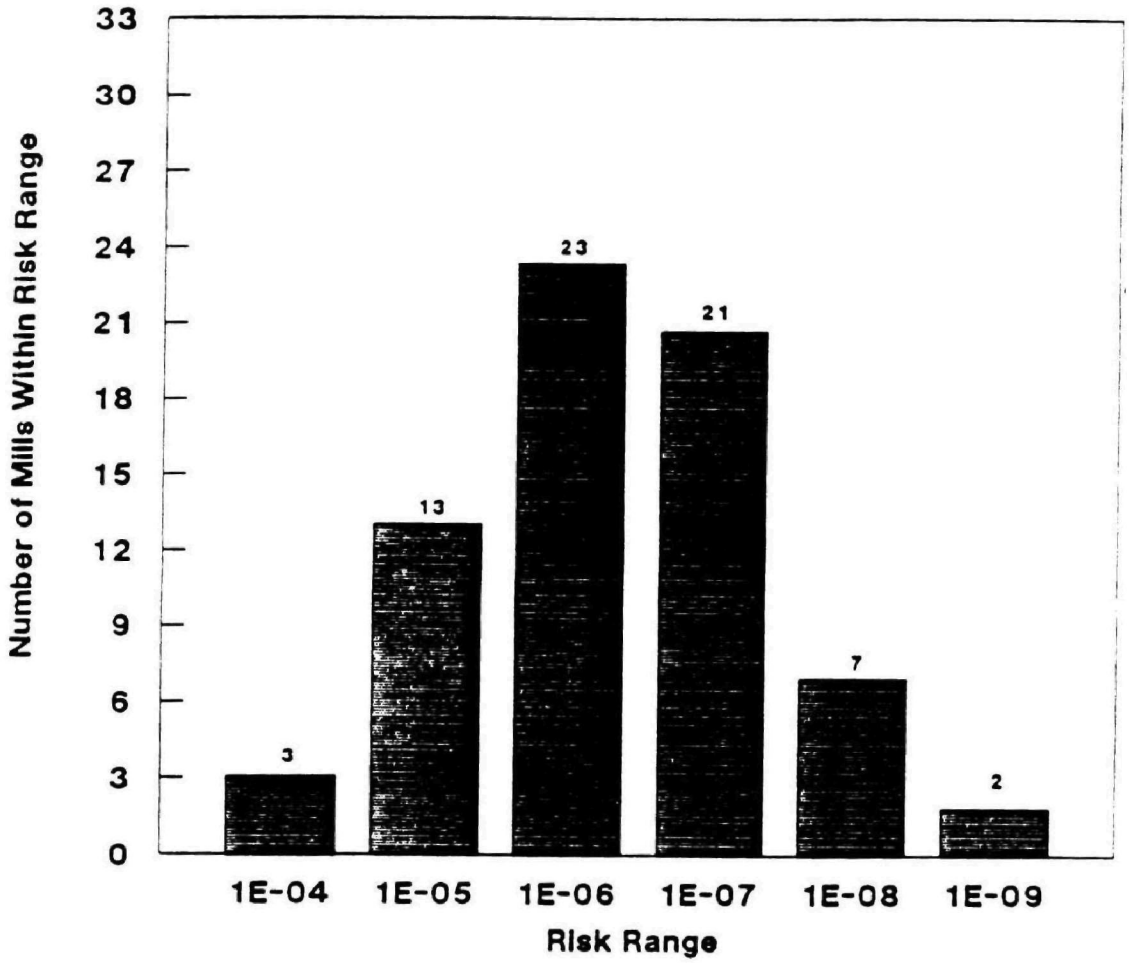

Figure 6-7. **Distribution of the number of mills for which discharges would result in a given range of lifetime cancer risk due to the ingestion of contaminated drinking water as estimated by the simple dilution method.**

Notes:

Total number of mills evaluated = 69.

Combined 2378 -TCDD/-TCDF risk predicted using TEQ.

Based on a 2 L/day ingestion rate.

Number of mills within risk ranges for which 2378-TCDD and/or 2378-TCDF were not detected in the effluent and therefore risk estimates are based on effluent concentrations of 1/2 the detection limit:

	1E-5	1E-6	1E-7	1E-8	1E-9
TCDD	1	3	3	3	
TCDF		1		1	
TCDD & TCDF		1	2		1

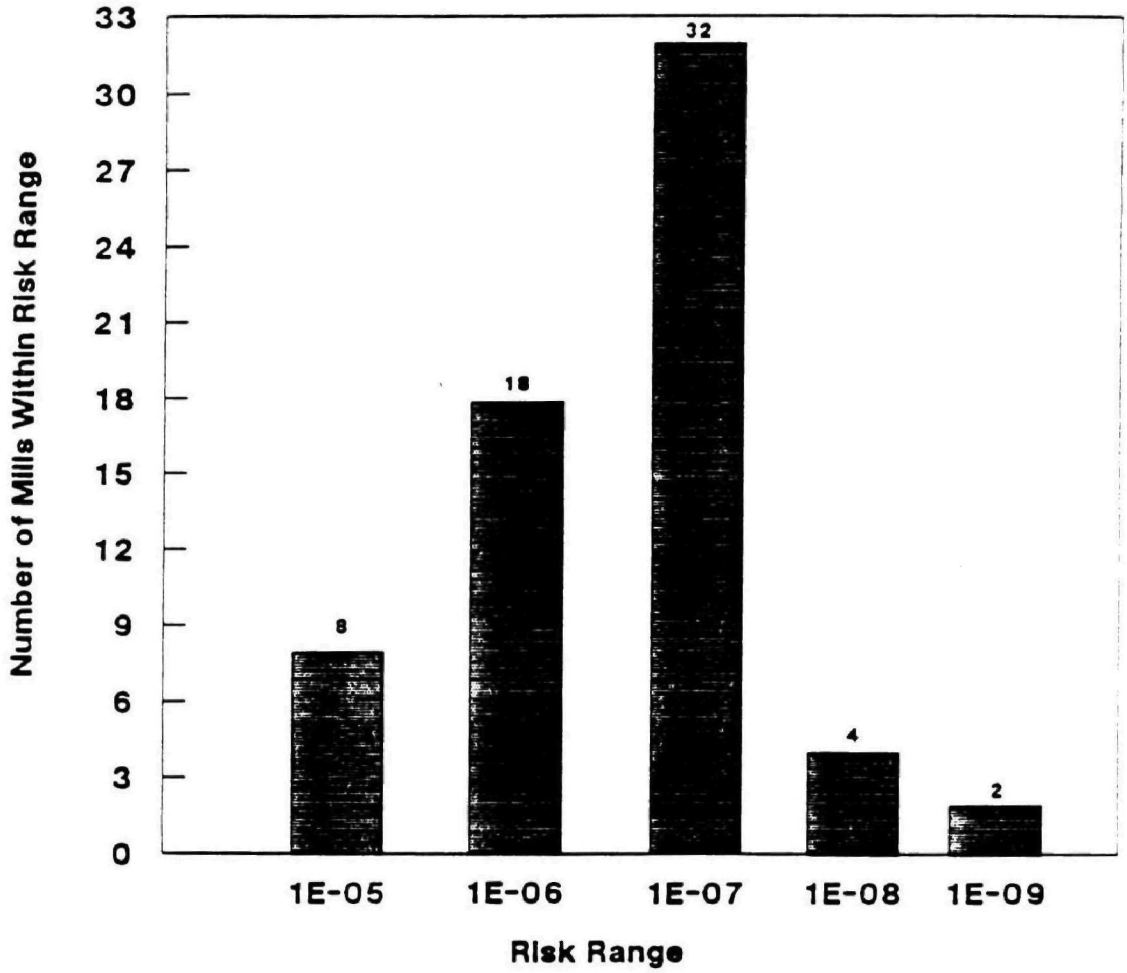

Figure 6-8. **Distribution of the number of mills for which discharges would result in a given range of lifetime cancer risk due to the ingestion of contaminated drinking water as estimated by the EXAMS II method.**

Notes:

Total number of mills evaluated = 64.

Combined 2378 -TCDD/-TCDF risk predicted using TEQ.

Based on a 2 L/day ingestion rate.

Number of mills within risk ranges for which 2378-TCDD and/or 2378-TCDF were not detected in the effluent and therefore risk estimates are based on effluent concentrations of 1/2 the detection limit:

	1E-5	1E-6	1E-7	1E-8	1E-9
TCDD	1	2	4	2	1
TCDF			1	1	
TCDD & TCDF			3	1	1

Based on the simple dilution method results (Figure 6-9), the dose associated with discharges from 29 of the 97 mills evaluated (27 percent) would equal or exceed the estimated one-day HA dose for protection from liver effects (100 pg/kg/day). Use of the EXAMS II method (Figure 6-10) estimates that the dose associated with discharges from nine mills of the 87 evaluated (10 percent) would equal or exceed the 100 pg/kg/day dose level.

6.6 Discussion of Results

6.6.1 Assumptions, Limitations, and Uncertainties

This section presents the assumptions that were made during the planning and conduct of this study and discusses significant results and the limitations and uncertainties associated with those results. The following is a list of assumptions used in this investigation:

- Mill-specific, five-day composite effluent contaminant concentrations were multiplied by mean plant flow rates to determine contaminant load. This resulting load to the receiving water is assumed to be continuous. The representativeness of the effluent sample as reflecting long-term mill operations is unknown; since then, the mills may have made plant process or operation changes to reduce dioxin and furan formation.

- The highest estimated in-stream concentrations in the immediate vicinity of the discharges (assuming steady-state, fully mixed conditions) were considered for fish exposure.

- Receiving water stream flow rates for estimating human health risks were calculated using the harmonic mean of historic flow measurements from nearby stream gaging stations. These flows may not be the same as those used by specific states to assess risk.

- Three bioconcentration factor (BCF) values were used for estimating 2,3,7,8-TCDD and 2,3,7,8-TCDF concentrations in fish: two for 2,3,7,8-TCDD and one for 2,3,7,8-TCDF. The resulting fish tissue concentrations were used to estimate human exposure to the contaminants through consumption of fish tissue. For 2,3,7,8-TCDD, a BCF of 5,000 was used in combination with a human consumption rate of fish tissue of 6.5 g/day, and a BCF of 50,000 was used in combination with consumption rates of 30 g/day and 140 g/day. The 6.5 g/day fish tissue consumption rate in combination with the BCF of 5,000 is considered a reasonable estimate for an average consumer of locally caught fish. The 30 and 140 g/day consumption rates in combination with the BCF of 100,000 are used as sensitivity comparisons and represent more extreme exposure scenarios for recreational and subsistence fishers. A single BCF for 2,3,7,8-TCDF of 3,900 was used in combination with each of the three consumption rates.

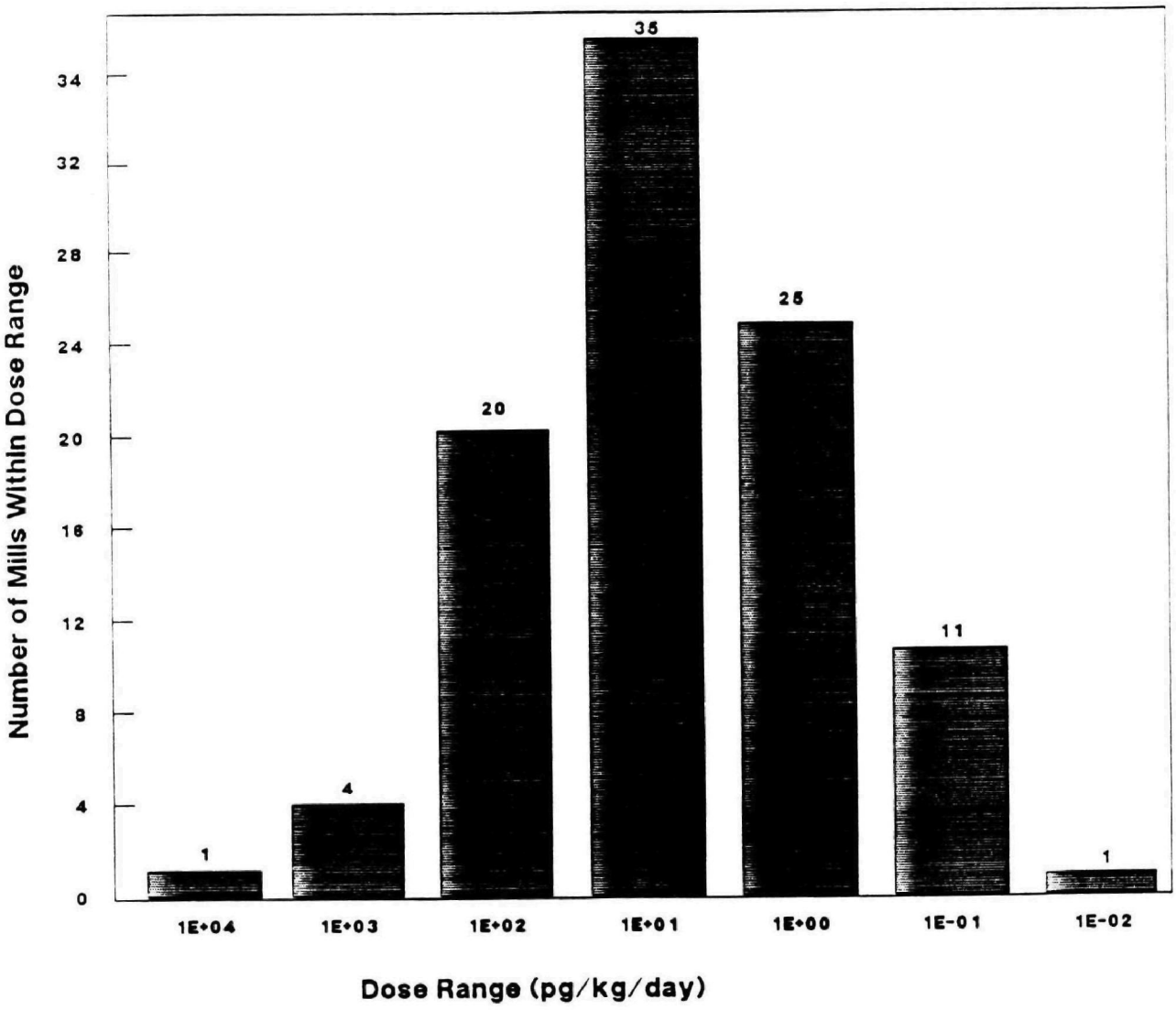

FIGURE 6-9. **Distribution of the number of mills for which discharges would result in a given range of human doses from a one-time exposure to contaminated fish tissue as estimated by the simple dilution method.**

Notes:

Total number of mills evaluated = 97.

Combined 2378 -TCDD/-TCDF dose predicted using TEQ.

Based on the consumption of a single 115 g portion of contaminated fish tissue and using a fish filet BCF of 50,000 for 2378-TCDD.

Number of mills within dose ranges for which 2378-TCDD and/or 2378-TCDF were not detected in the effluent and therefore dose estimates are based on effluent concentrations of 1/2 the detection limit:

	1E+2	1E+1	1E+0	1E-1
TCDD	1	7	4	3
TCDF		1		1
TCDD & TCDF		2	3	1

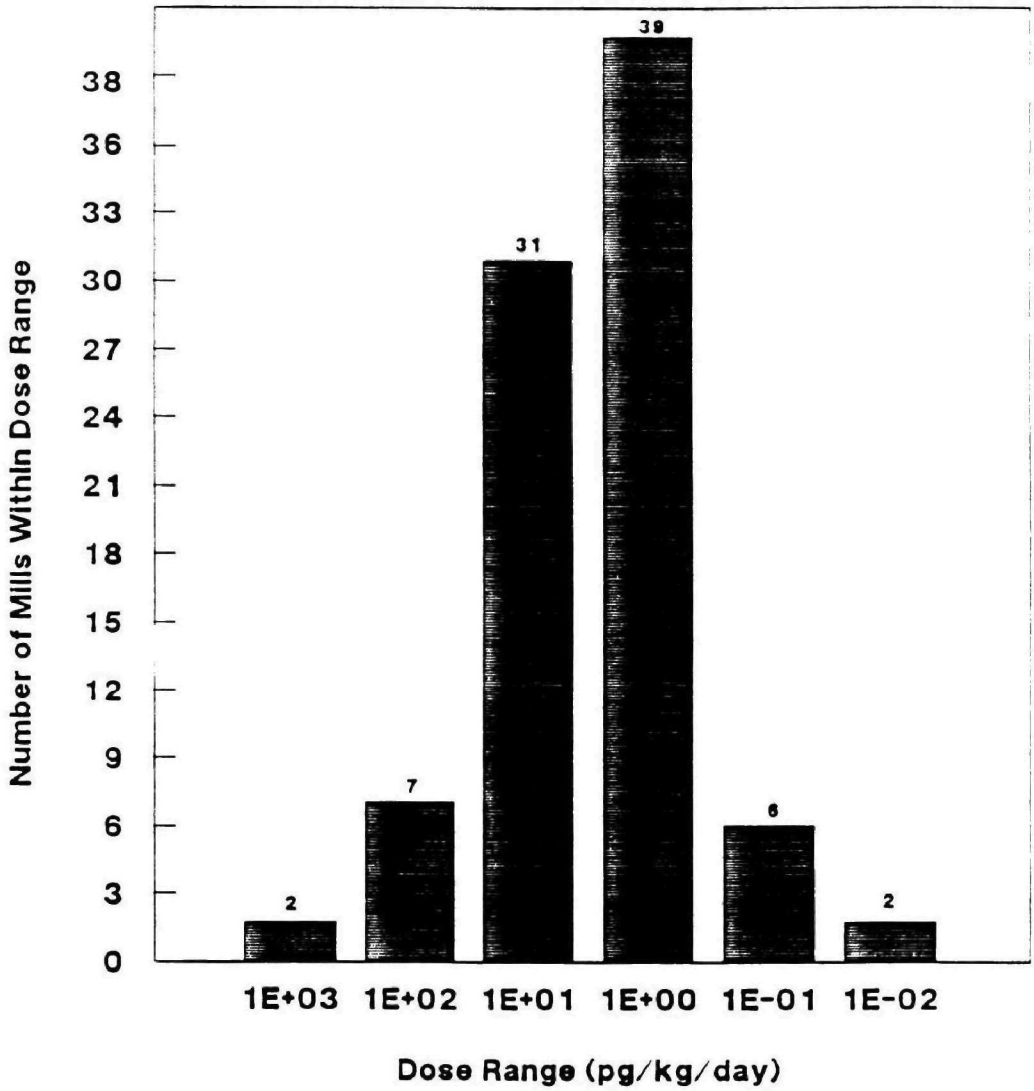

FIGURE 6-10. **Distribution of the number of mills for which discharges would result in a given range of human doses from a one-time exposure to contaminated fish tissue as estimated by EXAMS II method.**

Notes:

Total number of mills evaluated = 87.

Combined 2378 -TCDD/-TCDF dose predicted using TEQ.

Based on the consumption of a single 115 g portion of contaminated fish tissue and using a fish filet BCF of 50,000 for 2378-TCDD

Number of mills within dose ranges for which 2378-TCDD and/or 2378-TCDF were not detected in the effluent and therefore dose estimates are based on effluent concentrations of 1/2 the detection limit:

	1E+2	1E+1	1E+0	1E-1	1E-2
TCDD	1	5	5	2	1
TCDF		1		1	
TCDD & TCDF		1	3	1	1

- A drinking water ingestion rate of 2 l/day was used to estimate human exposures through ingestion of contaminated drinking water. It was assumed that the water consumed is taken from the point of highest in-stream pollutant concentration after the effluent is fully mixed in the receiving stream, and no treatment of the water is undertaken to remove contaminants prior to ingestion.

- Fish tissue contaminant bioavailability for humans was assumed to be 95 percent of oral dose. Contaminants in water were assumed to be 100 percent bioavailable to both fish and humans.

- Fish were assumed to be exposed to contaminants only in the water column. No food chain or sediment associated exposures were considered, other than for the simple dilution method in which the total in-stream contaminant level (both dissolved and adsorbed to suspended solids) were bioavailable.

In evaluating the results of this assessment, it should be noted that BCFs are highly species specific. The BCF for a contaminant in a given fish species is dependent on fish tissue lipid content, mode of contaminant uptake, and other factors. Thus, using a single BCF does not take into account interspecies differences in the rate and degree of contaminant bioconcentration. For example, the study conducted by Cook et al. (Unpublished) indicates that BCF values of 200,000, higher than those used in this study, may be applicable for 2,3,7,8-TCDD for some species of fish. Also, the 50,000 BCF for 2,3,7,8-TCDD used in conjunction with fish consumption rates of 30 and 140 g/day for recreational and susistence fishers is based on the assumption that only the filet portion of the fish is consumed. However, some subpopulations of subsistence fishers and certain ethnic groups eat whole fish in which the concentration of contaminants is likely to be higher than in the filet alone. Therefore, the use of a BCF of 50,000 may underestimate risks to these subpopulations.

The predictions also do not take into consideration the mobility of fish in the receiving waters. Both resident and migrating species will move in and out of the discharge area. This analysis assumed that the fish remain exposed to the predicted contaminant concentration up to the time they are caught, thus resulting in a conservative estimate of aquatic life impacts and human health risk.

No attempt has been made to estimate fish exposure to contaminants associated with suspended particulates, bed sediments, or the food chain becuase of a lack of sufficient and appropriate scientific data and understanding of the bioaccumulation of these contaminants by fish through these routes of exposure. An exception is in considering the results of the simple dilution method in which total contaminant concentrations, both dissolved and adsorbed to suspended particulates,

are evaluated. It is evident that food and sediment provide exposure routes to fish downstream, where the amount of dissolved 2,3,7,8-TCDD and 2,3,7,8-TCDF available for uptake across gills becomes much less; thus, the assumption that fish remain in the area immediately downstream from the point of discharge is sufficiently conservative to compensate for any lack of food chain or sediment associated exposure components. In addition, as a check and a sensitivity comparison, the results of the simple dilution calculation are considered to provide an upper bound on fish tissue contaminant levels since 100 percent of the in-stream contaminants are assumed to be bioavailable.

The assumed fish tissue consumption rates also have an impact on results of this assessment. The fish tissue consumption rate of 6.5 g/day (or less than two 1/4 lb meals per month) is considered an average level of fish and shellfish consumption in the United States. However, there appears to have been a significant increase in fish consumption rates in the United States since 1980 when the national average fish consumption rate of 6.5 g/day was derived. Therefore, risks estimated based on this consumption rate may, in some cases, significantly underestimate risk.

It should also be noted that, if multiple discharges to the same waterbody are present, the actual risk associated with a waterbody may be substantially greater than estimated in this study. For example, there are several chlorine-bleaching pulp and paper mills that discharge to the Columbia River basin. Calculations in this report assume that each mill discharges to a receiving stream with no background level of contamination. Therefore, in the case of multiple discharges to a receiving stream, estimating risks from one mill alone can result in a significant underestimate of risk.

The simple dilution method assumes that all contaminants in the water column, both dissolved and adsorbed to suspended solids, are bioavailable. As a result, for each of the mills analyzed, the simple dilution exposure assessment method resulted in higher contaminant concentrations and human health risks than did the EXAMS II water column method. The EXAMS II water column method, on the other hand, considers only those contaminants in the dissolved phase. In cases where the receiving water TSS was relatively low, the simple dilution and EXAMS II water column results are comparable. However, when suspended solids concentrations were high, the EXAMS II method estimated risks significantly lower than those predicted by the simple dilution method. Therefore, in those water bodies with relatively high suspended solids content, the EXAMS II method likely underestimated human health risks from consumption of contaminated fish tissue, since fish exposure to sediment-absorbed contaminants was not considered.

Results of this assessment indicate that the fish tissue exposure route poses a greater human cancer risk to the hypothetically exposed individual than does the drinking water exposure route. However, the upper bound cancer risk estimated from consumption of contaminated fish tissue based on the 6.5 g/day consumption rate and the BCF of 5,000 are relatively close to the cancer risk estimates based on ingestion of contaminated drinking water. It should be noted that fish tissue consumption may not pose a greater risk to the entire population than ingestion of contaminated drinking water. Determining which exposure route poses the greatest risk to the entire population requires knowledge of the number of persons eating contaminated fish tissue versus the number of persons who use contaminated surface water as a drinking water source. Such a population assessment was not conducted for this assessment.

A comparison of the cancer versus non-cancer risks associated with 2,3,7,8-TCDD and 2,3,7,8-TCDF discharges from pulp and paper mill effluents indicates that more mills would result in potential cancer risks than would result in non-cancer risks. However, the non-cancer risk may actually be the more sensitive end point. The cancer risk was estimated for the lifetime of a continuously exposed individual. The non-cancer risk, on the other hand, was predicted based on the consumption of a single portion of contaminated fish tissue. More of the population would likely be exposed to a single dose of contaminated fish tissue than to a lifetime of consuming contaminated fish tissue or drinking water taken from the vicinity of certain mills. In addition, the single dose used to predict the noncancer effects was a relatively modest serving of 115 g (about 1/4 lb.) which is less than an enthusiastic person might eat at one sitting.

It should be noted that the fish tissue contaminant concentrations and subsequent risk estimates developed from the NBS data may not be representative of ambient conditions at a given mill. The NBS samples that were used for this evaluation were taken from sites close to pulp and paper mills using chlorine for bleaching. However, the sites may have been several miles from the mill, and not immediately downstream. In some cases the samples were taken several miles upstream of the mills. In addition, the NBS sample analyses were performed on composites composed of several fish of different sizes (within a given range) from which aliquots were prepared and analyzed and, therefore, would tend to "average" contaminant concentration values. In addition, finfish migrate in and out of an area; therefore, the fish sampled from the NBS were not likely to be exposed to a constant level of contamination throughout their lifetime, as was assumed for the simple dilution and EXAMS II assessments.

The prediction of human health risk presented in this assessment apply to hypothetically exposed individuals in the immediate vicinity of discharges only, not to the entire population. Predictions of the

population exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF in the environment using site specific effluent and receiving stream characteristics were beyond the scope and resources of this study. A consequence of not conducting a population assessment is the uncertainty concerning the extent of human exposure and total population risks associated with discharges of 2,3,7,8-TCDD and 2,3,7,8-TCDF.

6.6.2 Conclusions

Taking into account the above assumptions, simplifications, and limitations, the results of this assessment indicate the potential exists for high levels of 2,3,7,8-TCDD and 2,3,7,8-TCDF contamination in the water column resulting from surface water effluent discharges from many of the chlorine-bleaching pulp and paper mills investigated. These predicted contaminant concentrations could represent significant implications for human health. Each of the exposure assessment approaches used in this analysis predict upper bound risks that should be carefully considered by risk managers while assessing potential impacts associated with the discharge of 2,3,7,8-TCDD and 2,3,7,8-TCDF in chlorine-bleaching pulp and paper mill effluents.

6.7 References

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7. ASSESSMENT OF RISKS TO THE GENERAL POPULATION FROM EXPOSURE TO DIOXINS AND FURANS RESULTING FROM PULP/PAPER WASTEWATER SLUDGE INCINERATION

7.1 Introduction

This chapter provides estimated exposures and risks to humans associated with emissions of dioxins and furans that may result from incinerating pulp and paper mill wastewater sludges. The chapter is a condensed version of the following report prepared by EPA's Office of Air Quality Planning and Standards (OAQPS) as part of the Interagency Dioxin-in-Paper Workgroup:

> Dusetzina M. 1989. Human health exposure and risk assessment for dioxins-pulp/paper waste water sludge incineration-subtask 5. U.S. Environmental Protection Agency, Office of Air Quality Planning and Standards. October 1989.

Dusetzina (1989) used air dispersion modeling to predict potential inhalation exposures to dioxins and furans to populations surrounding each of the twenty-one pulp and paper mills that reported employing incineration as a wastewater sludge disposal method in the 104 Mill Study (USEPA 1988). Both maximum individual cancer risks and aggregate (or population) cancer risks were then estimated. Section 7.2 of this chapter summarizes the methodology used by Dusetzina (1989). Section 7.3 and Section 7.4 summarize the results and uncertainties, respectively, of the assessment.

7.2 Methodology

7.2.1 Unit Risk Estimate

The numerical constant that defines the exposure-risk relationship used by OAQPS in its analysis of carcinogens is called the unit risk estimate. The unit risk estimate is the lifetime cancer risk occurring in a hypothetical population in which all individuals are exposed throughout their lifetimes (about 70 years) to an average concentration of 1 $\mu g/m^3$ of the agent in the air which they breathe. An upper bound incremental unit cancer risk estimate of 3.3×10^{-5} pg/m^3 was developed by EPA for 2,3,7,8-TCDD using a multistage extrapolation model that is linear at low doses (USEPA 1984). It was also assumed by EPA that the cancer risk from inhalation of 2,3,7,8-TCDF is one-tenth that of 2,3,7,8-TCDD or 3.3×10^{-6} pg/m^3.

7.2.2 EPA Human Exposure Model (HEM) (Background)

The EPA HEM is a general model capable of producing quantitative expressions of general population exposure to ambient air concentrations of pollutants emitted from stationary sources (USEPA 1986). The HEM

contains (1) a sector-averaged atmospheric dispersion model, with meteorological data included, and (2) a population distribution estimate based on Bureau of Census 1980 Master Area Reference File (MARF) data. The input data needed to operate this model are source data (e.g., plant location, height of the emission release point, volumetric rate of release and temperature of the off-gases). Based on the source data, the model estimates the magnitude and distribution of ambient air concentrations of the pollutant in the vicinity of the source and enumerates the population around each source. The model is programmed to estimate these concentrations for a specific set of points within a radial distance of 50 kilometers from the source.

The HEM uses the estimated ground level concentrations of a pollutant together with population data to calculate general population exposure. For each of 160 receptors located around a plant, the concentration of the pollutant and the number of people estimated by the HEM to be exposed to that particular concentration are determined. The HEM multiplies these two numbers to produce exposure estimates and sums these products for each plant.

7.2.3 Pulp and Paper Mill Source Data

Twenty-one incinerators were included in the analysis. The locations and names of the mills that incinerate wastewater sludge, according to the 104-Mill Study, are listed in Table 7-1. Table 7-2 contains incinerator stack parameter values for these mills as reported to EPA by the American Paper Institute (Van Hook 1989).

According to Van Hook (1989) actual data on stack gas concentrations of dioxins were available for only one incinerator. Therefore, emission rates were derived for each incinerator by two different methods. The first method (i.e., the stack gas method) used data on the concentration of CDDs/CDFs in the stack gas from the incinerator for which API provided data (Van Hook 1989). Emission rates were calculated by multiplying each incinerator's operating hours by each incinerator's stack gas volume (see Table 7-3) and the 2,3,7,8-TCDD equivalent stack gas concentration obtained for the incinerator for which API provided measured data (see Table 7-4). Emission rates were developed for both dioxins and furans in units of TEQ.

The second method (i.e., the sludge concentration method) used wastewater sludge concentration data from the 104-Mill Study. Since information on the effectiveness of existing control devices in removing CDDs/CDFs was unavailable, it was assumed as a worst case that these devices were unable to remove CDDs/CDFs and that all 2,3,7,8-TCDD/TCDF present in pulp and paper mill wastewater sludge was emitted to the atmosphere. Table 7-5 presents the amount of wastewater sludge

Table 7-1. Locations of Pulp and Paper Mill Wastewater Sludge Incinerators

Company[a]	Location[a]	Latitude[b] (deg/min/sec)	Longitude[b] (deg/min/sec)
Potlach Corp.	Lewiston, ID	46/25/28	116/58/26
International Paper Co.	Texarkana, TX	33/18/59	94/04/47
Alabama River Pulp	Claiborne, AL	31/34/54	87/29/54
Scott Paper Co.	Hinckley, ME	44/41/56	69/38/51
Boise Cascade Corp.	Jackson, AL	31/29/37	87/53/56
Scott Paper Co.	Mobile, AL	30/44/09	88/02/59
International Paper Co.	Pine Bluff, AR	34/13/11	91/54/32
Potlach Corp.	Cloquet, MN	46/43/29	92/25/46
International Paper Co.	Moss Point, MS	30/25/32	88/30/56
International Paper Co.	Georgetown, SC	33/21/53	79/18/08
Champion Intn'l	Houston, TX	29/52/51	95/06/32
Chesapeake Corp.	West Point, VA	37/32/21	76/48/19
Longview Fibre Co.	Longview, WA	46/06/15	122/55/07
Simpson Paper Co.	Tacoma, WA	47/16/02	122/25/39
Ketchikan Pulp Co.	Ketchikan, AK	55/23/30	131/44/30
Alaska Pulp Corp.	Sitka, AK	57/02/50	135/13/35
Scott Paper Co.	Everett, WA	47/59/02	122/12/58
ITT-Rayonier, Inc.	Hoquiam, WA	46/58/03	123/51/45
ITT-Rayonier, Inc.	Port Angeles, WA	48/07/00	123/24/25
Scott Paper Co.	Westbrook, ME	43/41/04	70/21/08
Appleton Papers, Inc.	Roaring Springs, PA	40/20/12	78/24/21

[a] From 104-Mill Study (USEPA 1988) and Van Hook (1989).
[b] From USGS topographical maps. These locations should be fairly representative of the plant if not the incinerator.

Table 7-2. Pulp and Paper Mill Incinerator Stack Parameters[a]

Mill	Stack height (m)	Stack diameter (m)	Stack exit velocity (m/sec)	Stack gas temperature (Kelvin)
Lewiston	91.4	4.10	13.00	462
Texarkana	64.3	3.95	10.78	445
	66.8	3.63	15.00	338
Claiborne	69.5	3.51	13.55	425
Hinckley[b]	84.0	3.51	28.50	443
Jackson[b]	69.5	3.51	13.55	425
Mobile	88.7	5.60	15.70	454
Pine Bluff	76.2	3.58	8.40	333
Cloquet	76.2	2.44	15.20	449
Moss Point	43.0	2.44	22.90	452
Georgetown	85.3	5.20	14.20	464
Houston[b]	69.5	3.51	13.55	425
West Point	76.2	3.51	13.55	450
Longview	31.0	2.67	13.55	331
Tacoma[b]	69.5	3.51	5.00	425
Ketchikan[b]	69.5	3.51	13.55	425
Sitka[b]	69.5	3.51	13.55	425
Everett	54.0	4.00	13.55	450
Hoquiam	30.2	2.13	14.00	502
	51.5	2.43	12.30	336
Port Angeles	45.7	1.83	9.00	472
Westbrook	107.6	5.49	1.10	373
	109.6	3.20	23.80	466
Roaring Spring[b]	69.5	3.51	13.55	425

[a] From Van Hook (1989).

[b] Information not available - stack parameters assigned are an average calculated from data provided by Van Hook (1989).

Table 7-3. Dioxin TEQ and Furan TEQ Emission Rates Based on Reported Stack Gas Concentrations

Mill	Operating hours per year[a]	Stack gas volume[a] (acm/min)	Dioxin TEQ emission rate (kg x 10^{-8}/yr)	Furan TEQ emission rate (kg x 10^{-8}/yr)
Lewiston	8,400	10,328	19.	12.
Texarkana	6,915	4,673	7.0	4.4
	7,990	9,057	16.	9.9
Claiborne[b]	7,942	8,202	15.	9.5
Hinckley	8,500	13,800	2.5	16.
Jackson[b]	7,942	8,202	1.5	9.5
Mobile	8,496	23,442	42.	27.
Pine Bluff	8,430	4,953	9.1	5.8
Cloquet	8,640	3,541	6.5	4.2
Moss Point	8,400	6,860	12.	7.9
Georgetown	8,616	17,999	34.	21.
Houston[b]	7,942	8,202	15.	9.5
West Point	8,520	9,204	17.	11.
Longview	8,040	9,628	17.	11.
Tacoma[b]	7,942	8,202	15.	9.5
Ketchikan[b]	7,942	8,202	15.	9.5
Sitka[b]	7,942	8,202	15.	9.5
Everett	8,712	5,700	11.	6.8
Hoquiam	3,000	3,000	4.4	2.8
Port Angeles	7,345	4,248	6.7	4.3
Westbrook	8,400	1,536	2.7	1.7
	8,568	11,470	21.	13.
Roaring Springs[c]	7,942	8,202	15.	9.5

[a] From Van Hook (1989).

[b] Stack Parameter data not available - emission rates assigned are an average of rates calculated.

Table 7-4. Dioxin/Furan Emissions and Stack Flow Rate Date Used to Calculate Emission Rates[a] Based on Stack Gas Concentrations

Isomer	Isomer Concentration (ng/dsem) Run 1 (max)	Run 2	Run 3	Average	Toxic equiv. factor
2,3,7,8-TCDD	<0.021	<0.0054	<0.010	0.0121	1.0
1,2,3,7,8-PeCDD	<0.016	<0.0067	<0.015	0.0126	0.5
1,2,3,4,7,8-HxCDD	<0.035	<0.0085	<0.0095	0.0177	0.1
1,2,3,6,7,8-HxCDD	0.16	<0.010	<0.0075	0.0592	0.1
1,2,3,7,8,9-HxCDD	0.10	<0.016	<0.011	0.0423	0.1
1,2,3,4,6,7,8-HpCDD	0.71	0.488	<0.0024	0.4001	0.01
2,3,7,8-TCDF	0.043	<0.0050	<0.011	0.0197	0.1
1,2,3,7,8-PeCDF	<0.024	0.0063	<0.0086	0.0130	0.05
2,3,4,7,8-PeCDF	<0.039	0.0067	<0.0066	0.0174	0.05
1,2,3,4,7,8-HxCDF	0.065	0.015	<0.013	0.031	0.1
1,2,3,6,7,8-HxCDF	0.080	0.021	<0.012	0.0377	0.1
1,2,3,7,8,9-HxCDF	0.089	<0.0096	<0.016	0.0382	0.1
2,3,4,6,7,8-HxCDF	<0.024	<0.0076	<0.0090	0.0135	0.1
1,2,3,4,6,7,8-HpCDF	0.63	0.078	0.037	0.2483	0.01
1,2,3,4,7,8,9-HpCDF	0.22	0.026	0.0097	0.0852	0.01

Stack Flow Rate Data

Dry standard condition			Actual condition	
68,130	dscfm	=	124,940	acfm
1,927.4	dscfm	=	3,538.3	acmm

Toxic Equivalency Factor

Dioxins/furans stack gas concentrations

	Average (ng/acm)	Maximum (ng/acm)
dioxins	0.0187	0.0358
furans	0.0147	0.0228

[a] Research Triangle Institute (RTI 1989) used the API data (Van Hook 1989) to create this table.

Table 7-5. 2,3,7,8-TCDD and 2,3,7,8-TCDF Emission Rates Based on Site-Specific Concentrations in Sludge

Mill	Sludge burned[a] (t/yr)	2,3,7,8-TCDD conc.[a] (ppt)	2,3,7,8-TCDF conc.[a] (ppt)	2,3,7,8-TCDD emission rate ($kgx10^{-3}$/yr)	2,3,7,8-TCDF emission rate ($kgx10^{-3}$/yr)
Lewiston	11,680	78	639	0.827	6.777
Texarkana	47,046	86	1,000	3.674	42.718
Claiborne	9,379	81	313	0.690	3.311
Hinckley	27,025	36	128	0.883	3.141
Jackson	6,961	18	169	0.107	1.068
Mobile	19,173	9.5	18	0.165	0.313
Pine Bluff	37,237	185	2,940	7.255	99.404
Cloquet	10,907	5	25	0.050	0.247
Moss Point	2,333	161	1,020	0.341	2.161
Georgetown	16,736	62	161	0.942	2.447
Houston	33,095	106	144	3.185	4.327
West Point	17,663	14	77	0.225	1.235
Longview	5,475	35	375	0.174	1.864
Tacoma	12,045	34	98	0.372	1.072
Ketchikan	14,600	3.5	366[b]	0.046	4.852
Sitka	26,280	4.7	42	0.112	1.002
Everett	11,526	46[b]	72	0.481	0.754
Hoquiam	2,615	4.8	25	0.011	0.059
Port Angeles	17,800	47	65	0.760	1.051
Westbrook	19,749	13	55	0.233	0.986
Roaring Springs	11,700	5	113	0.053	1.201

[a] From 104-Mill Study (USEPA 1988) and Van Hook (1989).

[b] Information not available - emission rates assigned are an average of rates calculated.

incinerated at each pulp and paper mill, concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF in sludge and estimated emission rates for 2,3,7,8-TCDD and 2,3,7,8-TCDF. The emission rates presented in Table 7-5 were estimated by multiplying the amount of sludge burned at each mill by the concentration of 2,3,7,8-TCDD and 2,3,7,8-TCDF reported to be present in each mill's sludge. For those pulp and paper mills for which wastewater sludge concentratons were not available at the time of this assessment, the average emission rate for all other pulp and paper mills incinerating sludge was used. Although in this method it was assumed as a worst case that all 2,3,7,8-TCDD and 2,3,7,8-TCDF present in pulp and paper mill wastewater sludge was emitted to the atmosphere, the potential for secondary formation of CDDs/CDFs as products of incomplete combustion was not considered.

7.2.4 Risk Calculations

Aggregate risk was calculated by multiplying the total exposure produced by HEM (for a single source, a category of sources, or all categories of sources) by the unit risk estimate. The product was cancer incidences among the population exposed for 70 years. The total exposure, as calculated by HEM, is illustrated by the following equation:

$$\text{Total Exposure} = \sum_{i=1}^{N} (P_i C_i) \qquad (7\text{-}1)$$

where

$\sum$ = Summation over all grid points where exposure is calculated
P_i = Population associated with grid point i
C_i = Long-term average 2,3,7,8-TCDD/TCDF concentration at grid point i
N = number of grid points to 2.8 kilometers and number of block group/enumeration district (BG/ED) centroids within 2.8 and 50 kilometers of each source.

Individual risk, expressed as "maximum lifetime risk," was calculated by multiplying the highest concentration to which the general population is exposed, as reported by HEM, by the unit risk estimate. The product, a probability of getting cancer, applies to the number of people which HEM reported as being exposed to the highest listed concentration. The concept involved a simple proportioning from the 1 $\mu g/m^3$ on which the unit risk estimate was based to the highest listed concentration. In other words:

$$\frac{\text{Maximum Lifetime Risk}}{\text{Highest Concentration to which People are Exposed}} = \frac{\text{The Unit Risk Estimate}}{1\ \mu g/m^3}$$

7.3 Results

Tables 7-6 through 7-9 present the total number of people encompassed by the exposure analysis, the total exposure, and the highest concentration to which anyone is predicted to be exposed. This means that exposure results were not reported for each incinerator, but were reported by source category (all 21 incinerators in the analysis). The tables separately show the results for 2,3,7,8-TCDD or dioxin TEQs, and 2,3,7,8-TCDF or furan TEQs using the two methods of estimating emissions. Table 7-10 presents the combined estimated dioxin/furan risks for each incinerator.

7.4 Analytical Uncertainties

7.4.1 The Unit Risk Estimate

The uncertainties associated with evaluating the human hazard potential of 2,3,7,8-TCDD and 2,3,7,8-TCDF are discussed in Chapter 3 of this Integrated Assessment.

7.4.2 Emission Estimates

Literature reviews suggest that chlorinated dibenzo-p-dioxin/chlorinated dibenzofuran (CDD/CDF) formation and destruction are affected in the following ways:

- Chlorine must be available to produce CDDs/CDFs.
- Organic compounds capable of being transformed to rings must be present.
- Temperature must be sufficient to initiate and carry out the formation reaction to an appreciable degree within the residence time of the combustion and post-combustion process.
- Temperatures above a certain level will destroy CDDs/CDFs and their precursor compounds.
- CDD/CDF formation probably takes place on surfaces with active reaction sites (e.g., fly ash) rather than in the gas phase.
- Competing species, such as sulfur compounds, can occupy reaction sites and reduce CDD/CDF formation.
- Release of CDD/CDF molecules from reaction sites to the gas phase is temperature-dependent.

Table 7-6. Site-Specific Exposure Analysis Based on 2,3,7,8-TCDD Concentrations in Sludge (Maximum Radius = 50 km)

Annual average concentration ($ug/m^3 x 10^{-8}$)	Cumulative population exposed (persons)[a]	Cumulative exposure (persons ug/m^3)[b]
1.00	8	0.0000000857
0.5	521	0.00000343
0.25	5,820	0.0000195
0.1	102,000	0.000153
0.05	213,000	0.000235
0.025	556,000	0.000345
0.01	2,620,000	0.000654
0.005	3,790,000	0.000743
0.0025	5,670,000	0.000810
0.001	7,220,000	0.000836
0.0005	7,830,000	0.000840
0.00025	8,310,000	0.000842
0.0001	8,640,000	0.000843
0.00005	8,680,000	0.000843
0.000025	8,690,000	0.000843

[a] This column displays the computed value rounded to the nearest whole number of the cumulative number of people exposed to the matching and higher concentration values found in the first column.

[b] The last column displays the computed value of the cumulative exposure to the matching and higher concentration values found in the first column.

Table 7-7. Site-Specific Exposure Analysis Based on 2,3,7,8-TCDF Concentrations in Sludge (Maximum Radius = 50 km)

Annual average concentration ($ug/m^3 x 10^{-7}$)	Cumulative population exposed (persons)[a]	Cumulative exposure (persons ug/m^3)[b]
1.00	219	0.0000262
0.5	545	0.0000537
0.25	16,000	0.000537
0.1	122,000	0.00224
0.05	199,000	0.00280
0.025	293,000	0.00315
0.01	434,000	0.00337
0.005	723,000	0.00358
0.0025	1,470,000	0.00383
0.001	4,520,000	0.00431
0.0005	6,610,000	0.00446
0.00025	7,800,000	0.00450
0.0001	8,480,000	0.00452
0.00005	8,600,000	0.00452
0.000025	8,690,000	0.00452

[a] This column displays the computed value rounded to the nearest whole number of the cumulative number of people exposed to the matching and higher concentration values found in the first column.

[b] The last column displays the computed value of the cumulative exposure to the matching and higher concentration values found in the first column.

Table 7-8. Site-Specific Exposure Analysis Based on Dioxin Concentrations in Stack Gas (Expressed as dioxin TEQs) - Maximum Values (Maximum Radius = 50 km)

Annual average concentration ($ug/m^3 x10^{-12}$)	Cumulative population exposed (persons)[a]	Cumulative exposure (persons ug/m^3)[b] ($x10^{-8}$)
3.51	9	0.00320
2.50	60	0.0161
1.00	481	0.0783
0.5	11,900	0.831
0.25	43,000	1.91
0.1	109,000	2.90
0.05	323,000	4.36
0.025	896,000	6.40
0.01	3,250,000	9.90
0.005	6,690,000	12.4
0.0025	8,420,000	13.0
0.001	8,680,000	13.1
0.0005	8,690,000	13.1

[a] This column displays the computed value rounded to the nearest whole number of the cumulative number of people exposed to the matching and higher concentration values found in the first column.

[b] The last column displays the computed value of the cumulative exposure to the matching and higher concentration values found in the first column.

Table 7-9. Site-Specific Expsoure Analysis Based on Furan Concentrations in Stack Gas (expressed as dioxin TEQs) - Maximum Values (Maximum Radius = 50 km)

Annual average concentration ($ug/m^3 x 10^{-12}$)	Cumulative population exposed (persons)[a]	Cumulative exposure (persons ug/m^3)[b] ($x10^{-8}$)
2.27	9	0.00207
1.00	119	0.0174
0.5	2,900	0.182
0.25	22,200	0.827
0.1	65,000	1.54
0.05	169,000	2.22
0.025	525,000	3.44
0.01	1,690,000	5.18
0.005	4,350,000	7.05
0.0025	7,630,000	8.26
0.001	8,630,000	8.46
0.0005	8,690,000	8.47

[a] This column displays the computed value rounded to the nearest whole number of the cumulative number of people exposed to the matching and higher concentration values found in the first column.

[b] The last column displays the computed value of the cumulative exposure to the matching and higher concentration values found in the first column.

Table 7-10. Combined Dioxin/Furan Risks and Annual Incidence[a]

	Maximum individual risk		Annual incidence	
	Sludge conc. method ($\times 10^{-7}$)	Stack gas method ($\times 10^{-7}$)	Sludge conc. method	Stack gas method
Pine Bluff	9.3	0.000055	0.00015	<0.000001
Port Angeles	3.1	0.00026	0.000013	<0.000001
Longview	2.5	0.0012	0.000027	<0.000001
Texarkana	1.8	0.000042	0.000098	<0.000001
Everett	1.4	0.00029	0.000042	<0.000001
Lewiston	1.4	0.000053	0.0000097	<0.000001
Houston	1.2	0.000055	0.0002	<0.000001
Moss Point	0.95	0.000049	0.000012	<0.000001
Westbrook	0.16	0.000021	0.000014	<0.000001
Georgetown	0.15	0.000044	0.0000016	<0.000001
Ketchikan	0.15	0.000045	<0.000001	<0.000001
Roaring Springs	0.12	0.00011	0.0000015	<0.000001
Tacoma	0.11	0.000037	0.000034	<0.000001
West Point	0.056	0.000029	<0.000001	<0.000001
Hinckley	0.049	0.000011	0.0000027	<0.000001
Claiborne	0.049	0.000012	<0.000001	<0.000001
Jackson	0.039	0.000029	<0.000001	<0.000001
Cloquet	0.038	0.0000035	<0.000001	<0.000001
Mobile	0.025	0.000057	0.0000023	<0.000001
Hoquiam	0.021	0.000056	<0.000001	<0.000001
Sitka	0.012	0.0000091	<0.000001	<0.000001

[a] Risks presented were calculated using the EPA unit risk estimate for 2,3,7,8-TCDD and the TEQ method. This unit risk estimate was derived using the EPA carcinogenic potency estimate for 2,3,7,8-TCDD (1.56×10^{-4} $(pg/kg/d)^{-1}$). Had risks been calculated using FDA's potency estimate (1.75×10^{-5} $(pg/kg/d)^{-1}$), then the risks and incidences would be a factor of 8.9 lower than those presented in the table. Had risks been calculated using CPSC's potency estimate (6.7×10^{-5} $(pg/kg/d)^{-1}$), then the risks would be "at least" a factor of 2.3 lower than those presented in the table. The term "at least" is used because, as discussed in Section 3.3 of this report, CPSC does not place the same emphasis on risks calculated by the TEQ method as it does for 2,3,7,8-TCDD itself when estimating carcinogenic potency.

- Overall formation of CDDs/CDFs is probably controlled by the number of active sites available given a conductive time/ temperature profile.

- Gaseous and particulate matter control devices after combustion sources may or may not be effective in removing or preventing formation of CDDs/CDFs after the combustion zone but prior to stack entry. Wet processes (scrubbers and spray drier/baghouse combinations) are probably more effective.

The above information suggests that, given the chemical constituents, CDD/CDF formation is maximized in processes that supply particulate matter for reaction sites and occur at moderate temperatures, in short, inefficient combustion. The two major sources of inefficient combustion should be open fires and poorly designed or operated boilers, furnaces, and incinerators.

7.4.3 Sludge Burning at Municipal Incinerators

It is known that at least one pulp and paper mill sends primary sludge to a municipal incinerator for incineration. However, OAQPS has no information on dioxin/furan emissions from incineration of pulp and paper sludges off-site at municipal incinerators.

7.4.4 Exposure Assumptions

(1) <u>The public</u>. The following are relevant to the public as dealt with in this analysis:

- The basic assumptions implicit in the methodology are that all exposure occurs at people's residences, that people stay at the same location for 70 years (approximate human lifetime) to maintain consistency with URE (lifetime annual studies), that the ambient air concentrations and the emissions which cause these concentrations persist for 70 years (URE), and that the concentrations are the same inside and outside the residences. From this, it can be seen that public exposure is based on a hypothetical premise.

- The people dealt with in the analysis are not located by actual residences. As explained previously, people are grouped by census districts and these groups are located at single points called the population centroids. The effect is that the actual locations of residences with respect to the estimated ambient air concentrations are not known and that the relative locations used in the exposure model may have changed since the 1980 census.

(2) Ambient air modeling. The following are relevant to the estimated ambient air concentrations of dioxins/furans used in this analysis:

- Flat terrain was assumed in the dispersion model. Concentrations much higher than those estimated would result if emissions impact on elevated terrain near a plant.

- The estimated concentrations do not account for the additive impact of emissions from plants located close to one another. Several incinerators in Washington State are located such that their concentration distributions overlap, e.g., they are sited within 50 kilometers of each other. This will not increase exposure significantly because they are not sited extremely close together, and the additional contribution to those exposed to emissions from more than one incinerator would be extremely small.

- The increase in concentrations that could result from re-entrainment of dioxins/furans dust from, e.g., city streets, dirt roads, and vacant lots, is not considered.

- Meteorological data specific to plant sites are not used in the dispersion model. As explained, HEM uses the meteorological data from the STAR station nearest the plant site. Site-specific meteorological data could result in significantly different estimates, e.g., the estimated location of the highest concentrations.

- The dioxin/furan emission rates are estimates that are based on assumptions rather than on measured data.

7.4.5 Conclusions

Population risks from inhalation of dioxin/furan emissions from incineration of pulp and paper sludge were estimated by two methods. The estimated risks appear to be low for both. These two methods were used to assess risks for several reasons. Relevant, although limited, data were used in each. Although the stack gas concentration data were more limited (stack gas concentrations were available only at one stack) than the concentration of 2,3,7,8-TCDD/TCDF in sludge, it was probably more relevant since destruction efficiency, partitioning to fly ash and bottom ash, and distribution of CDDs/CDFs in gaseous and particulate phases did not have to be characterized. Uncertainties regarding these parameters are important for the sludge concentration method. The major problem associated with the stack gas method was, of course, the limited data. Questions concerning secondary formation and effectiveness of control devices, particularly if a significant fraction of the dioxins/furans are in a gaseous phase, are also important for the sludge concentration method.

OAQPS contends that the first assessment method (i.e., the stack gas method) provides a better estimate of the performance of the boilers used at the 21 pulp and paper mill facilities that incinerate sludge than the worst-case estimates from the second method which assumes no destruction of CDDs/CDFs in the sludge feed. The sludge charged to the power boiler for which stack gas concentration data are available uses a high sludge feed content (10 to 15 percent of feed) relative to the feed content used by the other facilities (2 to 17 percent) and therefore may represent an overestimation of typical emissions. The second method (i.e., the sludge concentration method), although more conservative since no destruction of CDDs/CDFs is assumed, does not account for secondary formation of CDDs/CDFs as products of incomplete combustion.

7.5 References

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8. ASSESSMENT OF CANCER RISK FROM EXPOSURE TO PCDDs AND PCDFs IN CONSUMER PRODUCTS

8.1 Introduction

Exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF in consumer products may potentially occur by means of dermal absorption (ADL 1987, NCASI 1987). That is, when paper products come into contact with the skin, dioxin in the paper may migrate to the skin surface where it may subsequently be absorbed. The risk of dioxin exposure may be greater when the product is contacted by liquid, as may occur with disposable infant diapers, because the liquid may enhance the transfer of dioxin to the skin. This chapter is a condensed version of the following report prepared by the Consumer Product Safety Commission (CPSC) as part of the Interagency Dioxin-in-Paper Workgroup:

> Babich MA. 1989. CPSC staff assessment of the risks to human health from exposure to chlorinated dioxins and dibenzofurans in paper products. Memorandum from Dr. Michael A. Babich (CPSC) to Lois Dicker (EPA/OTS). January 25, 1990.

The paper products considered in Babich (1989) were limited to products under CPSC jurisdiction. They were divided into product categories, depending on the assumed exposure mechanism. Exposure was assumed to occur by means of either liquid mediated exposure or dry contact (explained under Subsection 8.2). Products involving liquid-mediated absorption included: disposable infants diapers, paper towels, facial tissue, and toilet tissue. Paper towels were further divided into two scenarios, drying hands and household cleaning. Exposure by means of contact with dry paper included dinner napkins and communication paper (i.e., uncoated sheets), such as bond paper, books, magazines, and newsprint, except for coated sheets. Exposure to communications paper was assessed for exposures at home and in school.

8.2 Methodology

8.2.1 Exposure Assessment

(1) Assumptions common to all exposure scenarios. For the purpose of exposure assessment, dermal exposure was treated as a two-step process: (1) migration or extraction of dioxin from paper or pulp into a liquid contacting the skin or to the surface of the skin itself, followed by (2) percutaneous absorption. The migration step may occur by either of two general mechanisms, liquid-mediated extraction or skin contact with dry paper (unmediated diffusion).

(a) Percutaneous absorption. Estimates of the rate and extent of dermal absorption for use in exposure assessment were based on prelimi-

nary data with human skin in vitro (Weber et al. 1989). These experiments were done using both acetone and mineral oil as vehicles and using both intact and "damaged" skin.

Measures of dermal exposure included the rate of absorption and the extent of absorption at a given time. The exposure model used and the exposure data which were available determined which measure would be required. The rate of absorption was given as the transfer coefficient, defined by:

$$J = kA \qquad (8\text{-}1)$$

where J is the flux ($ug/cm^2/h$), k is the transfer coefficient (/h), and A is the specific dose (ug/cm^2) (Scheuplein and Ross 1974). The extent of absorption was expressed as the percent of the applied dose which is absorbed by 24 h post exposure. Parameters for dermal absorption were based on solvent deposition experiments, i.e., experiments where volatile solvents such as methanol or acetone were used as vehicles.

On average, 18.5 percent of 2,3,7,8-TCDD was absorbed by 17 h post exposure (estimated from Weber et al. 1989). This estimate is based on the assumption that 2,3,7,8-TCDD in both the dermis and epidermis is available for systemic absorption. With a transfer coefficient k of 0.012 h^{-1}, the fraction of 2,3,7,8-TCDD estimated to be absorbed at 24 h post exposure is ~0.25. For the purpose of risk assessment, it was assumed that 2,3,7,8-TCDD and 2,3,7,8-TCDF are absorbed at a rate of 0.012 h^{-1} and that 25 percent is absorbed in 24 hours.

(b) Concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF. The limited data on 2,3,7,8-TCDD and 2,3,7,8-TCDF levels in consumer products which are available are summarized in Table 8-1. Some of the data are for European products and, therefore, cannot be used to assess the risk to U.S. consumers. However 2,3,7,8-TCDD and 2,3,7,8-TCDF levels were measured in bleached pulp at all 104 bleached kraft and bleached sulfite process paper mills in the U.S. (USEPA 1988a) In the absence of additional data, dioxin levels in pulp were used as surrogates for product levels. Since information regarding the mill sources used in manufacturing specific products is, in general, unavailable, the average 2,3,7,8-TCDD and 2,3,7,8-TCDF levels over all the mills were used (8.6 ppt for 2,3,7,8-TCDD and 103.6 ppt for 2,3,7,8-TCDF). In calculating these averages, non-detects were averaged as one-half the detection limit.

(2) Disposable infant diapers. Disposable infant diapers consist of both core components and ancillary components (Mizutani 1987). Core components are: a top sheet of non-woven polypropylene which contacts the skin; fluff pulp fiber, which may be thicker in the middle; super-absorbent diapers contain absorbent gelling material (AGM), a crosslinked polyacrylate gel, mixed into the fiber; and a polyethylene back sheet.

Table 8-1. Concentrations (ppt) of 2,3,7,8-TCDD and 2,3,7,8-TCDF in Consumer Products

Product	2,3,7,8-TCDD	2,3,7,8-TCDF	Reference
Disposable diapers	ND (2.6)	8.8	NCASI (1987a); Blosser (1987)
	ND (2.1)	7.2	NCASI (1987a); Blosser (1987)
	ND	3.7	WJLA TV[a]
Paper towels	3.7	32	NCASI (1987)
Bond paper	13	290	NCASI (1987)
	13	240	NCASI (1987)
Facial tissue	1.1	13	Beck et al. (1988)
Scrap paper	0.6	13	Beck et al. (1988)
Newsprint	ND	ND	Beck et al. (1988)
Tissue	1.3	31.1	LeBel et al. (1989)

[a] Assays for WJLA TV were performed by Triangle Laboratories, Research Triangle Park, NC.

ND, nondetectable; the number in parentheses is the detection limit.

Ancillary components include the fasteners, elastic, and adhesive. AGM has a higher affinity for liquids than fluff pulp. The total capacity of a superabsorbent diaper is ~300 g, depending on its ability to distribute the moisture throughout the core. Absorbed liquid may also be released from both the pulp and AGM under pressure, such as occurs when the child sits.

(a) **Exposure and risk calculations.** Exposure was assumed to be a 2 step process. First, dioxins in the pulp (solid, or adsorbed phase) are extracted into urine (aqueous phase). Aqueous phase dioxins are then available for absorption by the skin. Dermal absorption is assumed to be the rate limiting step. Extraction of dioxins from the pulp is assumed to be a rapid process relative to dermal absorption and quickly approaches a steady state.

Infants were assumed to use an average of six diapers per day (including four daytime, one naptime, and one overnight diaper) for three years.

The average daily dose (ADD) and the lifetime average daily dose (LADD) (pg/kg-d) were calculated as follows:

$$ADD = \frac{(k)(C)(Fa)(Fb)(Fc)\ [4(Usd)(Td) + (Usn)(Tn) + (Uso)(To)]}{(B)(K)} \quad (8\text{-}2)$$

where

k	=	the rate of dermal absorption;
C	=	the initial dioxin concentration in the pulp;
Fa, Fb, and Fc	=	correction factors to account for such conditions as anatomical site, damaged skin, and age, respectively;
Usd, Usn, and Uso	=	the amounts of urine in skin contact for daytime, naptime, and overnight diapers, respectively;
Td, Tn, and To	=	the exposure durations for daytime, naptime, and overnight diapers, respectively;
B	=	the average body weight; and
K	=	the urine:pulp equilibrium partition coefficient.

$$LADD = \frac{3(k)(C)(Fa)(Fb)(Fc)\ [4(Usd)(Td) + (Usn)(Tn) + (Uso)(To)]}{70(B)(K)} \quad (8\text{-}3)$$

where

3	=	the years of exposure; and
70	=	the average life expectancy.

Because different partition coefficient values are used for 2,3,7,8-TCDD and 2,3,7,8-TCDF, ADD and LADD must be calculated separately for each. Then the total ADD and total LADD in terms of TEQs are calculated.

The dioxin TEQ concentration in diapers which would result in a lifetime risk of 1 x 10^{-6}, or the "risk specific dioxin concentration", Crs, was given by:

$$Crs = \frac{70(B)(K)(Rsd)}{3(k)(Fa)(Fb)(Fc)\ [4(Usd)(Td) + (Usn)(Tn) + (Uso)(To)]} \qquad (8\text{-}4)$$

where:

Rsd = the risk specific dioxin TEQ dose (i.e., the dose at which the lifetime cancer risk is 1 x 10^{-6}).
K = partition coefficient for 2,3,7,8-TCDD, in this case.

(b) Parameters. The values of the parameters from which exposure and risk are estimated are given in Table 8-2. The rationale for the values assigned to each parameter is presented in detail in Babich (1989). In general, the parameters are based on infants wearing medium size diapers, i.e., infants from 12 to 24 pounds. It is assumed that these represent the average or typical infant in the age range from birth to 3 years, the time when infants are assumed to be in diapers (ADL 1987; NCASI 1987).

Certain parameters such as urine load were specific for the time of day. The abbreviations for these parameters were distinguished by the use of the suffixes d, n, and o for day, nap, and overnight, respectively.

(3) Paper towels, facial tissue, and toilet tissue. Exposure is a two-step process: (1) liquid-mediated extraction of dioxin from the paper or pulp, followed by (2) dermal absorption. The extraction step (Step 1) was assumed to be rapid, i.e., it was assumed to reach equilibrium, even though exposures for these products were brief. However, this model differed from the diaper model in that there were two components to the dermal absorption step (Step 2) (NCASI 1987).

During Step 1, dioxin is extracted from the pulp by a given amount of liquid phase (L). In general, some portion of the liquid phase is in contact with the skin (Ls). During Step 2a, the amount of dioxin dissolved in Ls is available for dermal absorption, at rate k, for the length of time that the product and the liquid phase are in contact with the skin (i.e., the exposure duration). Up to this point, the exposure model is similar to that for diapers. During Step 2b, it was assumed that a portion of Ls remains on the skin for 24 h (Lsr) (NCASI 1987) and that a fraction, f, of the 2,3,7,8-TCDD and 2,3,7,8-TCDF dissolved in Lsr will be absorbed.

(a) Exposure and risk calculations. The average daily dose (ADD), lifetime average daily dose (LADD), and the risk specific dioxin TEQ concentration (Crs), were calculated as follows:

$$ADD = \frac{(C)(N)(F)}{(K)(B)}\ [(k)(Ls)(T) + (f)(Lsr)] \qquad (8\text{-}5)$$

Table 8-2. Parameters for Estimating Exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF in Disposable Infant Diapers

Parameter		Value	
C,	2,3,7,8-TCDD concentration (pg/g)	3[a]	
	2,3,7,8-TCDF concentration (pg/g)	25[a]	
	TEQ concentration (pg/g)	5.5	
k,	Dermal absorption rate (/h)	0.012	
K,	Pulp:urine partition coefficient		
	2,3,7,8-TCDD	14,300[b]	
	2,3,7,8-TCDF	6,300[b]	
B,	Average body weight (kg)	10	
Fa,	Anatomic site correction	2	
Fb,	Damaged skin correction	1.5	
Fc,	Infant skin correction	1.5	
Ud,	Urine load, day (n=4) (g)	90[c]	
Un,	Urine load, nap (n=1) (g)	105[c]	
Uo,	Urine load, overnight (n=1) (g)	160[c]	
Ed,	Wear time, day (h)	2.6[c]	
En,	Wear time, nap (h)	3.6[c]	
Eo,	Wear time, overnight (h)	10.0[c]	
Td,	Wet time, day (h)	0.8[c]	
Tn,	Wet time, nap (h)	1.4[c]	
To,	Wet time, overnight (h)	7.0[c]	
		Superabsorbent[d]	Conventional[d]
Usd,	Urine in skin contact, day (g)	0.20	0.36
Usn,	Urine in skin contact, nap (g)	0.28	0.42
Uso,	Urine in skin contact, overnight (g)	0.55	4.8

[a] Average values. Based on data provided in the 104 Mill Study (USEPA 1988a) and on data provided by diaper manufacturers directly to CPSC.

[b] Coefficients were determined at 32°C using synthetic urine and Southern softwood fluff pulp (NCASI 1989).

[c] O'Reilly (1989).

[d] Based on NCASI (1987) and O'Reilly (1989).

where

C = the concentration of dioxin in the pulp,
N = the number of units per day,
K = the pulp:liquid partition coefficient,
B = the average body weight,
k = the dermal absorption rate,
f = the fraction of 2,3,7,8-TCDD absorbed by the skin in 24 h,
F = a correction factor applied to k and f,
Ls = the amount of liquid phase in skin contact,
Lsr = the amount of Ls which remains on the skin for 24 h, and
T = the exposure duration.

$$LADD = \frac{(C)(N)(Y)(F)}{(70)(K)(B)}[(k)(Ls)(T) + (f)(Lsr)] \qquad (8\text{-}6)$$

where:

Y = the number of years that the product is used, and
70 = the average life expectancy.

$$Crs = \frac{(70)(Rsd)(K)(B)}{(N)(Y)(F)\ [(k)(Ls)(T) + (f)(Lsr)]} \qquad (8\text{-}7)$$

where:

Rsd = the risk specific dioxin TEQ dose (i.e., the dose at which the lifetime cancer risk is $1x10^{-6}$).

(b) **Parameters.** The parameters used in estimating exposure to dioxin in paper towels, facial tissue, and toilet tissue are given in Tables 8-3 through 8-5, respectively.

(4) Communications paper and paper napkins. The exposure model for these products differed from the models discussed above, because there is no liquid phase to promote the migration of dioxin from the pulp to the skin. Hence, migration (Step 1) occurs by passive diffusion. This was assumed to be the rate limiting step. The amount of dioxin which migrates depends on the exposure duration. A fraction (f) of the dioxin which is transferred is absorbed by the skin (Step 2).

(a) **Exposure and risk calculations.** The average daily dose (ADD), lifetime average daily dose (LADD), and risk specific dioxin TEQ concentration in the product (Crs), were calculated as follows:

$$ADD = \frac{(r)(f)(C)(P)(T)(As)(F)(N)}{(Ap)(B)} \qquad (8\text{-}8)$$

Table 8-3. Parameters for Estimating Exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF in Paper Towels

Parameter		Drying hands	Household cleaning
C,	2,3,7,8-TCDD concentration (pg/g)	8.6	8.6
	2,3,7,8-TCDF concentration (pg/g)	103.6	103.6
	TEQ concentration (pg/g)	19	19.0
N,	Number of units per day	5	5
T,	Contact duration (sec)	15[a,b]	60
	Liquid phase surrogate	water[a]	8% EtOH[a]
K,	Partition coefficient:		
	2,3,7,8-TCDD	≥13,000[a]	≥2,000[a]
	2,3,7,8-TCDF	29,000[a]	2,000[a]
L,	Mass of liquid phase (g)	15[a]	15[a]
Ls,	Liquid in skin contact	1.2[a]	1.2[a]
Lsr,	Liquid remaining on skin	0.6[d]	0.3[d]
k,	Dermal absorption rate (/h)	0.012	0.012
f,	Fraction dermal absorption at 24 h	0.25	0.25
F,	Correction factor for k and f	1	1
B,	Average body weight (kg)	70[a,c]	70[a,c]
Y,	Years used	70[a]	70[a]
Rsd,	Risk specific dose (pg/kg/d)	0.015	0.015
As,	Skin surface are exposed (cm^2)	800[c]	400

[a] NCASI (1987).
[b] ADL (1987).
[c] USEPA (1989).
[d] Assuming 0.1 g per 100 cm^2 of exposed skin surface.

Table 8-4. Parameters for Estimating Exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF in Facial Tissue

Parameter		Normal use	Make-up removal
C,	2,3,7,8-TCDD concentration (pg/g)	8.6	8.6
	2,3,7,8-TCDF concentration (pg/g)	103.6	103.6
	TEQ concentration (pg/g)	19	19.0
N,	Number of units per day	6[a]	1[a]
T,	Contact duration (sec)	20[a]	300[a]
	Liquid phase surrogate	saline[a]	8% EtOH[a]
K,	Partition coefficient:		
	2,3,7,8-TCDD	≥14,300[e]	≥2,000[a]
	2,3,7,8-TCDF	5,300[e]	2,000[a]
L,	Mass of liquid phase (g)	1[a]	1[a]
Ls,	Liquid in skin contact	0.5[a]	1[a]
Lsr,	Liquid remaining on skin	0.005[d]	0.3[d]
k,	Dermal absorption rate (/h)	0.012	0.012
f,	Fraction dermal absorption at 24 h	0.25	0.25
F,	Correction factor for k and f	2	2
B,	Average body weight (kg)	70[a,c]	65[c]
Y,	Years used	70[a]	50[a]
Rsd,	Risk specific dose (pg/kg/d)	0.015	0.015
As,	Skin surface are exposed (cm^2)	5	300[c]

[a] NCASI (1987).
[b] ADL (1987).
[c] USEPA (1989).
[d] Assuming 0.1 g per 100 cm^2 of exposed skin surface.
[e] NCASI (1989).

Table 8-5. Parameters for Estimating Exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF in Toilet Tissue

Parameter		Male	Female
C,	2,3,7,8-TCDD concentration (pg/g)	8.6	8.6
	2,3,7,8-TCDF concentration (pg/g)	103.6	103.6
	TEQ concentration (pg/g)	19.0	19.0
N,	Number of units per day	30[a]	60[a]
T,	Contact duration (sec)	10[a]	10[a]
	Liquid phase surrogate	urine[a]	urine[a]
K,	Partition coefficient:		
	2,3,7,8-TCDD	≥14,300[e]	≥14,300[e]
	2,3,7,8-TCDF	6,300[e]	6,300[e]
L,	Mass of liquid phase (g)	1[a]	1[a]
Ls,	Liquid in skin contact	1[a]	1[a]
Lsr,	Liquid remaining on skin	0.025[d]	0.05[d]
k,	Dermal absorption rate (/h)	0.012	0.012
f,	Fraction dermal absorption at 24 h	0.25	0.25
F,	Correction factor for k and f	2	2
B,	Average body weight (kg)	75[c]	65[c]
Y,	Years used	70[a]	70[a]
Rsd,	Risk specific dose (pg/kg/d)	0.015	0.015
As,	Skin surface are exposed (cm^2)	50	100

[a] NCASI (1987).
[b] ADL (1987).
[c] USEPA (1989).
[d] Assuming 0.1 g per 100 cm^2 of exposed skin surface.
[e] NCASI (1989).

where:

C = the concentration of dioxin in the pulp,
P = the mass of pulp in the product,
r = the rate of transfer of 2,3,7,8-TCDD from the product to skin,
f = the fraction of 2,3,7,8-TCDD absorbed by the skin in 24 h,
T = the exposure duration,
As = the skin surface area contacted by the product,
Ap = the surface area of the product (one side),
N = the number of units per day,
B = the average body weight, and
F = correction factor.

$$LADD = \frac{(r)(f)(C)(P)(T)(As)(F)(N)(Y)}{(70)(Ap)(B)} \qquad (8\text{-}9)$$

where:

Y = the number of years that the product is used, and
70 = the average life expectancy.

$$Crs = \frac{(70)(Rsd)(Ap)(B)}{(r)(f)(P)(T)(As)(F)(N)(Y)} \qquad (8\text{-}10)$$

where

Rsd = the risk specific dioxin TEQ dose.

(b) **Parameters.** The parameters used in estimating exposure to dioxin in communications paper and dinner napkins are summarized in Tables 8-6 and 8-7, respectively. The surface area contacted by dinner napkins was assumed to be equal to the area of one side of both hands.

8.2.2 Risk Assessment

(1) Individual cancer risk. The individual cancer risk, that is the average lifetime excess cancer risk (R), was calculated by:

$$R = (LADD)(Q)\left[\frac{\text{human absorption fraction}}{\text{animal absorption fraction}}\right] \qquad (8\text{-}11)$$

where LADD is the lifetime average daily dose (pg/kg/d), calculated from Equations 8-3, 8-6, or 8-9. The carcinogenic potency estimate, Q (in units of $(pg/kg/d)^{-1}$), for 2,3,7,8-TCDD is 6.7×10^{-5} (CPSC Babich 1988), 1.8×10^{-5} (FDA), and 1.6×10^{-4} (EPA). The "human absorption fraction" is assumed to be 1.0 for the dermal exposures assessed here. The "animal absorption fraction" is an estimate of the absorption which occurred during the animal experiments from which the carcinogenic potency

Table 8-6. Parameters for Estimating Exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF in Communications Paper

Parameter		Home	School
Product parameters			
C,	2,3,7,8-TCDD concentration (pg/g)	8.6	8.6
	2,3,7,8-TCDF concentration (pg/g)	103.6	103.6
	Toxic equivalents (pg/g)	19.0	19.0
	Mass of product (g)	4.5[a]	4.5[a]
	% Pulp	90[b]	90[b]
P,	Mass of pulp (g)	4.05	4.05
Ap,	Surface area (cm^2)	603[a,b]	603[a,b]
N,	Number of units/day	30[a]	60
Subject parameters			
T,	Contact duration (min)	2[a]	4
As,	Skin surface area contacted (cm^2)	75[a]	55
r,	Rate of TCDD transfer to skin (/h)	0.0005[d]	0.0005[d]
f,	Fraction dermal absorption	0.25	0.25
F,	Correction factor	1	1
Rsd,	Risk specific dose (pg/kg-d)	0.015	0.015
B,	Average body wt. (kg)	70[a,c]	40[b,c]
Y,	Years used	52	12

[a] NCASI (1987).
[b] ADL (1987).
[c] USEPA (1989).
[d] Data on migration of tris-(2,3-dibromopropyl)phosphate (a.k.a., TRIS) from dry cloth to rabbit skin were used as a surrogate for 2,3,7,8-TCDD (Ulsamer et al., 1978).

Table 8-7. Parameters for Estimating Exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF in Paper Napkins

Product parameters		
C,	2,3,7,8-TCDD concentration (pg/g)	8.6
	2,3,7,8-TCDF concentration (pg/g)	103.6
	Toxic equivalents (pg/g)	19.0
	Mass of product (g)	9.5[a]
	% Pulp	98[a]
P,	Mass of pulp (g)	9.3
Ap,	Surface area (cm^2)	1,865[a]
N,	Number of units/day	3[a]
Subject parameters		
T,	Contact duration (min)	2[a]
As,	Skin surface area contacted (cm^2)	300
r,	Rate of TCDD transfer to skin (/h)	0.0005[c]
f,	Fraction dermal absorption	0.25
F,	Correction factor	1
Rsd,	Risk specific dose (pg/kg-d)	0.015
B,	Average body wt. (kg)	70[a,b]
Y,	Years used	70[a]

[a] ADL (1987).
[b] USEPA (1989).
[c] Data on migration of tris-(2,3-dibromopropyl)phosphate (a.k.a., TRIS) from dry cloth to rabbit skin were used as a surrogate for 2,3,7,8-TCDD (Ulsamer et al., 1978).

estimates were derived. CPSC assumed an animal absorption fraction of 0.75 whereas EPA and FDA assumed 0.55 (Farland 1987b).

(2) Population cancer risk. The population risk, i.e., the number of cancers per year in the U.S. (N), was estimated by:

$$N = R \times P / 70 \qquad (8\text{-}12)$$

where:

R = the individual risk,
P = the exposed population, and
70 = the average lifespan.

(3) Non-cancer endpoints. The risk of non-cancer adverse effects is represented by the hazard index (HI):

$$HI = ADD/HA \qquad (8\text{-}13)$$

where ADD is the average daily dose, and HA is the estimated health advisory level developed by EPA, for the purpose of this assessment, for protection against liver toxicity (10 days at 10 pg/kg/d) (USEPA 1988b; Lee 1989). When the HI is less than or equal to 1, risk is assumed to be absent or, at most, trivial.

8.3 Results

8.3.1 Individual Cancer Risk

The exposures and individual cancer risks estimated to result from 2,3,7,8-TCDD and 2,3,7,8-TCDD in consumer paper products under CPSC jurisdiction are summarized in Table 8-8. Table 8-8 presents risk estimates for each product type using the carcinogenic potency estimates of CPSC, FDA, and EPA. As discussed in Section 3.3 of this report, CPSC does not place the same emphasis on risks calculated by the TEQ method as it does for 2,3,7,8-TCDD itself when estimating carcinogenic potency. Therefore, Table 8-8 presents CPSC risk estimates based on 2,3,7,8-TCDD alone.

Estimates of individual cancer risk (using CPSC's cancer potency estimate) range from 10-in-a-trillion for facial tissues (normal use scenario) to 2-in-a-billion for paper towels. For all products combined, the individual risk is estimated to be 5 per billion. Use of the EPA cancer potency factor and TEQ method results in slightly greater risks. Use of the FDA cancer potency factor and TEQ method results in slightly lower risks.

Table 8-8. Individual Lifetime and Population Cancer Risks from 2,3,7,8-TCDD and 2,3,7,8-TCDF in Consumer Paper Products

Product	2,3,7,8-TCDD LADD (pg/kg/d)	TEQ LADD (pg/kg/d)	Lifetime individual cancer risk[c]			Exposed population	Excess cancers per year		
			CPSC	EPA	FDA		CPSC	EPA	FDA
Superabsorbent Diapers	2.4×10^{-7}	6.8×10^{-7}	2.1×10^{-11} (100)	2.0×10^{-10} (35)	2.2×10^{-11} (35)	-	-	-	-
Conventional Diapers	1.7×10^{-6}	5.0×10^{-6}	1.5×10^{-10} (100)	1.4×10^{-9} (35)	1.6×10^{-10} (35)	1.0×10^{7}[d]	0.00002	0.0002	0.00002
Paper Towels (Hand Drying)	7.1×10^{-6}	1.1×10^{-5}	6.3×10^{-10} (100)	3.1×10^{-9} (64)	3.6×10^{-10} (64)	2.4×10^{8}	0.002	0.011	0.001
Paper Towels (Cleaning)	2.3×10^{-5}	5.1×10^{-5}	2.1×10^{-9} (100)	1.5×10^{-8} (45)	1.6×10^{-9} (45)	2.4×10^{8}	0.007	0.051	0.005
Facial Tissue (Normal Use)	1.3×10^{-7}	5.6×10^{-7}	1.2×10^{-11} (100)	1.6×10^{-10} (23)	1.8×10^{-11} (23)	1.2×10^{8}	0.00002	0.0003	0.00003
Facial Tissue (Makeup Removal)	7.2×10^{-6}	1.6×10^{-5}	6.4×10^{-10} (100)	4.6×10^{-9} (45)	5.2×10^{-10} (45)	1.2×10^{8}	0.001	0.008	0.0009
Toilet Tissue (Males)	3.0×10^{-6}	1.1×10^{-5}	2.7×10^{-10} (100)	3.1×10^{-9} (22)	3.6×10^{-10} (22)	1.2×10^{8}	0.0005	0.005	0.0006
Toilet Tissue (Females)	1.4×10^{-5}	5.2×10^{-5}	1.2×10^{-9} (100)	1.5×10^{-8} (22)	1.7×10^{-9} (22)	1.2×10^{8}	0.002	0.026	0.003
Communication Paper (Homes)	5.8×10^{-6}	1.3×10^{-5}	5.1×10^{-10} (100)	3.7×10^{-9} (45)	4.2×10^{-10} (45)	2.4×10^{8}	0.002	0.013	0.001
Communication Paper (School)	6.8×10^{-6}	1.5×10^{-5}	6.1×10^{-10} (100)	4.3×10^{-9} (45)	4.9×10^{-10} (45)	2.4×10^{8}	0.002	0.015	0.002
Paper Dinner Napkins	2.3×10^{-6}	5.1×10^{-6}	2.1×10^{-10} (100)	1.4×10^{-9} (45)	1.7×10^{-10} (45)	2.4×10^{8}	0.0007	0.005	0.0006

Table 8-8. (continued)

Product	2,3,7,8-TCDD LADD (pg/kg/d)	TEQ LADD (pg/kg/d)	Lifetime individual cancer risk[c] CPSC	EPA	FDA	Exposed population	Excess cancers per year CPSC	EPA	FDA
All Products (Male)[a]	5.0×10^{-5}	1.1×10^{-4}	4.5×10^{-9} (100)	3.2×10^{-8} (45)	3.5×10^{-9} (45)	1.2×10^{8}	0.008	0.055	0.006
All Products (Female)[b]	6.8×10^{-5}	1.6×10^{-4}	6.1×10^{-9} (100)	4.6×10^{-8} (45)	5.2×10^{-9} (45)	1.2×10^{8}	0.010	0.079	0.009

[a] Includes all products except superabsorbent diapers, facial tissues (makeup removal), and toilet tissue (females).

[b] Includes all products except superabsorbent diapers and toilet tissue (males).

[c] Calculated using Eqn. 9-12 and each Agency's cancer potency estimate. Numbers in parentheses are the percent of estimated risk due to 2,3,7,8-TCDD.

[d] Assumes use only of conventional diapers. Resident population age 0 to 3 years.

8.3.2 Population Cancer Risk

Excess cancer risks in the U.S. population are given in Table 8-8 in terms of excess cancers per year. Less than one cancer per year is expected to occur from 2,3,7,8-TCDD and 2,3,7,8-TCDF in consumer paper products. Additivity was assumed in combining risks from different products or different scenarios.

8.3.3 Non-Cancer Endpoints

The ADD values for all products are well below the estimated health advisory level for protection against liver toxicity (10 days at 10 pg/kg/d). Thus, the hazard index, is much less than one, indicating that the risk for non-cancer effects is absent or, at most, trivial. ADD values and hazard index values are given in Table 8-9 for all products.

8.4 Uncertainty Analysis

8.4.1 Liquid Mediated Extraction

K values were used to estimate the concentration of 2,3,7,8-TCDD/F in a liquid medium (Caq) in contact with the skin. This concentration may be regarded as an upper limit, since equilibrium conditions are not necessarily expected to occur during the course of exposure. For exposures less than 2 h, the extent of migration of 2,3,7,8-TCDD from paper may be less than that predicted by equilibrium partition coefficients, leading to an overestimate of exposure. Any error introduced by this assumption would be greater for products involving brief exposures (up to 5 minutes), such as paper towels, facial tissue, and toilet tissue than for diapers, where the exposure duration is greater (up to 10 h).

Even when exposures are of sufficient duration for equilibrium to occur, Caq may still be overestimated by assuming equilibrium conditions. The steady-state value of Caq depends in part on the rate at which aqueous phase TCDD is removed from solution by dermal absorption. If the rate of dermal absorption were similar to the extraction rate, the steady state value of Caq would be much smaller than the equilibrium value. However, it was assumed that dermal absorption is rate limiting. Therefore, assuming an equilibrium value for Caq will not lead to a substantial overestimate of exposure.

One of the assumptions of the disposable diaper exposure assessment is that aqueous phase 2,3,7,8-TCDD is in equilibrium with 2,3,7,8-TCDD bound to the pulp. In other words, as aqueous phase 2,3,7,8-TCDD is removed by dermal absorption, it may be replenished by reextraction of the pulp. Although the extraction process is assumed to be continuous, periodic replenishment (as may occur when the child sits, for example) would result in similar exposures.

Table 8-9. Risks of Non-Cancer Adverse Effects from 2,3,7,8-TCDD and 2,3,7,8-TCDF in Consumer Paper Products

Product	2,3,7,8-TCDD ADD[a] (pg/kg/d)	TEQ ADD[a] (pg/kg/d)	2,3,7,8-TCDD Hazard Index[b]	TEQ Hazard Index[b]
Superabsorbent Diapers	5.3×10^{-6}	1.5×10^{-5}	5.3×10^{-7}	1.5×10^{-6}
Conventional Diapers	4.0×10^{-5}	1.2×10^{-4}	4.0×10^{-6}	1.2×10^{-5}
Paper Towels (Hand Drying)	7.1×10^{-6}	1.1×10^{-5}	7.1×10^{-7}	1.1×10^{-6}
Paper Towels (Cleaning)	2.3×10^{-5}	5.1×10^{-5}	2.3×10^{-6}	5.1×10^{-6}
Facial Tissue (Normal Use)	1.3×10^{-7}	5.6×10^{-7}	1.3×10^{-8}	5.6×10^{-8}
Facial Tissue (Makeup Removal)	1.0×10^{-5}	2.2×10^{-5}	1.0×10^{-6}	2.2×10^{-6}
Toilet Tissue (Males)	3.0×10^{-6}	1.1×10^{-5}	3.0×10^{-7}	1.1×10^{-6}
Toilet Tissue (Females)	1.4×10^{-5}	5.2×10^{-5}	1.4×10^{-6}	5.2×10^{-6}
Communication Paper (Home)	7.7×10^{-6}	1.7×10^{-5}	7.7×10^{-7}	1.7×10^{-6}
Communication Paper (School)	4.0×10^{-5}	8.8×10^{-5}	4.0×10^{-6}	8.8×10^{-6}
Paper Dinner Napkins	2.3×10^{-6}	5.1×10^{-6}	2.3×10^{-7}	5.1×10^{-7}

[a] ADD is the average daily dose.
[b] The hazard index is the ratio of ADD to the EPA 10-day health advisory of 10 pg/kg/d.

8.4.2 Unmediated Migration

Like 2,3,7,8-TCDD, TRIS is a nonvolatile, hydrophobic compound. Thus, it does not appear unreasonable to employ TRIS as a surrogate for 2,3,7,8-TCDD. Of course, the appropriateness of polyester cloth as a surrogate for paper is of concern. The basis weight of the cloth (~0.01 g cm^{-2}) is roughly equivalent to that of bond paper. However, the extent to which the two matrices act as barriers to diffusion may be different. In addition, the mechanism by which 2,3,7,8-TCDD is adsorbed to paper, as well as the strength of the interaction, may differ from the interaction between TRIS and cloth. Thus, it is impossible to be certain whether the TRIS cloth model adequately represents 2,3,7,8-TCDD exposure.

8.4.3 Use of *in vitro* Percutaneous Absorption Data

Data obtained from human studies are preferred for use in assessing human exposure and risk. In addition, the available dermal penetration data for 2,3,7,8-TCDD with human skin (Weber et al., 1989) have the advantage that the time course of 2,3,7,8-TCDD absorption was determined over a time range (0.5 to 17 h) which is particularly relevant to human exposure scenarious. The primary disadvantage of the human data is that they were obtained *in vitro*. However, *in vitro* percutaneous absorption studies have been shown to correlate well with *in vivo* studies, including studies with hydrophobic compounds (Bronaugh & Franz, 1986; Bronaugh & Stewart, 1986). The primary disadvantage of the *in vivo* studies with rats is that dermal absorption was generally determined at one time point only. In addition, the applicability of rat data to human exposure assessment is uncertain, because human skin is generally considered to be less permeable than rat skin (based on studies with various penetrants, but not 2,3,7,8-TCDD; Bartek et al., 1972; Wester & Maibach, 1983a,b). In consideration of the advantages and disadvantages of both kinds of studies, the human data were used to assess human exposure.

8.4.4 Factors Affecting Percutaneous Absorption

Numerous factors have been reported to affect either the rate or extent of percutaneous absorption (Wester and Maibach 1983a,b), including: anatomic site, occlusion and hydration, the vehicle, diseased or damaged skin, drug concentration and skin surface area, multiple or chronic dosing, and age. Examples of each factor and a discussion of the extent to which each may influence the rate of 2,3,7,8-TCDD absorption are discussed below.

(a) Anatomic site. The rate and extent of percutaneous absorption has been shown to depend on the anatomical site to which the penetrant is applied (Feldman and Maibach 1967; Maibach et al. 1971). Percutaneous absorption rates are highest on the skin of the genitals (Britz et al. 1980; Hurley 1985). The greatly enhanced permeability of

the genitals should be considered in assessing exposure to 2,3,7,8-TCDD in disposal diapers. However, the genitals are not the only portion of the infants' skin which is exposed to urine. Therefore, a correction factor for anatomic site (Fa) of 2 was used in this assessment for diapers and toilet tissue.

(b) Diseased and damaged skin (Fb). Diseased or damaged skin is generally more permeable than normal healthy skin (Wester and Maibach 1983a,b). Thus, it is likely that diaper dermatitis will increase the permeability of infant skin toward 2,3,7,8-TCDD. If we assume that diaper dermatitic skin is 5-fold (i.e., 500 percent) more permeable to 2,3,7,8-TCDD than healthy skin, that the incidence of dermatitis is 20 percent (Jordan et al. 1986), and that 50 percent of the urine exposed skin is dermatitic, then the permeability of diapered skin will average 50 percent or 1.5-fold greater than that of undiapered skin. Thus, a correction factor for diaper dermatitis (Fb) of 1.5 was assumed in this assessment.

(c) Correction factor for age (Fc). The skin of preterm infants is more permeable than that of full term infants during the first three weeks after birth (Wilson and Maibach 1982; Rutter 1987). It is unclear, however, whether the skin of full term infants differs from that of adults, as the data are contradictory. The degree of barrier function in infant skin may be specific for certain compounds (McCormack et al. 1982) or for certain classes of compounds (Wilson and Maibach 1982). However, it is quite possible that the rate of absorption of 2,3,7,8-TCDD could be greater in infant skin than in adult skin. The difference in absorption rate between infant and adult skin is likely to be no more than 10-fold. For the purpose of this assessment, it was assumed that infant skin is 1.5-fold more permeable to 2,3,7,8-TCDD than adult skin, i.e., Fc = 1.5.

8.5 References

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9. ASSESSMENT OF RISKS TO THE GENERAL POPULATION EXPOSURE TO DIOXINS AND FURANS RESULTING FROM THE USE OF PULP-CONTAINING MEDICAL DEVICES

9.1 Introduction

This chapter provides estimated exposures and risks associated with the use of medical devices containing bleached wood pulp. This chapter is, in large part, a condensed version of the following report prepared by EPA as part of the Interagency Dioxin-in-Paper Workgroup:

USEPA. 1989. U.S. Environmental Protection Agency. Assessment of exposures and risks to the general population from the use of pulp-containing medical devices. Draft Report. Washington, D.C.: U.S. Environmental Protection Agency, Office of Toxic Substances. Contract No. 68-02-4254.

The scope of this assessment is limited to "patients" (nonoccupational); occupational exposures and risks were not estimated. This assessment has been developed in cooperation with the Food and Drug Administration (FDA) Center for Devices and Radiological Health (CRDH). The Center identified those medical devices that contain bleached wood pulp, and provided numerous parameters/assumptions concerning product use and wood pulp content, and general guidance on how each product is used.

Table 9-1 provides a list of those medical devices believed to contain bleached wood pulp and the corresponding use for each device. Note, however, that FDA is not certain whether other medical devices may contain bleached wood pulp because manufacturers are not required to provide FDA information regarding the ultimate source of the raw materials. Therefore, the products listed in this table are FDA's best estimate of what products contain bleached wood pulp. These devices are used at medical facilities, and several may be purchased over the counter for home use.

This section is organized into three parts. Section 9.1 contains exposure and risk estimates for each device listed in Table 9-1., along with input parameters used to derive these estimates. A discussion on uncertainties is presented in Section 9.2, and Section 9.3 presents conclusions.

9.2 Estimates of Exposures and Risks from Dermal Contact with Pulp-Containing Medical Devices

9.2.1 Exposure Parameters

The exposure parameters used to estimate exposure and risks from dermal contact with pulp-containing medical devices are listed in Table 9-2.

Table 9-1. Medical Devices for Which Exposures and Risks Were Estimated and Their Corresponding Uses

Medical device	Use
Unscented menstrual pad	To absorb menstrual discharge
Scented menstrual pad	To absorb menstrual discharge
Unscented menstrual tampon	To absorb menstrual discharge
Scented menstrual tampon	To absorb menstrual discharge
Alcohol pads	To apply alcohol or other disinfectants to the surface of the skin
Skin preparation for dressing wounds	To clean cuts or wounds before applying a permanent bandage
Absorbable hemostatic agents	A small sponge used during surgery
Wound dressings containing carboxymethyl cellulose	To cover cuts or wounds
Surgical apparel	Worn by surgeons, nurses, and patients during surgery (e.g., hoods, caps, masks, gowns, foot coverings, drapes)
Adult diapers	To absorb urine or feces uncontrollably released by adults
Medical disposal bedding	To cover mattresses
Medical absorbent fiber	Cotton-like pads used to apply medication or to absorb small amounts of fluid from a patient's body surface
Absorbent tipped applicator	To apply medications or remove specimens from a patient
Examination gown	Worn by patients during examinations
Ophthalmic sponges	Small sponges used to absorb fluids during eye surgery
Hydroxpropymethyl cellulose	To replace fluids in the eye lost during surgery
Cottonoid paddie	To absorb body fluids (i.e., a cotton ball)
Electroconductive media	Conductive creams or gels used to reduce the impedance to the electrode from the surface of the skin
Cutaneous electrode	An electrode applied directly to the skin to either record physiological signals or apply stimulation
Anesthetic conduction filter	A microporous filter used to remove particulates from anesthesia or other gases
Breathing circuit bacteria filter	To filter microbiological and particulate matter from a breathing circuit (which administers medical gases to a patient)
Heat and moisture condensers	To preserve the purity and physical state of gases used in a respirator or as an anesthesia
Isolation gowns	Worn to isolate patients at a hospital

Table 9-2. Exposure/Risk Parameters for Medical Devices

Device name[a]	Contact type[a]	Device mass[a] (gm)	Pulp in product[a] (%)	Pulp mass in product[a] (gm)	Exposure duration[a] (days/ lifetime)	Volume of liquid on skin/ total volume[b] (%)	Wetting factor[b] (%)	Absorbtion rate through skin[c] (%)	Partition coefficient[c] TCDD	Partition coefficient[c] TCDF
Unscented Menstrual Pad	Skin	10	90	9	2,400	25	10	25	14,300	5,300
Scented Menstrual Pad	Skin	10	90	9	2,400	25	10	25	14,300	5,300
Unscented Menstrual Tampon	Intact Nat. Channel	3-5	90	3.6	2,400	100	100	100	14,300	5,300
Scented Mentrual Tampon	Intact Nat. Channel	3-5	90	3.6	2,400	100	100	100	14,300	5,300
Alcohol Pads	Skin	0.5-1	100	0.75	6	100	100	25	2,000	2,000
Skin Prep. Wipe for Dressing Wounds	External, Short Term	2			NA	50	10	25	14,300	5,300
Absorbable Hemostatic Agents (e.g., SurgicelR, Oxycel)	Internal, Short Term	3-5	100	4	NA	100	100	100	14,300	5,300
Wound Dressings Containing Carboxymethyl Cellulose	Compromised Tissue	4	100		NA	50	50	100	14,300	5,300
Surgical Apparel: Hood, Cap, Masks, Gowns, Foot Cov., Drapes	External	150 (GWNS) 7-10 (MSKS)	100	150 8.5	0.17	NA	NA	0.30	NA	NA
Adult Diapers	Skin	113.5	90	102.2	730	0.017	10	25	14,300	6,300
Medical Disposable Bedding	Skin	113.5	100	113.5	1	NA	NA	0.30	NA	NA
Medical Absorbent Fiber	Skin	<0.5	100	0.5	17.7	50	100	25	14,300	5,300
Absorbent Tipped Applicator	Skin	0.25	50	0.12	17.7	100	100	25	2,000	2,000
Examination Gown	Skin	113.5	100	113.5	0.6	NA	NA	0.30	NA	NA
Ophthalmic Sponges	Surgical Aids	0.5	100	0.5	0.08	100	100	100	14,300	5,300
Hydroxypropymethyl Cellulose	Intraocular Surg Aid	<1 ml	100	1	0.08	100	100	100	14,300	5,300
Cottonoid Paddie	Compromised Tissue	2	<1	0.002	0.5	100	100	100	2,000	2,000
Electro Conductive Media	Skin Surface(Intact)	1-5	<1	0.003	2	100	100	25	2,000	2,000
Cutaneous Electrode	Skin Surface(Intact)	1-5	<1	0.003	2	100	100	25	2,000	2,000
Anesthetic Conduction Filter	No Direct Contact	2-3	100	2.5						
Breathing Circuit Bacteria Filter	No Direct Contact	2-3	100	2.5						
Heat & Moisture Condensers	No Direct Contact	2-3	100	2.5						
Isolation Gowns	External	150	100	150	0.17	NA	NA	0.30	NA	NA

NA - Not applicable

[a] Data obtained from FDA/CDRH (Stratmeyer (1989) or telephone conversations between Versar and FDA).

[b] Assumptions by Versar and FDA based on best available data and expected use patterns.

[c] Based on data obtained from Babich (1989) and Babich et al. (1989) (see Chapter 9 and Appendix A of this assessment).

In addition to the exposure parameters listed in Table 9-2, the industry average concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF found in pulp in the 104 Mill Study were used to estimate exposures and risks for all medical devices, except those made from rayon. This was necessary because, in most cases, concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF in pulp at individual mills could not be traced to specific medical devices. In calculating the average values, one-half the detection limit was substituted for nondetected values (see Table 9-3). However, the average concentrations were similar to average concentrations calculated without nondetected values.

For those medical devices made from rayon, the identities of those mills that produce dissolving cellulose pulp used to make rayon were identified by the American Paper Institute. The locations of the sites and the concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF in pulp from those sites as found in the 104 Mill Study are presented in Table 9-4.

Of the devices listed in Table 9-2, the following subset belongs in the category of rayon-containing devices:

Unscented Menstrual Tampon
Scented Menstrual Tampon
Wound Dressings Containing Carboxymethyl Cellulose
Medical Absorbent Fiber
Hydroxpropymethyl Cellulose

The exposure parameters in Table 9-2 that require further explanation are detailed below.

(1) Exposure duration (days). Depending on the specific situation, alcohol pads are used rarely to daily. As a worst case assumption, it is assumed that each application of alcohol pads lasted 30 seconds and will be administered 365 days per year for 50 years.

The surgical apparel and isolation gowns are used only during surgery. It is assumed that surgery lasts 2 hours or 0.083 day and occurs twice over a 70-year lifetime. In addition, exposure to medical disposable bedding will occur for hours on a rare basis. It is assumed that exposure to medical disposable bedding would last 12 hours per visit and would occur twice over a 70-year lifetime. The examination gowns used by patients are worn occasionally for hours. It is assumed that the gowns are worn for 1 hour every 5 years over a 70-year lifetime.

Exposures to medical absorbent fiber and absorbent tipped applicators occur for seconds on an occasional to daily basis. As a worst-case assumption, it is assumed that these devices are used for 60 seconds at a rate of 365 days per year over 70 years.

Table 9-3. Average Concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF in Pulp Calculated Based on Results from the 104-Mill Data Base

	Highest conc. (pg/g)	Average conc. without nondet. (pg/g)	Average conc. with nondet. (pg/g)
2,3,7,8-TCDD	116	8.4	8.5
2,3,7,8-TCDF	2,620	84.4	84.4

Table 9-4. Concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF in Pulp at Pulp Mills that Produce Dissolving Cellulose

Company	Mill location	2,3,7,8-TCDD conc. (pg/g)	2,3,7,8-TCDF conc. (pg/g)
Alaska Pulp Corp.	Sitka, AK	0.7 (ND)	1.4
International Paper Co.	Natchez, MS	3.6	15.0
		2.2	3.0
ITT Rayonier, Inc.	Fernandina Beach, FL	0.2 (ND)	0.5 (ND)
	Jesup, GA	0.6 (ND)	0.8 (ND)
		0.3 (ND)	0.8
		0.7 (ND)	0.6
		0.7 (ND)	0.9
	Port Angeles, WA	0.6 (ND)	2.1
Ketchikan Pulp & Paper	Ketchikan, AK	0.3 (ND)	0.3 (ND)
Proctor & Gamble Co.	Mehoopany, PA	2.0	1.1
Weyerhaeuser Co.	Cosmopolis, WA	1.0 (ND)	6.3
		NQ	6.4
		0.3 (ND)	2.9
		0.3 (ND)	3.1
AVERAGE CONCENTRATION[a]		0.8	3.0

ND = Non-Detect.
NQ = Not Quantified.

[a] In calculating the average concentrations, ND values were assumed to be one-half the detection limit.

Both ophthalmic sponges and hydroxypropymethyl cellulose are used during eye surgery. Eye surgery lasts less than 1 hour and occurs once or twice per lifetime. Therefore, it is assumed that eye surgery will last 1 hour and that it occurs twice over a 70-year lifetime.

Cottonoid paddies are used several minutes to hours on an infrequent basis; therefore, it is assumed that the paddies are used once for a total of 12 hours over a 70-year lifetime. Electro conductive media and cutaneous electrodes are also used on an infrequent basis for durations of minutes to days. It is assumed that the exposure duration for these devices occur once for 2 days over a 70-year lifetime.

(2) Absorption rate. The rate of 2,3,7,8-TCDD transferred to the skin over a 24-hour period from surgical apparel, medical disposable bedding, and examination and isolation gowns is calculated as 0.0005/hr x 24 hr = 0.012. In addition, it was assumed that 25 percent of this amount will be absorbed. There is no partition coefficient since this is based on dry skin transfer. Therefore, assuming dry dermal contact, the amount transferred and absorbed over a 24 hour period is 0.012 x 0.25 = 0.003 (or 0.3%).

For those products in contact for long periods of time with internal body fluids or in contact with compromised tissue in a wetted state, 100 percent absorption was assumed.

(3) Partition coefficient. The partition coefficients used are those reported for paper pulp using ethanol, synthetic urine, or saline solution in Babich (1989a). The partition coefficient used for alcohol pads is based on the ethanol results. Ethanol closely approximates the rubbing alcohol solution actually used. The transfer medium for the use of absorbent tipped applicators, cottonoid paddies, electro conductive media, and cutaneous electrode is assumed to be analogous to the transfer medium assumed by Babich (1989b) for make-up removal using facial tissues (ethanol). This assumption provides a worst-case scenario for the partition coefficient.

For all other medical devices, with the exception of diapers, saline solution was assumed to be the most representative partitioning/transfer medium. For adult diapers, the results from the urine partitioning experiment were used.

The final general point about Table 9-2 is that when no actual data were available, reasonable or reasonable worst-case assumptions were used. For example, for the "volume of liquid on skin/total volume" and the "wetting factor," reasonable worst-case assumptions were used. For partition coefficients, the most reasonable case was selected; however, if no clear choice could be made, the worst-case option was used. The estimation of the exposure duration was also based on the most "reasonable"

assumptions. However, if accurate data were not available, reasonable worst-case assumptions were used.

9.2.2 Exposure/Risk Assessment for Medical Devices

Table 9-5 lists the exposure/risks associated with the use of the medical devices listed in Table 9-1. A few general points should be noted when reviewing this table. First, lifetime average daily dose (LADD) was estimated using three slightly different methods, depending on the way the product is used and the type of data available. The most common method was as follows:

$$LADD = \frac{(C)(PM)(ED)(V)(WF)(1/PC)(AR)}{\text{Body Weight (kg)} \times \text{Lifetime (70 years)} \times \text{365 Days/Year}} \qquad (9\text{-}1)$$

where:

C = Concentration (pg/g) of 2,3,7,8-TCDD or TCDF
PM = Pulp Mass (g/day)
ED = Exposure Duration (days/lifetime)
V = Volume of liquid on skin/total volume
WF = Wetting factor (unitless)
PC = Partition coefficient (unitless)
AR = Absorption Rate (%)

This method estimates the amount of 2,3,7,8-TCDD/TCDF available on the skin surface, the transfer rate of dioxin from the medical device to the surface of the skin (partition coefficient), and the absorption rate through the skin. For several products (skin preparation for dressing wounds, absorbable hemostatic agents, and wound dressing containing carboxymethyl cellulose), FDA provided the total mass of product an individual may reasonably be exposed to over a lifetime. Therefore, this altered the way that the amount of 2,3,7,8-TCDD/TCDF available on the skin surface was estimated. For these products, LADD was estimated as follows:

$$LADD = \frac{(C)(TM)(V)(WF)(1/PC)(AR)}{\text{Body Weight (kg)} \times \text{Lifetime (70 years)} \times \text{365 days/year}} \qquad (9\text{-}2)$$

where:

C = Concentration (pg/g)
TM = Total Mass Exposed
V = Volume of liquid on skin/total volume
WF = Wetting factor (unitless)
PC = Partition coefficient (unitless)
AR = Absorption Rate (%)

Table 9-5. Estimates of Risks to the General Population from the Use of Pulp-Containing Medical Devices

Device name	Lifetime average daily dose (LADD)[b] (pg/kg/day)		Lifetime individual cancer risk[c,d,e]						Potentially exposed population
	2,3,7,8-TCDD	TEQ	EPA	2,3,7,8-TCDD (%)	FDA	2,3,7,8-TCDD (%)	CPSC	2,3,7,8-TCDD (%)	
Unscented Menstrual Pad	4.49E-08	1.65E-07	4.68E-11	27	5.25E-12	27	4.01E-12	100	3.96E+07
Scented Menstrual Pad	4.49E-08	1.65E-07	4.68E-11	27	5.25E-12	27	4.01E-12	100	3.71E+07
Unscented Menstrual Tampon	2.70E-07	5.43E-07	1.54E-10	50	1.73E-11	50	2.41E-11	100	2.83E+07
Scented Menstrual Tampon	2.70E-07	5.43E-07	1.54E-10	50	1.73E-11	50	2.41E-11	100	5.20E+06
Alcohol Pad	2.67E-09	5.32E-09	1.51E-12	50	1.70E-13	50	2.39E-13	100	1.0E+06 - 1.0E+07
Skin Prep. Wipe for Dressing Wounds	2.08E-10	7.64E-10	2.17E-13	27	2.43E-14	27	1.86E-14	100	Millions
Absorbable Hemostatic Agent (e.g., Surgicel®, Oxycel)	1.66E-08	6.11E-08	1.73E-11	27	1.95E-12	27	1.48E-12	100	Millions
Wound Dressing Containing Carboxymethyl Cellulose	7.82E-12	1.57E-11	4.46E-15	50	5.01E-16	50	6.99E-16	100	Hundreds of Thousands
Surgical Apparel: Hood, Cap, Mask, Gown, Foot cov., Drape	3.64E-07	7.25E-07	2.06E-10	50	2.31E-11	50	3.25E-11	100	Millions (patients) Thousands (health care)
Adult Diaper	1.05E-10	3.43E-10	9.73E-14	31	1.09E-14	31	9.41E-15	100	1.0E+06 - 1.0E+07
Medical Disposable Bedding	1.62E-06	3.23E-06	9.15E-10	50	1.03E-10	50	1.45E-10	100	1.0E+06 - 1.0E+07
Medical Absorbent Fiber	3.46E-11	6.96E-11	1.97E-14	50	2.22E-15	50	3.09E-15	100	1.0E+06 - 1.0E+07
Absorbent-Tipped Applicator	1.26E-09	2.51E-09	7.13E-13	50	8.00E-14	50	1.13E-13	100	1.0E+06 - 1.0E+07
Examination Gown	9.71E-07	1.94E-07	5.49E-10	50	6.16E-11	50	8.67E-11	100	1.0E+06 - 1.0E+07
Ophthalmic Sponge	1.58E-11	5.15E-11	1.46E-14	31	1.64E-15	31	1.42E-15	100	
Hydroxypropymethyl Cellulose	2.50E-12	5.03E-12	1.43E-15	50	1.60E-16	50	2.24E-16	100	1.5 Million Cataract Oper./Year
Cottonoid Paddie	2.38E-12	4.74E-12	1.34E-15	50	1.51E-16	50	2.12E-16	100	Millions
Electro-Conductive Media	3.56E-12	7.10E-12	2.01E-15	50	2.26E-16	50	3.18E-16	100	Millions
Cutaneous Electrode	3.56E-12	7.10E-12	2.01E-15	50	2.26E-16	50	3.18E-16	100	Millions
Anesthetic Conduction Filter[a]									Millions
Breathing Circuit Bacteria Fltr.[a]									Millions
Heat & Moisture Condensers[a]									Millions
Isolation Gown	3.64E-07	7.25E-07	2.06E-10	50	2.31E-11	50	3.25E-11	100	Millions (patients) Thousands (health care)

Table 9-5. (continued)

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[a] There will be no direct contact for these products. The only potential exposure route is through inhalation of dioxin that leaves the filter or condenser and enters the indoor air. Exposure through this pathway is expected to be negligible because only a very small amount of dioxin will leave these products and enter the air, and of the amount that does enter indoor air, very little will actually enter the lungs and be absorbed.

[b] LADDs were calculated as follows:

$$\frac{\left(\text{Concentration } \left[\frac{\text{pg}}{\text{g}}\right] \times \text{Pulp Mass } \left[\frac{\text{g}}{\text{day}}\right] \times \text{Exposure Duration (Days)} \times \text{Volume of Liquid on Skin/Total Volume} \times \text{Wetting Factor (unitless)}\right. \left. \times \text{1/Partition Coefficient (unitless)} \times \text{Absorption Rate (\%)}\right)}{\text{Body Weight (Kg)} \times \text{Lifetime (70 years)} \times \text{365 days/year}}$$

There were two exceptions, however. The first exception was the method to estimate LADD for surgical apparel, medical disposable bedding, examination gowns, and isolation gowns, and this was explained in Equation 8-3. The other exception was for products where FDA already estimated the total mass of the product available for exposure (skin prep. wipe for dressing wounds, absorbable hemostatic agents, and wound dressings containing carboxymethyl cellulose). In this case, LADD was estimated as follows:

$$\frac{\text{(Concentration} \times \text{Total Mass Exposed} \times \text{Volume of Liquid on Skin/Total Volume} \times \text{Wetting Factor} \times \text{1/Partition Coefficient} \times \text{Absorption Rate)}}{\text{Body Weight} \times \text{70 years} \times \text{365 days/year}}$$

[c] The slope factors are as follows for 2,3,7,8-TCDD: EPA = 1.56×10^{-4} $(\text{pg/kg day})^{-1}$; FDA = 1.75×10^{-5} $(\text{pg/kg day})^{-1}$; CPSC = 6.7×10^{-5} $(\text{pg/kg/day})^{-1}$.

[d] The slope factors are as follows for 2,3,7,8-TCDF: EPA = 1.56×10^{-5} $(\text{pg/kg day})^{-1}$; FDA = 1.75×10^{-6} $(\text{pg/kg day})^{-1}$; CPSC = 0.

[e] For EPA and FDA cancer slope factors, risk was estimated as follows: Risk = potency factor $(\text{pg/kg-day})^{-1}$ x LADD (pg/kg-day)/0.55. However, for the CPSC cancer slope factor, risk was estimated as follows: Risk = potency factor $(\text{pg/kg-day})^{-1}$ x LADD (pg/kg-day) / 0.75. The divisor is changed to 0.75 (from 0.55) because a different bioassay was used. The total risk is the sum of the risks from TCDD and TCDF.

For four other devices (surgical apparel, medical disposable bedding, isolation gowns, and examination gowns), the rate of 2,3,7,8-TCDD/TCDF transferred to the skin and the absorption rate were combined. This transfer and absorption rate was used by Babich (1989), and it applies to products that will undergo dry contact with the skin surface. In these situations, LADD was estimated as follows:

$$LADD = \frac{(C)(PM)(ED)(TR)(AR)}{\text{Body Weight (kg)} \times \text{Lifetime (70 years)} \times \text{365 days/year}} \qquad (9\text{-}3)$$

where:

C = Concentration (pg/g) of 2,3,7,8-TCDD or TCDF
PM = Pulp Mass (g/day)
ED = Exposure Duration (days/lifetime)
TR = Transfer Rate (unitless)
AR = Absorption Rate (%)

As shown in Table 9-5, LADDs for 2,3,7,8-TCDD were found to range from 2.38×10^{-12} pg/kg-day for cottonoid paddies to 1.62×10^{-6} pg/kg-day for medical disposable bedding. LADDs for 2,3,7,8-TCDFs were found to range from 2.36×10^{-11} pg/kg-day for cottonoid patties to 1.61×10^{-5} pg/kg-day for medical disposable bedding. The other categories with the highest exposure levels are examination gowns, isolation gowns, surgical apparel, and tampons. Exposures for medical absorbent bedding, examination gowns, isolation gowns, and surgical apparel were estimated using the transfer and absorption rate of 0.3 percent used by Babich (1989) because they involve dry skin contact. This method may be yielding unrealistically high estimates since it is expected that, in reality, dry skin contact would yield a lower dose.

Estimated risks were found to vary from 2.22×10^{-16} to 9.15×10^{-10} using EPA slope factors. They were found to vary from 2.49×10^{-17} to 1.03×10^{-10} using FDA slope factors and 5.07×10^{-17} to 1.45×10^{-10} to using the CPSC factor. As discussed in Section 3.3. of this report, CPSC does not place the same emphasis on risks tabulated by the TEQ method as it does for 2,3,7,8-TCDD itself when estimating carcinogenic potency. Therefore, Table 9-5 presents CPSC risk estimates based on 2,3,7,8-TCDD alone.

9.3 Uncertainty Analysis

The goal of an analysis of uncertainties is to provide decision makers with the complete spectrum of information concerning the quality of an assessment, including the variability in the estimated exposures and risks, the inherent variability in the input parameters, data gaps, and the effect these gaps have on the accuracy or reasonableness of the expo-

sure and risk estimates developed. The general causes of uncertainty in an exposure/risk assessment are as follows:

- Measurement error
- Use of indirect empirical or generic data
- Variability
- Use of models to estimate exposure/risk
- Use of professional judgment/disagreement

For this assessment, uncertainties will occur from all of the above areas. All areas are important, with the possible exception of measurement errors. Measurement errors will occur (e.g., in determining the product mass), but compared to other errors, they will usually be insignificant. The remainder of this section discusses how the specifics of this assessment apply to the major areas of uncertainty.

Indirect or empirial data create uncertainties when the surrogate data used do not directly apply. The most important example is the partition coefficient. Most partition coefficients used were not estimated using the transfer medium in which the exposure will take place. It is anticipated that the partition coefficient can affect the results by over an order of magnitude, and this may be the single most important area of uncertainty.

Use of models to approximate the process of transfer and absorption of dioxin thru human skin introduces uncertainty into the assessment. Uncertainty may be further compounded by the selection of the input parameters because errors associated with these parameters may be propagated by the use of these models.

Variability and professional judgment are most important in terms of the input parameters used in the exposure and risk models. All parameters are affected to some degree by these two areas of uncertainty, with exposure duration likely to have the largest effect on the results. For some categories (e.g., menstrual products), exposure duration is known within reasonable limits. In most other categories, however, a wide range of possible exposure durations is expected, and thus a high level of uncertainty will occur. Professional judgment is also particularly important for "volume of liquid on skin/total volume" and "wetting factor," since in most cases, measured data were not available.

9.4 Conclusions

Based on the analysis presented in this chapter, risks from individual medical devices are very small. The most significant risk, medical disposable bedding, was found to be 9.15×10^{-10}. It is possible that risks to health care workers could be greater than other subpopulations because this population will have significantly higher exposure durations

and may be exposed to multiple medical devices. Unfortunately, this subpopulation could not be characterized with the existing data. If additional work is done on risks from dioxins and furans in medical devices, additional data should be gathered, and risks to health care workers should be characterized.

9.5 References

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Babich MA. 1989. CPSC staff assessment of the risks to human health from exposure to chlorinated dioxins and dibenzofurans in paper products. Memorandum from Dr. Michael A. Babich (U.S. Consumer Product Safety Commission) to Lois Dicker (U.S. Environmental Protection Agency, Office of Toxic Substances). January 25, 1990.

Stratmeyer ME. 1989. Letter from Dr. Mel E. Stratmeyer (U.S. Food and Drug Administration, Center for Devices and Radiological Health) to Greg Schweer (U.S. Environmental Protection Agency, Office of Toxic Substances). June 5, 1989.

10. ASSESSMENT OF RISKS TO THE GENERAL POPULATION FROM DIOXINS AND FURANS IN FOODS PACKAGED IN OR CONTACTING BLEACHED PAPER PRODUCTS

10.1 Introduction

This chapter is a condensed version of a report prepared by the Quantitative Risk Assessment Committee (QRAC) of the U.S. Food and Drug Administration (FDA) as part of the Interagency Dioxin-in-Paper Workgroup:

> USFDA. U.S. Food and Drug Administration. 1990. Carcinogenic risk assessment for dioxins and furans in foods contacting bleached paper products. Report of the Quantitative Risk Assessment Committee. April 20, 1990.

Trace amounts of dioxin, mostly 2,3,7,8-chlorine-substituted congeners, are formed during the bleaching step in the manufacture of paper pulp. Studies carried out by the paper industry and the EPA in 1987-1988 confirmed the presence of trace amounts of dioxins and furans in bleached pulp and in bleached paper used for food packaging, as well as for personal care and other products. In addition, a 1988 preliminary Canadian survey of milk packaged in polyethylene-coated bleached paper cartons (Ryan et al. 1988) indicated that dioxin transfer to milk could occur.

At the request of EPA and FDA, paper manufacturers and the American Paper Institute (API) initiated studies to determine the migration of dioxin from various paper packaging materials into food. The possibility that dioxin congeners might transfer to food from paper articles was verified in brewing experiments with coffee filters conducted at Wright State University (Tiernan 1987) and by the National Council of the Paper Industry for Air and Stream Improvement (NCASI 1988). Also, in the spring of 1989, FDA collected and analyzed samples of homogenized whole milk packaged in polyethylene- coated bleached paper cartons for dioxins and furans. Based on these data, FDA performed a quantitative risk assessment for cancer risk to consumers of milk contaminated with dioxins (USFDA 1989).

Because of the enormous size of the paper market, FDA requested that the paper industry provide information which could be used for assessing (1) which food-contact articles had the greatest potential for contaminating food with dioxin congeners and (2) which food-contact situations needed to be the subject of detailed migration studies.

Based on the results of a detailed survey by the American Paper Institute of the end use market of paper products in contact with food, FDA evaluated the need for additional migration tests based on the potential for human exposure to dioxin congeners from paper food-contact articles. The following criteria were used to decide which articles were of greatest concern:

- the concentration of the dioxin congeners in the pulp used to manufacture the food-contact article;
- the basis weight of the paper in the article;
- the presence or absence of a coating on the paper;
- the nature of the coating;
- the temperature of the food during contact with the paper article;
- the duration of food contact;
- the food weight-to-surface area ratio during contact with the paper;
- the nature of the food; and
- the amount of food consumed per day that might contact the paper article.

The paper industry has conducted a series of migration tests on the high priority paper food-contact articles identified by FDA. These studies were performed under conditions that were intended to simulate as closely as possible actual food-contact applications. The results of these studies have formed the basis of a series of research reports from NCASI. These reports also served as the primary basis for our estimates of dioxin migration and exposure from paper food packaging. This information was analyzed by the Food and Color Additives Review Section (FCARS) and the Division of Nutrition and communicated to the Quantitative Risk Assessment Committee (QRAC). Because migration levels to food are affected by the level of residual dioxin congeners in the packaging material, changes in bleaching techniques that are currently underway in the paper industry are expected to reduce the amount of dioxin migrating to food and therefore estimates of exposure to dioxin congeners.

10.2 Dioxin Concentrations in Bleached Wood Pulp and Paper Food-Contact Articles

Bleached wood pulp and paper food-contact articles have been analyzed using similar analytical methods. The high resolution GC/MS methods described in NCASI Technical Bulletin No. 551 (NCASI 1989a) ("NCASI Procedures for the Preparation and Isomer-Specific Analysis of Pulp and Paper and Industry Samples for 2,3,7,8-TCDD and 2,3,7,8-TCDF") have been used for the development of essentially all of the analytical data submitted to FDA as well as for most of the pulp data analyzed under the Industry/EPA Cooperative Study Agreement (the 104-Mill Study). The paper

Industry has conducted full congener analyses of bleached pulp that demonstrate that other chlorinated dioxins and furans may also be present in bleached pulp (NCASI 1989b); however, the two congeners covered by the above methodology account for 93 to 100 percent of the dioxin toxic equivalents (TEQ).

The results from the 104-Mill Study provide one view of the range of dioxin congener levels in bleached pulp and a perspective on the range of values that might be encountered in food packaging. A limited set of analyses of dioxin residues in "representative" paper articles, as shown in Table 10-1, performed as part of the various NCASI studies of dioxin migration from high priority food-contact articles, indicate that dioxin residues in paper articles contacting food are comparable to or slightly less than the average pulp levels found in the 104-Mill Study.

Recent information from the paper industry (API 1990) breaking out the results from the 104-Mill Study according to those mills producing pulp for specific applications was used by FCARS for the exposure assessment. This group of food-contact materials included coffee filters, microwave popcorn bags, ovenable board stock, paperplate stock, and cup stock. For the remaining articles (bakery cartons, ice cream cartons, tea bags, and margarine wrap), the average dioxin residue levels for the 104-Mill Study were assumed (i.e., 8.5 ppt 2,3,7,8-TCDD and 85 ppt 2,3,7,8-TCDF; 17 ppt dioxin TEQ). The assumption for this last group of typical "food-contact" articles is based on FCARS' observations that the dioxin levels in those "representative" articles used in the migration studies are reasonably close to the average levels of the 104-Mill Study.

In addition, the paper industry has recently submitted updated dioxin residue data for all manufacturers of milk carton stock, which is also used for the packaging of juice products and half and half. Recent analyses of bleached pulp and paperboard show consistently low dioxin residue levels for all milk carton manufacturers ($\leq$ 1 ppt 2,3,7,8-TCDD and $\leq$ 0.6-2.0 ppt dioxin TEQ). Because a previous FDA risk assessment (Carcinogenic Risk Assessment for Dioxins in Milk Based on the FDA 1989 Survey, memorandum dated August 2, 1989, QRAC to R.J. Scheuplein, Ph.D.) for milk packaged in paperboard cartons was based on the results of milk samples obtained from cartons manufactured prior to these substantial reductions in dioxin residue levels, revised estimates of dioxin exposure reflective of present carton manufacturing practices are presented in this assessment.

10.3 Food-Paper Migration Studies

10.3.1 Types of Articles Investigated

Essentially all of the information on migration of dioxin congeners from paper food-contact articles has been derived from studies conducted

Table 10-1. Representative Dioxin Congener Levels in Pulp and Paper Matrices

Product type	2,3,7,8-TCDD (ppt)	2,3,7,8-TCDF (ppt)
Bleached wood pulp	<1 - 116[a] 8.5[b]	<1 - 2620[a] 85[b]
Coffee filters	1 - 4	4 - 23
Coffee cups[c]	4 - 13	5 - 115
Cups - soup[c]	11 - 13	86 - 99
Trays dual ovenable PET[d] coated	0.5 - 3	1 - 4
Plates - uncoated and clay coated	5 - 10	11 - 90
Milk cartons	1 - 12	1 - 74
Half and half cartons	4	25
Juice cartons EVA[e] coated	5	44

[a]Range of values from the 104-Mill Study.
[b]Average value from the 104-Mill Study.
[c]Low density polyethylene - coated cups.
[d]Polyethylene terephthalate (uncoated base stock analyzed).
[e]Ethylene vinyl alcohol.

by NCASI. These studies were conducted with the intent of providing the FDA information on the extent of migration from high priority food-contact articles. Protocols for the migration studies were designed with FDA input with the intent of simulating as closely as possible actual use conditions, or in some instances, the worst use conditions that the article was likely to experience with food. NCASI has submitted the results of migration studies for the following food-contact articles: coffee filters, milk cartons, cream cartons, orange juice cartons, paper cups for hot beverages, paper cups for soups, paper plates for hot foods, and dual ovenable trays (NCASI 1988, 1989d-1989i, 1990a). Results of these studies and preliminary findings in a study of microwave popcorn (NCASI 1990b) have been evaluated by FCARS.

FCARS assumed that the food migration data developed for one type of paper article could serve as a surrogate for related food-contact articles. The results of some of the migration studies as well as certain assumptions have been used to estimate migration levels for those food-contact articles (cartons for bakery products, cartons for ice cream, tea bags, and wraps for butter and margarine) that were not subject to detailed migration studies. In other instances, FCARS assumed that potential dioxin congener transfer would either be low (e.g., from dry food packages such as flour bags) or the amount of food exposed would be low. In either case, FCARS considered that migration studies would be unnecessary because the expected contribution to dietary exposure to dioxin congeners would be insignificant relative to the exposures that are likely for the high priority food-contact articles.

10.3.2 Conclusions from the Migration Studies

All of the migration studies demonstrated detectable levels of transfer of the dioxin congeners to the test foods. Migration studies with milk in paperboard were able to demonstrate that the migration levels to milk increased during refrigerated storage as a linear function of the square root of the storage time (thus establishing that the transfer process to food of dioxin congeners followed familiar migration kinetics).

The temperature of foods, their composition, the nature of the paper article, as well as the dioxin congener levels in the paper articles were all found to influence the extent of transfer to test foods. In the case of coffee filters, the action of the hot liquid flowing through the filter during brewing was able to facilitate nearly quantitative extraction of 2,3,7,8-TCDF (and presumably 2,3,7,8-TCDD). When hot coffee was held in polyethylene-coated heavy cup stock, that is, not flowing through the paper, very little extraction of either congener was observed ($\leq$ 0.4 percent of the amounts in the cup). When the test food was changed to hot chicken soup held under similar conditions, the percent extraction from the heavy cup stock was found to increase to 6

percent for 2,3,7,8-TCDD and 10 percent for 2,3,7,8-TCDF. The highest extraction levels were found with clay-coated paper plates exposed to hot corned beef hash (13 percent 2,3,7,8-TCDD extraction and 18 percent 2,3,7,8-TCDF extraction). Dioxin congener levels in milk were found to be roughly proportional to the levels in cartons used to hold the milk.

The test articles used in the migration studies were selected by NCASI and FCARS as "representative" of the articles in use. FCARS observed that the dioxin congener levels in the test articles were somewhat different from the average levels reported in pulp from the 104-Mill Study for all mills production pulp used in each of the applications. The predicted dioxin TEQ levels in foods used in this assessment represent migration levels that were either determined during migration studies performed under "worst-case" scenarios that each paper article might experience with food or estimated from migration values developed for one type of paper article that was assumed to be a surrogate for another.

One of the complicating factors encountered in the migration studies was the presence of dioxin congeners in food matrices prior to their contact with paper articles. Although NCASI made no attempt to identify the environmental sources of these contaminants, they attempted to account for the background levels of dioxin congeners in food when evaluating the contribution of paper articles to human exposure to dioxin congeners. FCARS believes that such an adjustment is essential. Where adequate data were available, FCARS used such corrected figures for estimating exposure.

10.4 Food Intake Information

The estimation of exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF (i.e., exposure to dioxin TEQ) from consumption of foods contacting paper articles requires consideration of appropriate food intake information. Available nationally representative food consumption data bases, however, do not provide information on whether foods are sold, held, heated, cooked, or served in contact with different types of materials. Although production data on paper articles were provided by industry , use of these data to adjust food consumption intake to account for the many food-contact scenarios was not possible. Therefore, for estimating the total dioxin TEQ intake from all foods that may contact bleached paper, available food consumption data for average consumers of each of the foods were used in conjunction with the assumption that all such foods have indeed been in contact with bleached paper prior to being consumed. This necessary assumption leads to an overestimate of consumption of food that has been in contact with paper and, hence, to an overestimate of total dioxin TEQ exposure.

This upper-bound estimate of total dioxin TEQ exposure was adjusted through a comparison with a lower-bound estimate of dioxin TEQ exposure,

specifically, a per capita exposure, obtained using industry-provided production figures and estimates of the amount of food that might contact the paper. Additionally, exposure to dioxin TEQ on a food-by-food basis for individuals consuming mean and 90th percentile amounts of foods contacting each paper article was also calculated.

Because 100 percent of the U.S. population are "eaters" of at least one food contacting a bleached paper article, it was assumed that 100 percent of the U.S. population are "eaters" of dioxin. Therefore, estimates of food intake used to estimate total dioxin TEQ intake should be representative of the total population. We selected the Market Research Corporation of America's (MRCA) data base, five-year Menu Census 1982-87 (MRCA 1987) as our source of food intake data. This data base contains 14 consecutive days of data and is likely to capture a representative diet including both frequently and infrequently consumed foods.

10.5 Estimated Exposures

10.5.1 Estimated Total Dioxin TEQ Intake: All Foods Contacting Bleached Paper

Total-sample intakes for each food of interest in the MRCA data base were multiplied by the dioxin TEQ levels in the corresponding foods (see Table 10-2). The resulting dioxin TEQ values were summed to provide a total population dioxin TEQ intake of 12.7 pg per person per day. This can be considered to be an upper-bound estimate of mean dioxin TEQ intake because of the assumption that all food had been in contact with bleached paper prior to being consumed.

The availability of industry production figures (NCASI 1989c) allowed for the estimate of a lower-bound exposure to dioxin TEQ of 5.5 pg/person/day (see Table 10-3). Recognizing that a reaonable estimate should fall between the upper-and lower-bound estimates, we were able to derive a "best estimate" of mean total dioxin TEQ exposure of 9.1 pg/person/day. This value is the a mid-point between the upper- and lower-bound estimates.

The lower-bound estimate was obtained in two steps. First, the same dioxin TEQ food levels as used for the upper-bound estimate were combined with per capita food intakes derived from industry production figures and estimates of the amount of food that might contact each of the paper articles of interest. In three instances (i.e., bakery cartons, tea bags, and margarine wrap), where no data were provided, we used the total sample population MCRA intake for the appropriate food categories. The per capita dioxin TEQ values obtained for each food were added to obtain the total per capita dioxin TEQ disappearance (11 pg/person/day)

Table 10-2. Carcinogenic Risk for Consumers Resulting from Total Dioxin TEQ Intake from All Foods Contacting Bleached Paper ("mean consumer - total sample basis")

Food[a] (paper article)	Paper TEQ[b] levels (ppt)	TEQ levels in food (ppq)	Food intake per eating event (g)	Avg. daily food intake per person (grams/day)	Daily TEQ intake per person (pg/day)
1. Milk (cartons)	2	5	191	124	0.62
2. Coffee (filters)	8.8	3.2	332	136	0.44
3. Cream (cartons)	2	5	23.8	1.4	0.007
4. Juice (cartons)	2	15	190	36	0.54
5. Coffee (cups)	10.1	0.8	332	136	0.11
6. Soup (cups)	10.1	23	292	56	1.3
7. Meals-seasoned meat, vegs. (dual-oven trays)	10.6	35	215	61	2.1
8. Meals-seasoned meat (paper plates)	7.9	140	340[c]	37	5.2
9. Popcorn (microwave bags)	5.9	45	[d]	1.6	0.072
10. Donuts, sweet rolls (bakery cartons)	13.8	50	67.8	7.7	0.39
11. Frozen dairy desserts (ice cream cartons)	13.8	50	110	23	1.2
12. Tea (bags)	17	8	301	22	0.12
13. Margarine (wrap)	17	82	10.8	7.3	0.60
				Upper-bound	12.7
				Lower-bound[e]	5.5
				Best estimate[f]	9.1**

**The corresponding upper bound lifetime risks were estimated to be 2.4×10^{-6} based on FDA's slope factor, 2.1×10^{-5} based on EPA's slope factor, and 6.7×10^{-6} based on CPSC's slope factor.

[a]Food intake reported by Market Research Corporation of America (MRCA) obtained by multiplying the MRCA mean frequency of eating occasions (1982-87 5-year Menu Census) by the mean grams/eating occasions from USDA/NFCS, 1977-78 (Pao et al. 1982). Data are for the 2+ years age group, males and females, total sample population.

[b]104-mill average for producers (except updated for milk, cream and juice carton producers) (API 1990). The value for items 10-13 is the total 104-Mill Study average for bleached wood pulp.

[c]Food intakes based on NCASI production of 20 billion plates/yr and assumptions of 80 plates/person/year, 2 plates/per eating occasion, and 340 g food per eating occasion.

[d]NCASI per capita consumption estimate based on 1.4 billion bags sold in the U.S. in 1989. No survey data available for microwaveable popcorn.

[e]Estimate based on industry production figures for paper products.

[f]Midrange of lower and upper bound estimates.

Table 10-3. Industry-Based Per Capita Dioxin TEQ Exposures

Food (paper article)	Paper TEQ[a] levels (ppt)	TEQ levels in food (ppq)	Food intake (g/p/day)	TEQ intake (pg/p/day)
1. Milk (cartons)	2	5	100[b]	0.5
2. Coffee (filters)	8.8	3.2	176[c]	1.0
3. Cream (cartons)	2	5	-[l]	
4. Juice (cartons)	2	15	59[d]	0.9
5. Coffee (cups)	10.1	0.8	45[e]	0.04
6. Soup (cups)	10.1	23	45[f]	1.0
7. Meals-seasoned meat, vegs. (dual-oven trays)	10.6	35	5.9[g]	0.2
8. Paper plates	7.9	140	37[h]	5.2
9. Microwave popcorn	5.9	45	1.6[i]	0.07
10. Donuts, sweet rolls (bakery cartons)	13.8	50	7.7[j]	0.4
11. Frozen dairy desserts (ice cream cartons)	13.8	50	23[k]	1.1
12. Tea bags	17	8	22[j]	0.18
13. Margarine (wrap)	17	82	7.3[j]	0.60

[a] 104-mill average for producers (except updated for milk, cream and juice carton producers) (API 1990). The value for items 12 and 13 is the total 104-mill average for bleached wood pulp.

[b] NCASI estimate based on total production of milk in paperboard cartons.

[c] NCASI estimate based on coffee consumption data from the International Coffee Organization, fraction of the population that consumes coffee, and fraction of coffee that is brewed using filters.

[d] NCASI estimate based on total production of paperboard cartons used to package juice.

[e] NCASI based on estimate on all cup stock used only for coffee consumption.

[f] NCASI based its estimate on use of all cup stock for soup consumption only.

[g] NCASI used production figures for dual-ovenable trays to estimate per capita annual use of 6.4 trays. This was combined with the assumption of 340 g of food contacting each tray.

[h] NCASI estimated annual production of 20 billion plates or 80 plates/person/year. They assumed two plates are used to hold 340 g of food.

[i] NCASI estimate based on 1.4 billion bags (100 g/bag) sold in U.S. in 1989.

[j] No NCASI estimate available. Figure is from MRCA (see Table 10-2, footnote[a]).

[k] NCASI estimate based on production figures from the International Ice Cream Association.

[l] NCASI did not estimate. FCARS concluded that the per capita intake would be quite small, and would be subsumed within the figure provided for milk in paperboard cartons.

unadjusted for waste, inventory stock, and any other diversions from the consumer. Second, in order to adjust for such wastage and losses, we have divided the 11 pg/person/day by 2 to obtain the lower-bound of 5.5 pg/person/day.

This adjustment by a factor of 2 was used because comparisons of per capita estimates of intake (or "disappearance") for food components and nutrients with available survey data on food intake by individuals show that per capita estimates generally exceed survey intake estimates by 1.5 to 3-fold (Park and Yetley 1990, USDA 1984, USDHHS/USDA 1986, Glinsman et al. 1986). Most of the comparisons examined show approximately a 2-fold difference. Finally, as noted above, we selected a best estimate of dioxin TEQ exposure to fall midway between the upper- and lower-bound values.

10.5.2 Estimated Dioxin TEQ Intake: Food-by-Food Basis for Individual Paper Products

Although the above analysis is intended to provide a picture of overall dioxin TEQ exposure for the total U.S. population from consumption of foods that may have contacted bleached paper, the question arises about potential dioxin exposure to individuals who specifically have been identified as "eaters" of the various foods. The MCRA data base contains food intake information for the "eaters-only" population as well as for the total sample population. "Eaters-only" food intake data were combined with the dioxin TEQ levels in the corresponding foods to obtain potential dioxin TEQ intakes that would result from individuals consuming the foods at the mean and the 90th percentile levels of intake (see Table 10-4). Again, it was assumed that all foods consumed have indeed been in contact with bleached paper prior to being consumed. The other assumptions used for the total population exposure analysis also apply to the "eaters-only" analysis. Therefore, the dioxin TEQ intakes in Table 10-4 may also be considered as upper-bound estimates of mean and 90th percentile for "eaters" of each of the foods. The dioxin TEQ exposures in Table 10-4 must not be summed because the population of eaters is not the same for each food category.

10.6 Risk Assessment

10.6.1 Cancer Risks

Table 10-2 presents the results of the cancer risk assessment for the "mean consumer - total sample basis." The estimates of lifetime upper bound individual lifetime cancer risk range from 2.4×10^{-6} (using FDA's cancer potency slope factor) to 2.1×10^{-5} (using EPA's cancer potency slope factor).

Table 10-4. Upper Bound Carcinogenic Risk for Consumers of Foods Contacting Bleached Paper Contaminated with Dioxin ("eaters only - food-by-food basis")

Food[a,b] (paper article)	TEQ levels in food (ppq)	Food intake[c] (g/p/d) mean/90th %tile	Upperbound lifetime risk[f,g]					
			Mean intake			90th percentile intake		
			FDA	EPA	CPSC	FDA	EPA	CPSC
Milk (cartons)	5	170/408	2.2×10^{-7}	2.0×10^{-6}	6.2×10^{-7}	5.5×10^{-7}	4.9×10^{-6}	1.5×10^{-6}
Coffee (filters)	3.2	278/641	2.4×10^{-7}	2.1×10^{-6}	6.7×10^{-7}	5.3×10^{-7}	4.7×10^{-6}	1.5×10^{-6}
Cream (cartons)	5	7.3/18.7	1.0×10^{-8}	8.9×10^{-8}	2.8×10^{-8}	2.6×10^{-8}	2.3×10^{-7}	7.3×10^{-8}
Juice (cartons)	15	72/179	2.9×10^{-7}	2.6×10^{-6}	8.1×10^{-7}	6.0×10^{-7}	5.3×10^{-6}	1.7×10^{-6}
Coffee (cups)	0.8	278/641	5.9×10^{-8}	5.2×10^{-7}	1.6×10^{-7}	1.4×10^{-7}	1.2×10^{-6}	3.9×10^{-7}
Soup (cups)	23	74/148	4.5×10^{-7}	4.0×10^{-6}	1.3×10^{-6}	9.1×10^{-7}	8.1×10^{-6}	2.5×10^{-6}
Meals-seasoned meat, vegs. (dual-oven trays)	35	64/108	5.9×10^{-7}	5.2×10^{-6}	1.6×10^{-6}	9.9×10^{-7}	8.8×10^{-6}	2.8×10^{-6}
Metals-seasoned meat (paper plates)	140	37/74[d]	1.4×10^{-6}	1.2×10^{-5}	3.9×10^{-6}	2.7×10^{-6}	2.4×10^{-5}	7.5×10^{-6}
Popcorn (microwave bags)	45	16/32[e]	1.9×10^{-7}	1.7×10^{-6}	5.3×10^{-7}	3.7×10^{-7}	3.3×10^{-6}	1.0×10^{-6}
Donuts, sweet rolls (bakery cartons)	50	15/29	2.1×10^{-7}	1.9×10^{-6}	5.9×10^{-7}	4.0×10^{-7}	3.6×10^{-6}	1.1×10^{-6}
Frozen dairy desserts (ice cream cartons)	50	32/63	4.3×10^{-7}	3.8×10^{-6}	1.2×10^{-6}	8.5×10^{-7}	7.6×10^{-6}	2.4×10^{-6}

Table 10-4. (continued)

Food[a,b] (paper article)	TEQ levels in food (ppq)	Food intake[c] (g/p/d) mean/90th %tile	Upper bound lifetime risk[f,g]					
			Mean intake			90th percentile intake		
			FDA	EPA	CPSC	FDA	EPA	CPSC
Tea (bags)	8	120/284	2.6×10^{-7}	2.3×10^{-6}	7.3×10^{-7}	6.1×10^{-7}	5.4×10^{-6}	1.7×10^{-6}
Margarine (wrap)	82	9/19	2.0×10^{-7}	1.8×10^{-6}	5.6×10^{-7}	4.3×10^{-7}	3.8×10^{-6}	1.2×10^{-6}

[a]Food intake obtained by multiplying the MRCA mean and 90th percentile frequencies of eating occasions (14-day average, 1982-87 5-year Menu Census) (MRCA 1987) by the mean grams/eating occasion from USDA/NFCS, 1977-78 (Pao et al. 1982). Data are for the 2+ years age group, males and females, eaters-only population.

[b]See Table 15, footnote [b] for TEQ levels in the paper article.

[c]Mean/90th percentile values.

[d]See Table 15, footnote [c]. Because of the conservatism of the per capita estimate, we have selected 37 g/p/d to represent the mean food intake eaters-only value as well. The 90th percentile value was assumed to be 2 times the mean. This is reasonably consistent with the relationship between the mean and 90th percentile figures for the other entries in the table.

[e]NCASI estimated per capita consumption of 1.6 g/person/day based on 1.4 billion bags sold in the U.S. in 1989. To obtain the eaters-only mean intake, we have assumed that all microwaveable popcorn is consumed by only 10% of the U.S. population. The 90th percentile value was assumed to be 2 times the mean.

[f]Assumes a typical body weight of 60 kg for an adult.

[g]EPA classifies 2,3,7,8-TCDD as a "B2" carcinogen.

Table 10-4 presents the results of the cancer risk assessment for the "eaters only - food-by-food basis." The maximum estimated risk for mean consumers (eaters only) of any one of the food products is less than 1.4×10^{-6} using FDA's cancer potency slope factor and less than 1.2×10^{-5} using EPA's cancer potency slope factor. The risks for 90th percentile (eaters-only) consumers of individual foods are approximately two times greater than risks for the mean (eaters-only) consumer.

10.6.2 Non-Cancer Risks

FDA used an ADI of 1-10 pg/kg/day to assess non-cancer risks of potential exposures. This is the most sensitive non-cancer toxicological endpoint associated with dioxin exposure in animal studies. Although the estimated daily exposures in units of pg/kg/body weight/day are not shown in Tables 10-2 and 10-3, the calculated exposures were all less than the ADI.

10.6.3 Uncertainties

The major uncertainties inherent in this assessment concern assumptions regarding food intake rates and dioxin migration rates. The estimation of exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF from consumption of all foods contacting paper articles requires consideration of appropriate food intake information. Available nationally representative food consumption data bases, however, do not provide information on whether foods are sold, held, heated, cooked, or served in contact with different types of materials. Therefore, for estimating the total dioxin TEQ intake from all foods that may contact bleached paper, available food consumption data for average consumers of each of the foods were used in conjunction with the assumption that all such foods have indeed been in contact with bleached paper prior to being consumed. This necessary assumption leads to an overestimate of consumption of food that has been in contact with paper and, hence, to an overestimate of total dioxin TEQ exposure.

This upper-bound estimate of total dioxin TEQ exposure (12.7 pg/p/day) was adjusted through a comparison with a lower-bound estimate of dioxin TEQ exposure, specifically, a per capita exposure, obtained using industry-provided production figures and estimates of the amount of food that might contact the paper. Recognizing that a reasonable estimate should fall between the upper- and lower-bound estimates, FDA derived a "best estimate" of mean total dioxin TEQ exposure of 9.1 pg/person/day. This value is the mid-point between the upper- and lower-bound estimates.

For the "eaters only - food-by-food basis" assessment, it was also assumed that all foods consumed have indeed been in contact with bleached paper prior to being consumed. The other assumptions used for the total

population exposure analysis also apply to the "eaters-only" analysis. Therefore, the dioxin TEQ intakes in Table 10-4 may also be considered as upper-bound estimates of mean and 90th percentile for "eaters" of each of the foods. The dioxin TEQ exposures in Table 10-4 must not be summed because the population of eaters is not the same for each food category.

All of the migration studies demonstrated detectable levels of transfer of the dioxin congeners to the test foods. The temperature of foods, their composition, the nature of the paper article, as well as the dioxin congener levels in the paper articles were all found to influence the extent of transfer to test foods. Although recent production lots of paper and paperboard used in food application have been shown to have significantly lower levels of dioxin congeners than the levels used for this assessment and risks from these articles should be expected to be lower, FDA has not used these recently submitted figures to try to estimate lower risks because the quality and the completeness of these reports have not been assessed and migration studies which might show the extent of reduced exposure (and risk) have not been conducted.

Even considering these uncertainties, the results of the assessment of individual lifetime cancer risks indicate a potentially significant risk is posed by "current" (i.e., as used in this assessment) levels of dioxin in paper food contact product. Although individual risks are not high, the potentially exposed population is much larger than any other exposed population group addressed in the Integrated Assessment.

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11. ASSESSMENT OF RISKS TO THE GENERAL POPULATION FROM DIOXINS AND FURANS IN CELLULOSE DERIVATIVES USED IN FOOD, DRUG, AND COSMETIC FORMULATIONS

11.1 Introduction

This chapter is a condensed version of two reports prepared by the Quantitative Risk Assessment Committee (QRAC) of the U.S. Food Drug Administration (FDA) as part of the Interagency Dioxin-in-Paper Workgroup:

USFDA. U.S. Food and Drug Administration. 1990a. Carcinogenic risk assessment for dioxins and furans in cosmetic products containing cellulose derivatives produced from bleached wood pulp. Report of the Quantitative Risk Assessment Committee. March 20, 1990.

USFDA. U.S. Food and Drug Administration. 1990b. Carcinogenic risk assessment for dioxins and furans in cellulose derivatives used in foods and ingested drug products. Report of the Quantitative Risk Assessment Committee. April 24, 1990.

The Center for Food Safety and Applied Nutrition's (CFSAN) Quantitative Risk Assessment Committee (QRAC) assessed the potential risks to persons using various food, drug, and cosmetic products containing cellulose derivatives prepared from bleached wood pulp that may be contaminated with dioxin congeners. Information on exposure to dioxin congeners from the use of these products was provided to the QRAC by the Food and Color Additives Review Section of CFSAN.

11.2 Exposure to Dioxins and Furans from Use of Cosmetic Products

11.2.1 Identity and Use of Cellulose Derivatives

Much of the information on cellulose derivatives usage in cosmetic products was provided to CFSAN by the paper industry. Powdered cellulose and various cellulose ethers are used in a wide range of leave-on type cosmetic products such as lotions, creams, and powders and in wash-off products such as shampoos, conditioners, and non-fluoride toothpastes. The leave-on products are reported to contain less than 2 percent cellulose derivatives, while wash-off products contain less than 1 percent. The cellulose derivatives that are used in cosmetic products fall into two different classes: water-insoluble cellulose derivatives including powdered cellulose and microcrystalline cellulose, and water-soluble, or dissolving cellulose derivatives, that include a variety of cellulose ethers and esters.

11.2.2 Dioxin Concentration in Cellulose Derivatives

Although analytical information is not available on the concentrations of dioxin congeners in cellulose derivatives, information is

available on the concentration of dioxin congeners in pulp that is subsequently converted into the various cellulose derivatives. The available data from the 104-Mill Study show that none of the pulps used to prepare cellulose-derived products that are added to cosmetic formulations contain detectable levels of either 2,3,7,8-TCDD or 2,3,7,8-TCDF. Rather than assuming that these congeners are not present at any level in the cellulose derivatives, FDA assumed their presence at no greater than one-half of the average detection limit (range 0.2-1 ppt, avg. 0.6 ppt) of the analytical method used, or about 0.3 ppt of dioxin TEQ.

11.2.3 Cosmetic Product Use

The paper industry indicated that a wide range of cosmetic products contains cellulose derivatives in their formulation; however, the information was not product specific. Therefore, it was assumed that (1) all cosmetic products applied to the skin are likely to contain such derivatives and (2) each cosmetic product contains these derivatives at the maximum level reported by the industry.

Information on the frequency of use and amount of cosmetic product applied per use was derived from USFDA (1983). To account for the high amount of cosmetic products used by some individuals, a 90th percentile daily use rate for each of the cosmetic products was used. This rate was derived by combining information on the average amount of cosmetic product used per application with 90th percentile frequency of use. The amount of cosmetic product used each day was multiplied by the use level of the cellulose derivative and the assumed maximum concentration of dioxin in the cellulose derivative to obtain an upper bound estimate of the potential dioxin exposure from each cosmetic product. However, to estimate probable dioxin exposure, it is necessary to account for the extent to which wiping, wearing off, or washing remove the cosmetic products from the skin (thus, decreasing the potential for exposure to dioxin contaminants). Correction factors proposed by the Color Additive Scientific Review Panel in September 1985 to describe the percent of the cosmetic product that is available for absorption are used in the risk calculations.

When information on the amount of cosmetic product used each day was combined with information on the extent of cosmetic product that remains available for absorption, a few cosmetic products were found to dominate the potential for dioxin exposure. Rather than examining the potential dioxin exposure that may occur from each cosmetic product, only those cosmetic products thought to present the greatest potential for consumer exposure to dioxin have been addressed. Examples are dentifrices, body lotions, and hair shampoos. Other cosmetic products have either lower daily use rates, lower levels of the cellulose derivative, or most of the product is washed off following use. It was concluded that dioxin and

furan exposures that may occur with other cosmetic products will be subsumed within the exposures estimated to occur from dentifrices, body lotions, and hair shampoos.

11.2.4 Dermal Absorption of Dioxin from Cosmetic Products Applied to the Skin

Limited information is available on the rate of percutaneous absorption of dioxin. Many factors affect this rate, such as the nature of the cosmetic product, the duration of dermal contact, the amount of dioxin deposited on the skin, the anatomic site, and the age and health of the skin. The Consumer Product Safety Commission (CPSC) has assembled a review of available information on studies of dioxin dermal transport. CPSC reported that in in vitro experiments with human skin (Weber et al. 1989) and in absorption studies with rats (Poiger and Schlatter 1980), 19 to 29 percent solvent-deposited dioxin was absorbed. CPSC used 25 percent as a measure of the fraction of dioxin likely to be absorbed by human skin 24 hours after dioxin contact. This value is used in this assessment as well. Because of the limited amount of data on dioxin skin absorption and the low levels of exposure estimated for individuals using high amounts of cosmetic products, no attempt was made to develop exposure estimates for individuals with damaged or aged skin.

11.2.5 Dioxin Exposure from Cosmetic Products Applied to the Skin

Table 11-1 contains a summary of the average daily TEQ exposures that were estimated for users of dentrifrices, lotions, and shampoos as well as the parameters used to calculate the exposures. Daily exposures were estimated to range from 0.0001 pg/person/day from use of shampoos to 0.005 pg/person/day from use of lotions.

11.3 Exposure to Dioxins and Furans from Use of Cosmetic Wet Wipes

11.3.1 Identity and Use of Cosmetic Wet Wipes

Cosmetic wet wipes used to wipe hands and in diaper changes are manufactured using either synthetic fibers or bleached pulp. Information on the composition and use of these wipes was provided by the National Council of the Paper Industry for Air and Stream Improvement (NCASI 1989). Wipes dispensed from pop-up containers do not contain any pulp; however, those which are dispensed from tubs in which the wipes lie flat in a stack are composed of bleached pulp (75 to 85 percent of dry weight) with the remaining material being binders and synthetic fibers.

Potential exposure to dioxin congeners in the wet wipes made from bleached pulp can occur as a result of migration from the web into the lotion during extended storage, transfer of the lotion to the skin during wet wipe usage, and dermal uptake during the time between wet wipe usage and subsequent washing of the skin.

Table 11-1. Dioxin Exposure From Cosmetic Products Applied to the Skin

Product type	Grams used per day[a]	Conc. of deriv. in prod.	Percent available for absorption[b]	Percent absorbed	Dioxin TEQ absorbed[c] (pg/p/day)
Dentifrices	0.15	$\leq$ 1%	100	100	0.0005
Lotions	7.0	$\leq$ 2%	50	25	0.005
Shampoo	16.4	$\leq$ 1%	1	25	0.0001

[a]90th percentile (USFDA 1983).
[b]Hart et al. (1986).
[c]It is assumed that the dioxin TEQ level in cellulose derivatives is 0.3 ppt.

11.3.2 Dioxin Concentration In and Extraction From Wet Wipes

Analyses of pulp and web from two manufacturers have shown 2,3,7,8-TCDD levels in the 0.5 ppt to 4 ppt range and 2,3,7,8-TCDF levels ranging from 2 ppt to 39 ppt (NCASI 1989). Due to the lack of analytical data for all manufacturers of wet wipes, it was assumed that all of the wet wipes are manufactured from pulp containing dioxin congeners at a level equivalent to the average pulp value found in the 104-Mill Study (17 ppt dioxin TEQ) rather than the highest level in webs analyzed by the paper industry (8 ppt dioxin TEQ) (NCASI 1989).

Information on the extent of extraction of dioxin congeners from the web during storage and on the skin penetration rate by dioxin is currently being developed by the paper industry. However, the paper industry has provided preliminary findings from experiments conducted with two representative types of wipe lotions and two levels of surfactants (1 and 3 percent) for periods up to 90 days. The partition coefficients (the ratio of the concentration of the dioxin congener in the pulp to the concentration in the lotion) for the dioxin and the furan were approximately 900 for the 1 percent solution and approximately 300 for the 3 percent solution.

11.3.3 Wet Wipe Use Information

Total wet wipe use for six daily diaper changes was estimated to be eight uses per day (NCASI 1989). Wet wipe use information for periods other than during diaper changes has not been developed; it was assumed that because of the high frequency and duration of wet wipe use during years of diaper changes that wet wipe use for other periods will be much less frequent and will contribute little to total exposure. Therefore, it was further assumed that infants will be exposed to wet wipes for a period of three years and that primarily one adult will use wet wipes for six years caring for an average of two children.

The amounts of lotion deposited on the skin of an infant during diaper changes and on the hands of adults during these periods of wet wipe use are dependent on the area of exposed skin area and the duration of skin contact during wiping. NCASI (1989) has estimated the amount of lotion transferred to skin for each use scenario, accounting for such factors as weight of lotion lost by evaporation during wiping, whether the wipe came from the top, middle or bottom of the tub, and differences between three different wet wipe manufacturers. The average amount of fluid transferred from wipes to the skin was 0.83 ml/166 in^2.

NCASI (1989) also used the surface area of a medium-sized diaper (195 in^2) as a measure of the area of skin that is likely to be contacted by the lotion of a wet wipe. It was estimated that 0.98 ml of lotion will be transferred to infants for each diaper change. Using the

surface area of the palm and outstretched fingers of adult males and females (23 in^2 and 19.4 in^2, respectively), it was estimated that fluid transfer for males and females will be 0.117 ml and 0.097 ml per wipe, respectively (NCASI 1989). For simplicity, this assessment is based on conversion of these figures to percent of lotion in the wet wipe that is left on the skin - 20 percent for infant use and 2 percent for adult use.

11.3.4 Dermal Absorption of Dioxin Congeners

Studies of the effect of skin contact time on dermal absorption of dioxin for children vs. adults have not been performed. Therefore, it was assumed that all of the 2,3,7,8-TCDD and 2,3,7,8-TCDF in the applied lotion is available for absorption. The absorption coefficient of 25 percent has been used as an estimate of the fraction of dioxin and furan absorbed during the shorter exposure intervals associated with infant and adult use of wet wipes. Using the 24-hour absorption coefficient for modeling dermal uptake during shorter periods is assumed to be conservative. Therefore, a correction factor to account for the relatively fast dermal absorption rates of damaged skin that might be experienced by infants with diaper rash was not used.

11.3.5 Dioxin Exposure from Wet Wipes

Table 11-2 contains a summary of the average daily dioxin TEQ exposures that were estimated for infants and adults for periods of wet wipe use during diaper changes as well as the parameters used to calculate the exposures. Daily infant exposures were estimated to be 0.13 picograms (pg) dioxin TEQ which is 10 times higher than the figure (0.013 pg) calculated for adults.

11.4 Exposure to Dioxins and Furans in Cellulose Derivatives Used in Foods and Drug Products

11.4.1 Identity and Use of Cellulose and Cellulose Derivatives

Cellulose (powdered and microcrystalline) and a variety of cellulose-derived esters and ethers are used in food and drug formulations to accomplish a variety of technical effects. The paper industry has indicated that food uses of powdered cellulose include baked goods (e.g., bread, cookies, rolls, crackers, pie fillings, icings), dairy products (e.g., ice cream, whipped toppings, milk shakes), pasta, sausage casings, diet beverages, tablets, and "miscellaneous items" such as candy, dried fruits, and flavor carriers. Microcrystalline cellulose and cellulose-derived ethers and esters are added to baked goods (similar uses as powdered cellulose), dairy products and related substitutes, pasta, tablets, fried foods, syrups, soups, sauces, dressings, and

Table II-2. Dioxin Exposure From Wet Wipes

User	WP	CP	WL	PD	K	PA	FU	DA
Infants	2.1	17	6.0	20	300	25	8	0.13
Adults	2.1	17	6.0	2	300	25	8	0.013

DA = Dioxin TEQ absorbed/person/day in picograms (pg)

$$= WL \times PD \times CL \times PA \times FU$$

$$= \frac{WL \times PD \times CP \times WP \times PA \times FU}{(K \times WP) + WL}$$

WL = Weight of lotion per wet wipe in grams
PD = Percent of lotion deposited on the skin as decimal
CL = Concentration of dioxin TEQ in lotion (ppt)

$$CL = \frac{CP \times WP}{(K \times WP) + WL}$$

CP = Concentration of dioxin TEQ in web used for wet wipes (ppt)
WP = Weight of paper used for each wet wipe in grams
K = Partition coefficient of dioxin between paper and lotion
PA = Percent of dioxin on skin absorbed as decimal
FU = Number of wet wipes used per day

Sample Calculation for Wet Wipe Exposure (Infants):

$$DA = \frac{WL \times PD \times CP \times WP \times PA \times FU}{(K \times WP) + WL}$$

= (6.0 g lotion/wet wipe) x (0.20) x (17×10^{-12} g dioxin TEQ) x (2.1 g pulp/wipe) x (0.25) x (8 wipes/day) divided by [(300 x 2.1) + 6.0]

= 0.13×10^{-12} g dioxin TEQ

"miscellaneous items" such as candy, dried fruits, and flavor carriers. The paper industry also provided general information on the range of use levels for these derivatives in most of the food categories. For these applications, the technical effects accomplished by these cellulose derivatives include those of an anticaking agent, formulation aid, processing aid, stabilizer and thickener.

Cellulose derivatives are also used widely in the formulation of drug tablets, suspensions, and creams. Although the cellulose derivatives serve as inactive ingredients in nearly all of these applications, methylcellulose and sodium carboxymethylcellulose serve as active ingredients when they are used as bulking agents in laxatives. Because of the large amounts required to accomplish the bulking effects, laxatives represent the greatest potential for exposure to cellulose derivatives from drug products.

11.4.2 Dioxin Concentration in Cellulose Derivatives

The available data from the 104-Mill Study show that none of the pulps used to prepare cellulose-derived products that are added to foods and ingested drug formulations contain detectable levels of 2,3,7,8-TCDD. But, one of the mills reported low levels of 2,3,7,8-TCDF (equivalent to less than 0.5 ppt dioxin TEQ). Rather than assuming that these congeners are not present at any level in the pulps used to prepare the cellulose derivatives, FDA assumed that they are present at one-half of the average detection limit (range 0.2 - 1 ppt, ave. 0.6 ppt) of the analytical method used (i.e., about 0.3 ppt of dioxin TEQ).

Further, FDA assumed that the chemical and mechanical processing steps used to prepare the cellulose derivatives do not increase the residual levels of dioxins and furans above the levels assumed to be present in the bleached wood pulp used to manufacture these derivatives. Using dioxin equivalency factors, total dioxin TEQ in cellulose derivatives used in food and ingested drug products is estimated by FDA to be no greater than 0.3 ppt.

11.4.3 Dioxin Exposure Estimates

(1) General approach used. One approach to the estimation of possible exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF from use of cellulose derivatives in food and drug applications involves the use of information on the annual poundages of the cellulosics directed for use in foods and drugs. Such information allows a per capita exposure estimate assuming that total production of cellulose derivatives is evenly distributed across the entire U.S. population. Per capita exposures, however, tend to underestimate exposure to individuals consuming those items in which a particular substance is most heavily used, or to individuals who consume many products at high levels in which the substance is used. Per capita

exposures (or per capita disappearances) can be useful, however, as a check or validation of other types of exposure estimates that more accurately reflect the characteristics (e.g., age, sex, ethnicity) of the exposed population.

For the current problem, the paper industry has provided data only for total bleached wood pulp-derived cellulose (59,000 tons) of which approximately 25 percent was reported to go towards foods, drugs, and cosmetics. Furthermore, the NAS 1987 food additive/ingredient poundage survey of industry contains information on annual poundages of a number of cellulosics directed into the food supply. These data are known to be underreported. Therefore, FDA has foregone the presentation of a per capita exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF and has based the assessment on food consumption survey data in the case of exposure to dioxin TEQ from foods containing cellulosics and on information from the FDA Center for Drug Evaluation on drug use.

(2) Exposure estimates for foods. The estimation of exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF from consumption of foods containing cellulose derivatives requires consideration of the appropriate food intake information and information on the specific foods to which cellulosics may be added. The paper industry has provided some information on the food uses of cellulosics. Additional information on use is contained in the National Academy of Sciences 1977 Survey of Industry and in agency memoranda in CRP 4T0133. In general, cellulosics are used in a wide variety of food products and can be considered to be ingested by a broad segment of the U.S. population--with perhaps one exception--*alpha*-cellulose used to make the "high fiber" breads. These breads are selected by consumers for their high fiber, i.e., their cellulose content.

The use of available nationally representative food consumption data for those foods to which cellulosics would be added, when combined with appropriate use level information, will lead to an overestimate of exposure to cellulose derivatives and, hence, to an overestimate of total dioxin TEQ exposure. This is because of the necessary assumption that all foods which may contain a cellulosic do contain a cellulosic.

Because we believe that 100 percent of the U.S. population are "eaters" of at least one food containing a cellulose derivative on a frequent or chronic basis, it was assumed that 100 percent of the U.S. population are "eaters" of 2,3,7,8-TCDD and 2,3,7,8-TCDF. Therefore, estimates of food intake used to estimate total dioxin TEQ intake should be representative of the total population. We selected the Market Research Corporation of America (MRCA) database, the five-year Menu Census, 1982-87, as our source of food intake data. This data base contains 14 consecutive days of data and is likely to capture a representative diet including both frequently and infrequently consumed foods.

Total-sample intakes for each food of interest in the MCRA data base were multiplied by the use levels of the appropriate cellulose derivative to obtain the intake estimate for the cellulose derivative. The intake estimate for each cellulosic was multiplied by its dioxin TEQ concentration to obtain the set of dioxin TEQ intakes that could be summed to provide an upper-bound total population mean dioxin TEQ intake, i.e., 1.4 pg/person/day (see Table 11-3).

As seen from Table 11-3, the prime contribution to total mean dioxin TEQ exposure derives from baked goods, and bread, in particular. Because of the high probability that cellulose-containing bread would be selectively consumed by individuals interested in high-fiber foods, it is evident that for those individuals who do not consume high-fiber bread, the total mean exposure to dioxin TEQ of 1.4 pg/person/day represents a substantial overestimate. For those consumers who do select for high-fiber bread, however, we have looked at potential dioxin TEQ intake using those individuals identified as "eaters" of bread during the 14-day MRCA survey. We have assumed that an eater of high-fiber bread consumes this product at the same level as an eater of "regular" bread, because the survey data available to us do not contain intake data specific to high-fiber bread. Thus, the eaters-only bread intakes (white bread) are: 41 g/person/day (mean) and 73 g/person/day (90th percentile). Assuming the bread contains 7.5 percent *alpha*-cellulose with a dioxin TEQ level of 0.3 ppt, the eaters-only dioxin TEQ exposures are: 0.92 pg/person/day (mean) and 1.6 pg/person/day (90th percentile). The eaters-only mean exposure of 0.92 pg/person/day is essentially the same as that for the total-sample value given in Table 11-3 of 0.90 pg/person/day. This is because the percentage of eaters of bread in the MRCA survey is essentially 100 percent.

Although it is evident that the total population dioxin TEQ exposure is highly conservative, it may not be as evident for the exposure estimate for those individuals selectively consuming high-fiber bread. Several factors should be noted that reflect the conservatism of this exposure as well. First, the assumed use level of *alpha*-cellulose (7.5 percent) is believed to be a maximum level rather than a typical level. Second, it is assumed that all bread consumed by an individual is high-fiber bread. Third, it is assumed that a consumer of high-fiber bread consumes this bread at the same rate as one who eats regular bread. Fourth, the concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF in all the cellulose derivatives are expected in nearly all instances to be substantially lower than the 0.3 ppt value we have used. The current industry trend appears to be toward ever lower levels as industry more carefully controls dioxin levels in its various manufacturing processes.

(3) Exposure estimates for drugs. For drug uses of cellulosics, available information indicates that the major uses are either as an inactive ingredient as a binder in drug tablets or as an active

Table 11-3. Total Sample Basis Dioxin TEQ Exposure from Foods

	Food use (cell. deriv.)[a]	Cell. deriv. level in food[b] (g/p/d)	Food intake[c,d] (g/p/d)	Cell. deriv. intake (mg/p/d)	Dioxin TEQ intake (fg/p/d)
1.	Bread, wht. or dk. (alpha)	7.5	39	2,900	900
2.	Eng. muf., bagels (alpha)	5	3.4	170	51
3.	Crackers (alpha)	5	2.1	110	33
4.	Stuffings (alpha)	5	6.5	300	90
5.	Diet baked goods (alpha)	5	0.7	40	12
6.	Diet baked goods (HPMC)	0.4	0.7	3	0.9
7.	Other baked goods mixes (alpha)	5	12	600	180
8.	Cakes (SCMC)[f]	0.1	11	11	3.3
9.	Donuts (SCMC)	0.1	7.7	7.7	2.3
10.	Rice (EC)[g]	0.6	12	69	21
11.	Non-stnd. dressing (SCMC)	0.06	4.5	2.6	0.8
12.	Diet fats, oils (SCMC)	0.06	1.7	1.0	0.3
13.	Juice drinks, ades (SCMC)	0.03	8.6	2.5	0.8
14.	Other frt. juices (MC)	0.5	4.3	22	6.6

Table 11-3. (Continued)

Food use (cell. deriv.)[a]	Cell. deriv. level in food[b] (g/p/d)	Food intake[c,d] (g/p/d)	Cell. deriv. intake (mg/p/d)	Dioxin TEQ intake (fg/p/d)
15. Dried wht. potatoes (HPMC)	0.5	0.6	2.9	0.9
16. Sugar panned candy (HPC)	0.2	7.0	14	4.2
17. Diet swt. sauces (SCMC)	1.2	0.13	1.6	0.5
18. Dry beverage mixes (SCMC)	0.01	0.27	2.7	0.8
19. Diet beverages non-cola (SCMC)	0.02	9.5	1.9	0.6
20. Toppings, dairy (HPMC)	0.4	0.4	1.5	0.5
21. Coffee whiteners (alpha)	1.3	0.64	83	25
22. Meat products (alpha)	0.1	1.7	1.7	0.6
23. Nut products (alpha)	0.6	6.9	40	12
24. Other foods[h] (alpha)	0.1	200	200	60
TOTAL = 1,407 fg/p/d or 1.4 pg/p/d				

[a] Cellulose derivative abbreviations:
alpha = alpha-cellulose (microcrystalline and powdered cellulose share many of the same food uses; we have assumed substitutability)
HPMC = hydroxypropylmethylcellulose
SCMC = sodium carboxymethylcellulose
EC = ethylcellulose
MC = methylcellulose
HPC = hydroxypropylcellulose

Table 11-3. (Continued)

Footnotes (continued):

b Use levels for SCMS, MC, HPMC, EC, and HPC are from the NAS 1977 Survey of Industry. Use levels for alpha-cellulose are from memoranda and information in GRP 4T0133.

c Obtained by multiplying the MRCA mean frequency of eating occasions (14-day average, 1982-87, 5-year Menu Census) by the mean grams/eating occasion from USDA/NFCS, 1977-78. Data are for the 2+ years age group, males and females, total sample populations.

d Grams/eating occasion for numbered items: 1-44.2; 2-57.2; 3-20.0; 4-65.7; 5-37.7; 6-37.7; 7-105; 8-79.6; 9-67.8; 10-146; 11-31.5; 12-18.3; 13-276; 14-142; 15-164; 16-51.3; 17-25.8; 18-277; 19-303; 20-13.5; 21-8.2; 22-59.2; 23-40.0.

e The Dioxin TEQ level in all cellulose derivatives was taken as 0.3 ppt. This is one-half of the nondetect level (0.6 ppt) for analytical methods used to measure dioxin congener levels in the pulps used to manufacture the cellulose derivatives.

f SCMC and carboxymethylethylhydroxycellulose are assumed interchangeable.

g EC and methylethylcellulose are assumed interchangeable.

h According to a memorandum of 8/24/81 (J. Modderman, Ph.D. to L. Gosule, GRP 4T0133), alpha-cellulose was considered technologically useful in a number of food categories at use levels no greater than 0.1 percent. It was assumed that the individual daily intake for these "other foods" would total to 204 g; this represented an "eaters only" value. We have included this value (200 g) in our tabulation. The dioxin TEQ intake from this "category" is insignificant so no effort was made to correct the total sample basis.

ingredient as a bulking agent in laxatives. A review of FDA files of drug formulations showed that when various cellulose derivatives (e.g., powdered cellulose, microcrystalline cellulose, hydroxypropylmethylcellulose, and ethylcellulose) are used as binders of tablets that may be consumed on a chronic basis, daily ingestion of the cellulose derivatives will total less than 1 g/person/day. In contrast, the use of either methylcellulose or sodium carboxymethylcellulose in laxatives can result in daily doses of either of these derivatives as high as 6 g/person/day (Handbook of Non-Prescription Drugs, 7th ed., 1982). Although the recommended use of laxatives containing cellulosics is for a maximum period of just 2 weeks, the FDA Center for Drug Evaluation has indicated that elderly individuals (60-70 years) with reduced gastric motility may use such laxatives on a chronic basis. However, this would involve less than lifetime exposure.

For estimating the upper-bound intake of dioxin TEQ from drugs, we have provided separate estimates for the maximum exposures that may occur from tablet binders and laxatives (Table 11-4). While both estimates are expected to overestimate actual exposure because they are based on assumed dioxin TEQ levels in the derivatives of half of the detection limit, the exposure estimated for laxative use has an additional exaggeration because lifetime use of such products is not expected.

11.5 Cancer Risk Estimates

Table 11-5 summarizes the estimated exposures (normalized for body weight) and lifetime cancer risks for cosmetic products, wet wipes, and food and drug products containing cellulose or cellulose derivatives. Risk estimates were calculated using the cancer slope factors derived by FDA, EPA, and CPSC for 2,3,7,8-TCDD. The slope factors, which were derived from the results of animal experiments where rats were exposed to 2,3,7,8-TCDD in the diet, are expressed in terms of administered dose. EPA and FDA have estimated that 55 percent of 2,3,7,8-TCDD administered to rats in the diet is absorbed into the body. CPSC has estimated that 75 percent is absorbed. Therefore, in Table 11-5, the upper bound lifetime risks were corrected for oral bioavailability where appropriate, i.e., in the case of lotion, shampoo, and wet wipe (adult and infant) use.

Also, although non-cancer risks were not assessed in the source document, comparison of the daily exposure estimates in Table 11-5 with the estimated RFD of 1 pg/kg/day and the estimated health advisories of 10 to 300 pg/kg/day that were developed by EPA for the purposes of this assessment indicates that the exposures pose minimal risk of non-cancer effects.

11.6 Analysis of Uncertainties

It should be emphasized that the risks presented in Table 11-5 are not additive. Several conservative assumptions about exposure have been

Table 11-4. Upper Bound Intake of Dioxin TEQ from Drugs

Cellulose product[a] application	Cellulose product intake (g/person/day)	Upper-bound intake dioxin TEQ (pg/person/day)
Tablet binders	≤ 1	0.3
Laxatives	6[b]	1.8[b]

[a] It is assumed that the cellulose products contain dioxin TEQ at 0.3 ppt, i.e., one-half of the non-detect level (0.6 ppt) of analytical methods used to measure dioxin congener levels in pulps used to manufacture the cellulose derivatives.

[b] Estimated intakes of cellulose derivatives and dioxin TEQ from laxatives exceed lifetime average exposure because chronic use of the laxatives only occurs for a fraction of a lifetime.

Table II.5 Upper Bound Carcinogenic Risk for Users of Food, Drug, and Cosmetic Products Containing Cellulose Derivatives

User	Lifetime average dioxin TEQ exposure (pg/kg/day)	Upper bound lifetime risk[c] FDA	EPA	CPSC
Cosmetic Products				
Dentifrice user	8×10^{-6}	1.3×10^{-10}	1.2×10^{-9}	3.6×10^{-10}
Lotion user	8×10^{-5}	2.4×10^{-9}	2.1×10^{-8}	6.7×10^{-9}
Shampoo user	1.6×10^{-6}	4.7×10^{-11}	4.2×10^{-10}	1.3×10^{-10}
Cosmetic Wet Wipes				
Wet wipe (adult)	1.8×10^{-5} (2.2×10^{-4})[a]	5.8×10^{-10}	5.2×10^{-9}	1.6×10^{-9}
Wet wipe (infant)	5.6×10^{-4} (1.3×10^{-2})[a]	1.6×10^{-8}	1.4×10^{-7}	4.5×10^{-8}
Food and Drugs				
All foods	2.3×10^{-7}	4×10^{-7}	3.6×10^{-6}	1.1×10^{-6}
High fiber bread	1.5×10^{-7} (2.7×10^{-7})[b]	2×10^{-7} (4×10^{-7})[b]	1.8×10^{-6} (3.6×10^{-6})[b]	5.6×10^{-7} (1.1×10^{-6})[b]
Tablet binders	5×10^{-3}	8×10^{-8}	7.1×10^{-7}	2.2×10^{-7}
Laxatives	3×10^{-2}	5×10^{-7}	4.4×10^{-6}	1.4×10^{-6}

[a] Value in parenthesis denotes the daily dose during the period of exposure (i.e., 3 years for infant and 6 years for adult).

[b] Value in parenthesis denotes exposure and risk for 90th percentile consumption rate.

[c] EPA has classified 2,3,7,8-TCDD as a "B2" carcinogen

made earlier in this report which affect the risk estimates. First, dioxin congeners have been assumed to be present at one-half the detection limit when such congeners may be present at a lower level or not present at all. Second, products which may contain a cellulosic have been assumed to do so. Third, while 100 percent of the U.S. population is likely to consume regularly at least one food containing a cellulose derivative, it is unlikely that 100 percent of the population would consume all foods containing a cellulose derivative. Fourth, for the high-fiber bread consumer, such breads have been assumed to contain a maximum, rather than a typical, level of cellulose; it has also been assumed that the high-fiber bread consumer eats only high-fiber bread and at the same rate as one who eats regular bread. Fifth, exposure to laxatives and tablets containing cellulose tablet binders would be less than lifetime. The risk figures presented in Table 11-5 are for lifetime exposure since FDA does not believe that methodology currently exists to accurately adjust these figures for less-than-lifetime exposure.

11.7 References

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12. FDA ASSESSMENT OF RISKS FROM EXPOSURE TO DIOXINS AND FURANS IN FISH CONTAMINATED BY BLEACHED KRAFT PULP AND PAPER MILLS

12.1 Introduction

This chapter is a condensed version of a report prepared by the Quantitative Risk Assessment Committee (QRAC) of the U.S. Food and Drug Administration (FDA) as part of the Interagency Dioxin-in-Paper Workgroup:

> USFDA. U.S. Food and Drug Administration. 1990. Carcinogenic risk assessment for dioxins and furans in fish contaminated by bleached-paper mills. Draft Report of the Quantitative Risk Assessment Committee. January 19, 1990.

Historically, FDA has dealt with hazards and risks of contaminants in fish as part of the agency's mission to protect the public from food-borne hazards. Fish collected in the vicinity of pulp mills have been found to contain levels of dioxins and related furans that are substantially higher than those levels in fish caught in areas remote from the mills. The populations judged by QRAC to be most at risk are subsistence fishers and sports fishers since they are likely to consume above-average amounts of fish on a regular basis.

FDA had previously addressed the issue of dioxin exposure resulting from the ingestion of fish contaminated with dioxin. As part of congressional testimony on the issue of a Great Lakes fish advisory, on June 30, 1983, Dr. Sanford Miller, Director of the Bureau of Foods, presented before the Subcommittee on Natural Resources, Agriculture, Research and Environment, Committee on Science and Technology, a safety assessment in support of an advisory that expressed concern about persons consuming fish on a chronic basis containing dioxin at greater than 25 parts per trillion (ppt).

It is important to note that QRAC does not have accurate information on the numbers of subsistence and sports fishers in the vicinity of pulp mills. QRAC also lacks accurate fish consumption data for these persons, as well as a thorough profile of dioxin congener levels in fish living in waters near pulp mills. In an attempt to bridge these gaps in knowledge, QRAC has used estimates discussed in detail in the Food and Color Additives Review Section (Center for Food Safety and Applied Nutrition-CFSAN) memorandum of November 13, 1989. (Memorandum, G. Cramer, Ph.D. to S. Henry, Ph.D.). These estimates will be summarized in the following discussion.

12.2 Levels of Dioxin Congeners in Fish

EPA's Environmental Research Laboratory in Duluth, Minnesota, recently conducted the National Bioaccumulation Study (USEPA 1989a). This study

involved the analysis of fish tissue for dioxin and furan (and about 65 other pollutants) in whole body and fillet portions of bottom-feeding and predator fish collected at 400 locations across the U.S. Levels of 2,3,7,8-TCDD and furan and other isomers and congeners were reported as dioxin toxic equivalents (TEQ). This study confirmed earlier findings that fish obtained from waters in the vicinity of pulp mills contain higher levels of dioxin and furan than fish caught in areas remote from the mills (USEPA 1988).

EPA's fish analyses included a variety of predator and bottom-feeding fish, such as bass, walleye, perch, trout, catfish, carp, and buffalo fish. The highest levels of dioxin TEQs in fillets were found in perch (24.3 ppt), trout (22.5 ppt), crappie (22.1 ppt), and large mouth bass (20.4 ppt); the lowest values were reported to be less than 0.1 ppt.

The levels of dioxin and furan congeners in fish samples as part of the National Bioaccumulation Study, although limited, offer the best available measure of likely levels of dioxin congeners in fish near pulp mills. Only a few samples of fish were collected at most of the mills and most analyses were done on whole fish. Since dioxin and furan congeners concentrate in the organs of the fish and people typically eat cleaned fish without the organs, the results of fillet composites from the few samples collected in the region of pulp mills have been used. Dioxin TEQs were calculated using EPA's 1987 interim procedures for estimating risks from exposure to mixtures of dioxins and furans (USEPA 1989b). Calculating dioxin TEQs using the EPA's March 1989 revised procedures would not substantially change the TEQ values.

Although some fishers probably consume on a chronic basis fish caught downstream from only one of these mills, QRAC does not believe that there are adequate fish data for the individual mills to warrant a mill-by-mill analysis of possible exposure to dioxin and furan congeners. Therefore, all the fish fillet data have been combined in order to develop an average concentration of TEQs in fish located in the region of pulp mills. By multiplying the levels of TEQs in 47 fish fillet composites by the number of fish in the composite, summing these values, and dividing by the total number of fish analyzed, an estimated weighted mean level of 6.5 ppt TEQs in fish downstream from pulp mills is obtained, as shown in Table 12-1. The highest levels in fish fillets reported above are about 3 times higher, i.e., about 20 ppt.

12.3 · Sources of Information on Fish Intake

In general, quantitative information on fish consumption by individuals is limited. Additionally, data on consumption by specific subgroups, such as sports fishers and subsistence fishers and their families, are virtually nonexistent. Reliable data on fish consumption have been difficult to obtain for a variety of reasons. Within the general popula-

Table 12-1. Dioxin TEQ Intake by Subsistence and Sports Fishers

Fisher	Fish intake (g/person/day)	Dioxin TEQ levels[1] (ppt)	Dioxin TEQ intakes[2] (pg/person/day)
Subsistence			
Average	69	6.5, 20	450, 1380
90th percentile	116	6.5, 20	750, 2320
Sports			
Average	13	6.5, 20	85, 260
90th percentile	39	6.5, 20	254, 780

[1] In fish fillets: first value is weighted mean level and second is highest level.

[2] First value assumes weighted mean level in fillets and second assumes highest level in fillets.

tion, finfish and shell fish are eaten sporadically and selectively. Therefore, fish intake is often not adequately reflected in established and widely used national consumption surveys which provide data for relatively short time periods, usually 3-14 days. More importantly, the commonly available nationally representative surveys contain very few persons who are representative of the at-risk subgroups, i.e., sports fishers or subsistence fishers. Furthermore, descriptions of the fish products consumed often are not sufficiently detailed to allow for estimation of fish intake on the basis of species or source. For some foods, data on national availability or production of the food can be used to derive per capita availability estimates which can be helpful in estimating intakes by individuals when specific information on consumption is unavailable. However, there are currently no reliable sources of general poundage data relative to non-commercially caught freshwater fish. Therefore, the available data have been considered, and consumption levels have been estimated using "common sense" scenarios.

Three established data bases which contain information on fish consumption were initially reviewed. These data bases were: The Fish Consumption Study conducted during 1973-74 for the Tuna Research Institute by NPD Research, Inc.; the Nationwide Food Consumption Survey (NFCS) conducted by the U.S. Department of Agriculture; and the 1977-78 Menu Census conducted by the Market Research Corporation of America (MRCA). These surveys were carried out using different methodologies and for different periods of time, but they all were based on nationally representative samples and were not targeted to certain at-risk groups, e.g., subsistence and sports fishers.

The NPD data indicate that intake of all freshwater fish species averaged 20.2 g/person/day and the 90th percentile averaged 41.7 g/person/day. The NFCS reports a mean and 90th percentile intake for finfish and shellfish combined (all age categories) of 48 and 94 g/person/day, respectively. The corresponding intakes from this survey for finfish, other than canned, dried, and raw, are 54 and 96 g/person/day. Data from the MRCA survey indicate that consumption of fish (fresh or frozen) averaged 14.3 g/person/day with a 90th percentile of 25.3 g/person/day.

The data from these surveys are reflective of the national consumption levels for primarily marine species which were commercially obtained. These data were not considered directly useful for risk estimation purposes because intakes relative to freshwater sports and subsistence fishing were lacking.

Three other sources of data on fishing practices and consumption were available: a 1982 study by Puffer et al.; a Canadian study of fishermen conducted in 1984 by the Ontario Ministry of the Environment; and a U.S. Department of Interior data base on fishing and hunting practices in the U. S. (USDOI 1988).

The first two of these sources can be characterized as relatively informal surveys, the results of which have been reported by others. The third source considered was not oriented toward food consumption. Therefore, the reliability and appropriateness of these data for QRAC purposes are not clear and the data were reviewed from this perspective.

The Puffer et al. (1982) study conducted for EPA was reportedly based on interviews with 1,059 fishers in southern California. According to the report, 50 percent of the sample fished 1-2 times per week and 14 percent reported fishing 3-7 times per week, thus suggesting that the sample may have contained a mixture of sports fishers and subsistence fishers. The self-reported intake of fish by these persons was 37 g/person/day for the median intake and 225 g/person/day for the 90th percentile of intake.

The information available to FDA from the Ontario Ministry of the Environment survey of fishers did not include information on survey methodology. The fishers reported eating locally caught fish once every 21 days, on average. The mean amount of fish consumed per eating occasion was reported to be 10 ounces, which reflects an intake of 13.8 grams per day. About one quarter of the respondents reported consuming an average of one pound of fish or more per eating occasion which results in an intake of about 21.6 g/person/day.

The U.S. Department of the Interior did not obtain consumption data in their data collection; their report indicates that U.S. sports fishers average 21 days of fishing per year. In order to use this information to estimate consumption, assumptions about the frequency of fishing and the amount of fish consumed per day of fishing are needed.

12.4 Estimation of Fish Intake by Subsistence and Sports Fishers and Their Families

12.4.1 Subsistence Fishers

Those persons turning to local waters on a regular and frequent basis as their predominant source of flesh food are expected to consume considerably more fish than individuals with a broader source of food. In the typical U.S. diet, the predominant form of flesh food is red meat as documented by several national consumption surveys. Thus, in order to estimate intake of fish among subsistence fishers and their families, a "common sense" scenario has been derived by assuming that fish intake among this group is equal to the consumption of red meat among the general population. In order to estimate intake, data on red meat consumption were obtained from the 1977-78 NRCA survey. The NRCA intake figures are based on food consumption during a 14-day survey period, and therefore are likely to provide a reasonable estimate of chronic meat intake. Mean red meat consumption for all age groups in the NRCA survey was reported

to be 69 g/person/day and the 90th percentile intake was reported to be 116 g/person/day. Chronic fish consumption by subsistence fishers and their families is estimated to be:

mean consumption	69 g/person/day
90th percentile consumption	116 g/person/day

As expected, for both mean and 90th percentile values, these figures range from 1.2 to 4.8 times greater than the amount of fish estimated to be consumed by the general population based on national surveys.

12.4.2 Sports Fishers

Fish intake by sports fishers is likely to be lower than fish intake by subsistence fishers, but the intake by sports fishers may be quite variable. Given the lack of data concerning fish consumption by sports fishers, a "common sense" scenario based on fishing practices as reported by the U.S. Department of the Interior has been developed. Their 1985 survey indicated an average of 21 days of fishing among sports fishers. It was assumed that each day of fishing would result in one eating occasion. It is likely that some fishing trips would result in no catches, while others could result in a large catch of fish which could be taken home, frozen and eaten later, if at all. But, in any case, one eating occasion per trip was considered reasonable. Therefore, frequency of eating recreationally caught fish was estimated to be 21 times per year. In order to estimate serving size, data from the U.S. Department of Agriculture's 1977-78 NFCS were used. The 90th percentile of serving size for filleted finfish was reported to be 8 ounces (227 g) and was the serving size value used in this scenario because the perishable nature of fish is likely to encourage large serving size. This serving size value is also in line with the 10-ounce mean serving size reported in the Canadian study as documented by EPA. Eight ounces of recreationally caught fish consumed 21 times per year results in an intake of 13.1 g/person/day. This value is very close to the Ontario study which reported 13.8 g/person/day. For QRAC's purposes, the value of 13 g/person/day derived from this scenario will be used. The available data did not allow a direct estimate of a 90th percentile of intake for fishers and their families who are more frequent consumers of recreationally caught fish. However, a World Health Organization document (WHO 1983) suggests that for most foods the 90th percentile level of consumption is approximately 2.5 to 3 times the value for mean consumption, and use of this factor in the absence of data has been CFSAN policy for some time. The value of 3 times the mean estimated consumption relative to intakes by sports fishers was used. About one quarter of the respondents in the Canadian study consumed at least 21.6 g/person/day which is slightly less than and consistent with the 90th percentile estimated via the CFSAN scenario. We estimate that chronic fish consumption among sports fishers is:

mean consumption	13 g/person/day
90th percentile consumption	39 g/person/day

Given the lack of available data, estimating consumption by family members of sports fishers was felt to be unjustifiable. It was assumed that lifetime consumption for family members would be no higher and most probably lower than the estimate for the sports fishers because of the possibility that at least some or all of the catch would be consumed by the fisher during the fishing expedition. Also, it should be noted that the CFSAN scenario assumes that sports fishing practices and therefore consumption are constant throughout the life span. Fishing practices may not necessarily remain constant. But, because there are no data which would allow for a reasonable adjustment of the estimate, the assumption is used.

12.5 Risk Assessment for Cancer

Levels of TEQs in fish fillets have been given earlier, i.e., an estimated weighted mean level of 6.5 ppt TEQ in fish fillets and the highest levels in fish fillets reported of about 20 ppt. The fish consumption figures for subsistence and sports fishers have been discussed above. The estimates of exposure to dioxin TEQ of subsistence and sports fishers are presented in Table 12-1.

FDA has used a level of 0.064 pg/kg/day of 2,3,7,8-TCDD as the level which will result in at most a lifetime risk of 10^{-6} of cancer. This equates to a carcinogenic unit risk of 16×10^{-6} risk for an intake of 1 pg/kg/day of 2,3,7,8-TCDD. This level is based on a linear-at-low-dose extrapolation from animal bioassays. EPA and CPSC have estimated this level to be 0.006 pg/kg/day and 0.015 pg/kg/day, respectively, based on similar modeling of animal bioassay data (see Section 3 of this report for more details). Using the dioxin TEQ intakes and fish consumption figures presented in Table 12-1, the upperbound lifetime risks to subsistence and sports fishers are presented in Table 12-2.

Interpreting these risk figures is very difficult. Risks appear to be high for subsistence fishers consuming average amounts of fish contaminated with dioxin TEQ at the weighted mean level; risks for sports fishers are somewhat lower. The risks calculated in Table 12-2 must be seen in the context of the following factors.

First, epidemiology studies have not as yet indicated increased risk of cancer for humans exposed to dioxins; such studies are equivocal at best. Second, the risks presented in Table 12-2 are upperbound risks based on exposure data which would not normally be considered by the agency as adequate for use in a quantitative risk assessment. The populations of sports fishers and subsistence fishers are poorly defined; the numbers of fish samples collected for analysis for dioxin were too small

Table 12-2. Cancer Risk for Subsistence and Sports Fishers

Fisher	Fish intake[1] (g/kg/day)	Dioxin TEQ intake[2] (pg/kg/day) mean	high	Upper bound lifetime risk[3] FDA mean	FDA high	EPA mean	EPA high	CPSC mean	CPSC high
Subsistence									
Average	1.2	7.5	23	1.2×10^{-4}	3.6×10^{-4}	1.2×10^{-3}	3.8×10^{-3}	5.0×10^{-4}	1.5×10^{-3}
90th percentile	1.9	12.5	38.7	2.0×10^{-4}	6.0×10^{-4}	2.1×10^{-3}	6.4×10^{-3}	8.3×10^{-4}	2.6×10^{-3}
Sports									
Average	0.22	1.4	4.3	2.2×10^{-5}	6.7×10^{-5}	2.3×10^{-4}	7.2×10^{-4}	9.3×10^{-5}	2.9×10^{-4}
90th percentile	0.65	4.2	13	6.5×10^{-5}	2.0×10^{-4}	7.0×10^{-4}	2.2×10^{-3}	2.8×10^{-4}	8.7×10^{-4}

[1] From Table 12-1, Subsistence (average): 69 g/person/day / 60 kg body wt. = 1.2 g/kg/day. Subsistence (90th percentile): 116 g/person/day / 60 kg body wt. = 1.9 g/kg/day. Sports (average): 13 g/person/day / 60 kg body wt. = 0.22 g/kg/day. Sports (90th percentile): 39 g/person/day / 60 kg body wt. = 0.65 g/kg/day.

[2] First value assumes a weighted mean level (mean) of 6.5 ppt in fillets and the second assumes the highest level (high) in fillets of 20 ppt.

[3] Sample calculation: [(Daily intake of 23 pg/kg/day for average subsistence fisher) ÷ (FDA daily TEQ dose equivalent to 10^{-6} cancer risk: 0.064 pg/kg/day)] x (10^{-6}) = (23/0.064) x (10^{-6}) = 3.6×10^{-4}

to represent an adequate profile of dioxin congeners in fish near pulp mills. Third, the risk calculations are based on linear-at-low-dose, no-threshold extrapolations from animal bioassay data; the risk calculations may be several orders of magnitude greater than the actual risk. FDA has no data which indicate that humans are more sensitive than animals to the effects of dioxin.

However, QRAC has frequently been required to perform quantitative risk assessments with imperfect and incomplete data. It is necessary to emphasize that the risk numbers presented in Table 12-2 are upper bound risks, and that the true carcinogenic risk to humans from dioxins and furans lies between 0 and the risk numbers presented.

12.6 Non-Cancer Toxicological Effects of Dioxins

2,3,7,8-TCDD has been shown to produce toxicological effects other than cancer, primarily in animal models. These include teratogenic, fetotoxic and reproductive effects in rodents and subhuman primates. An acceptable daily intake (ADI) of 1-10 pg/kg bw/day can be derived for the most sensitive non-cancer endpoint of 2,3,7,8-TCDD, namely reproductive toxicity. In deriving this ADI, the no-observed-effect-level reported in a three-generation rat feeding study can be used, along with an appropriate uncertainty factor, either 100 or 1,000 (Cordle 1981). Reproductive effects seen included significant decreases in fertility and neonatal survival in the f_0-generation rats receiving 0.1 ug dioxins/kg/day, and, at 0.01 ug dioxins/kg/day, a significant decrease in fertility in the f_1 and f_2 generations, decreases in litter size at birth, gestational survival, and neonatal survival and growth. Immunotoxic effects of 2,3,7,8-TCDD have also been reported at low doses (low ng amounts/kg bw/day) in animals. Other non-cancer effects that have been noted in animal models at low dose levels (ng/kg bw/day) include dermal lesions, toxic hepatitis and amyloidosis of the kidney, liver and spleen. The dose ranges of the non-cancer effects noted above appear to be higher than those for reproductive effects, and therefore, the ADI of 1-10 pg/kg bw/day developed for reproductive effects should subsume these other non-cancer endpoints. It is also important to note that this ADI range is applicable to durations of exposure of months or longer and that for shorter durations of exposure a higher ADI would be more appropriate.

There is little evidence that 2,3,7,8-TCDD causes reproductive effects in humans, other than the finding of increases in minor birth defects among Vietnam veterans exposed to Agent Orange. The epidemiologic data on the association between adverse reproductive outcomes and 2,3,7,8-TCDD exposure suffer from limitations in assessing and quantifying exposure, and in assessing the effect of multiple chemical exposures. In addition, reproductive endpoints such as early fetal loss, infertility, and minor birth defects are extremely difficult to ascertain. Given the high background rate for these events, most studies are not sensitive enough to

detect small increases. Furthermore, the vast majority of the data on reproductive events and dioxin exposure is concerned with paternal exposure only. In contrast, toxicological data provide evidence for adverse reproductive outcomes associated with maternal exposure to 2,3,7,8-TCDD.

Changes in immunological parameters in humans have been shown to occur after exposure to 2,3,7,8-TCDD, but there has been no indication that clinical disease has occurred as a result. The observed immune effects associated with short-term 2,3,7,8-TCDD exposure seem to disappear after the cessation of exposure. It is unknown how repeated doses or low doses given chronically may affect the immune system. Also, perinatal exposure or exposure during childhood may be more pronounced to the immune system, but there are problems with measuring such an effect. Immunological baseline data as well as normal response data are generally not available for children. As a result, currently no unequivocal data exist indicating that 2,3,7,8-TCDD is a human immunologic hazard.

In summary, based on laboratory studies in animals, an ADI of 1-10 pk/kg bw/day can be developed for the reproductive toxicity induced by 2,3,7,8-TCDD, which is the most sensitive non-cancer toxicological endpoint. Epidemiological studies of exposed populations suffer from a number of limitations, including a lack of sensitivity, and therefore, do not provide data that are definitive enough to assess the reproductive and immunological hazard of 2,3,7,8-TCDD.

12.7 Risk of Non-Cancer Toxicological Effects to Subsistence and Sports Fishers

QRAC has used an ADI of 1-10 pg/kg bw/day dioxin for reproductive effects of dioxin; this is the most sensitive non-cancer toxicological endpoint associated with dioxin exposure in animal studies. The dioxin TEQ exposure levels calculated in Table 12-2 for subsistence fishers, both at the average and 90th percentile levels, clearly exceed 10 pg/kw bw/day, except for the dioxin TEQ intake based on an average fish consumption level (not 90th percentile) and weighted mean dioxin fillet level. These estimates would seem to indicate that subsistence fishers who either consume fish which are caught in waters in the vicinity of pulp mills and which are contaminated with 20 ppt dioxin TEQ, or who consume greater than average amounts of these fish, are at risk for reproductive effects if humans are more sensitive than rats to reproductive effects. Various assumptions as described in this report have been made to bridge information gaps, and these assumptions generally would tend to make the risk estimates more conservative. There is little evidence, as pointed out earlier, that dioxins cause reproductive effects in humans.

Risks of reproductive effects to sports fishers are much less significant. Fish intake for sports fishers in Tables 12-1 and 12-2 was

calculated on the basis of lifetime fishing, which is fairly unlikely; therefore the risk may be overestimated.

12.8 References

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USEPA. 1989b. U.S. Environmental Protection Agency. Interim procedures for estimating risks associated with exposures to mixtures of chlorinated dibenzo-p-dioxins and dibenzofurans (CDDs and CDFs) and 1989 update. EPA 625/3-89-016.

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13. ESTIMATES OF RISKS TO TERRESTRIAL AND AVIAN WILDLIFE FROM LAND APPLICATION OF PULP AND PAPER MILL SLUDGE AND TO AQUATIC LIFE FROM DISCHARGE OF EFFLUENTS

13.1 Introduction

This chapter provides estimated exposures and risks to wildlife from land application of pulp and paper mill sludge and to aquatic life from discharge of pulp and paper mill effluents containing 2,3,7,8-TCDD and 2,3,7,8-TCDF. The information presented was compiled from:

NYDEC. 1987. New York Department of Environmental Conservation. Niagara River biota contamination project: fish flesh criteria for piscivorous wildlife. Technical report 87-3. Division of Fish and Wildlife, Bureau of Environmental Protection.

Rabert WS. 1990. An update on the environmental effects of TCDD and TCDF releases from pulp and paper mills on aquatic and terrestrial animals. U.S. EPA, Office of Toxic Substances, Health and Environmental Review Division. Memorandum to P. Jennings, EPA, Exposure Assessment Branch. June 26, 1990.

USEPA. 1990a. U.S. Environmental Protection Agency. Risk assessment for 2,3,7,8-TCDD and 2,3,7,8-TCDF contaminated receiving waters from U.S. chlorine-bleaching pulp and paper mills. Washington, DC: Office of Water Regulation and Standard, U.S. Environmental Protection Agency. August 1990.

USEPA. 1990b. U.S. Environmental Protection Agency. Assessment of risks from exposure of humans, terrestrial and avian wildlife, and aquatic life of dioxins and furans from disposal and use of sludge from bleached kraft and sulfite pulp and paper mills. Washington, DC: Office of Toxic Substances and Office of Solid Waste. EPA 560/5-90-13.

The land application of contaminated sludges to agricultural sites, mine reclamation sites, and silvicultural sites can lead to wildlife exposures to 2,3,7,8-TCDD and 2,3,7,8-TCDF. Such exposure may have adverse effects on individual organisms and may also affect the overall structure and health of the ecosystem. This analysis examines exposures and risks resulting from direct ingestion of soils and ingestion of prey items that have bioconcentrated 2,3,7,8-TCDD or 2,3,7,8-TCDF. Other routes of exposure exist (e.g., direct contact with soil). However, these two exposure pathways were chosen for assessment because of the potential for significant terrestrial wildlife exposure via these pathways, and because of the availability of data needed to perform the exposure and risk assessment. Furthermore, this assessment estimates only the effects of 2,3,7,8-TCDD and 2,3,7,8-TCDF on individual organisms and their

ability to produce viable offspring. The analysis does not attempt to predict the effects of the pollutants on whole populations of wild species of birds and mammals, or on ecosystems.

Similarly, the discharge of wastewater effluents containing 2,3,7,8-TCDD and 2,3,7,8-TCDF from pulp and paper mills can exert adverse effects on both the individual organisms downstream from the discharge point and on the overall structure and health of the ecosystem. The resident aquatic life (i.e., fish, plants, and benthic organisms) can also be exposed, in addition to those organisms feeding on aquatic life (i.e., fish-, or plant-, eating birds and mammals).

13.2 Terrestrial and Avian Wildlife Risk Assessment

13.2.1 Development of Benchmark Doses to Which Terrestrial Wildlife Exposures (Adjusted for Absorption) Are Compared

Estimated wildlife exposures (adjusted for absorption) are compared to benchmark doses that have been identified as causing adverse effects in laboratory species. When these exposure levels approach or exceed the selected benchmark, the exposed animal is at risk for experiencing adverse effects. Where possible, doses observed to cause adverse reproductive effects were selected as benchmarks; the exposure of a number of individual members of a species to a dose exceeding such a reproductive effect benchmark may lead to adverse overall population effects.

For adult birds the estimated daily dose is compared to the concentration level that had no observable adverse effects in laboratory experiments (the NOAEL). The concentration in bird eggs is compared to the lowest concentration level observed in a laboratory that caused observed adverse effects (the LOAEL). For mammals, the dose is compared to the lowest dose observed to cause reproductive effects in laboratory animals. These methods assume that the wild species are as sensitive or more sensitive to 2,3,7,8-TCDD and 2,3,7,8-TCDF than the laboratory species.

(1) NOAEL for birds. In order to compare the estimated exposure to wildlife species from the food chain to the NOAEL benchmark, the exposure is adjusted by the percent of 2,3,7,8-TCDD or 2,3,7,8-TCDF assumed to be absorbed from the diet. The NOAEL benchmark was determined from a study where Schwertz et al. (1973) administered 100 ng/kg body weight/day of 2,3,7,8-TCDD in a corn oil/acetone vehicle to 3-day old white leghorn chickens. This dose was administered for 21 days and produced no adverse effects. It is assumed that 2,3,7,8-TCDD is 100 percent absorbed from the corn oil/acetone vehicle (USFDA 1989). However, it is not expected the absorption of 2,3,7,8-TCDD from food would be the same as the absorption from a corn oil vehicle. Accordingly, the estimated dose to wildlife species from the ingestion of prey items is adjusted by the

percent of 2,3,7,8-TCDD assumed to be absorbed from the diet. Values for this percentage are found in a recent review of the literature performed by USFDA (1989). In addition, the laboratory dose must be converted to an equivalent dose over the length of time that wild species of birds are exposed to 2,3,7,8-TCDD from the sludge applied to the land-treated area. All of the migratory birds in this analysis are assumed to reside in the land-treated area for 6 months (180 days); for these birds, the NOEL is adjusted by a factor of 180/21, or about 9 (Keenan 1986). In this case, the adjusted NOAEL is 100/9 ng/kg/day, or 11 ng/kg/day. The loggerhead shrike is assumed to remain onsite for the entire year. For this non-migratory species, the NOAEL is adjusted by a factor of 365/21, or about 17. In this case, the adjusted NOAEL is 100/17 ng/kg/day, or 6 ng/kg/day. The toxicity of 2,3,7,8-TCDF is assumed to be 1/10 that for 2,3,7,8-TCDD as is assumed for toxicity to humans (see Chapter 3). Therefore, the NOAEL value used for comparison to doses of 2,3,7,8-TCDF estimated from this analysis are 10 times the NOAEL for 2,3,7,8-TCDD.

(2) LOAEL for bird eggs. Bird eggs can contain 2,3,7,8-TCDD transferred from the mother's body burden of 2,3,7,8-TCDD. Eggs are an important endpoint to consider because of their sensitivity to 2,3,7,8-TCDD. The LOAEL used for bird eggs is 65 ppt, based on a study by Sullivan et al. (1987) that found a 2-fold increase in cardiovascular malformations in chicken embryos at an estimated egg concentration of 65 pg/g (65 ppt). Although effects were found at lower concentrations of 2,3,7,8-TCDD, Sullivan et al. (1987) concluded that the evidence for effects at these lower levels was inconclusive. The 65 ppt value is used in this analysis for comparison with predicted egg concentrations for wild species. The toxicity of 2,3,7,8-TCDF is assumed to be 1/10 that for 2,3,7,8-TCDD as is assumed for toxicity to humans (see Chapter 3). Therefore, the LOAEL value used for comparison to doses of 2,3,7,8-TCDF estimated from this analysis are 10 times the LOAEL for 2,3,7,8-TCDD, or 650 ppt.

(3) LOAEL for mammals. This analysis compares exposures (adjusted for absorption) for small mammals (i.e., mammals less than 1 kg) to the lowest observed adverse reproductive effect level in laboratory rats. The LOAEL for small mammals is 10 ng/kg/day, based on a study by Murray et al. (1979) (as cited by Kociba and Schwetz (1982)) in which rats were administered 2,3,7,8-TCDD at 100, 10, or 1 ng/kg/day through the diet to study the effects on subsequent generations. This analysis compares exposure estimates to the 10 ng/kg/day level, at which Murray et al. (1979) found decreased fertility in the f_1 and f_2 generations.

For larger mammals, the expected dose for wild species is compared to the lowest dose observed to produce adverse reproductive effects in rhesus monkeys. The LOAEL for large mammals is 1.7 ng/kg, based on a study by Schnantz et al. (1982) (as cited by Kociba and Schwetz (1982)) in which rhesus monkeys given 1.7 ng/kg body weight of 2,3,7,8-TCDD in

the diet showed that four of seven pregnancies had been terminated in abortion. This value is used in this analysis for comparisons with doses received in larger wild mammals.

In both the Murray et al. (1979) and the Schnantz et al. (1982) laboratory studies, doses were administered in the diet. It is assumed that absorption from a laboratory diet is similar to the absorption from a wild diet and that these doses are directly comparable to the daily exposures in wild species from the ingestion of prey items. The toxicity of 2,3,7,8-TCDF is assumed to be 1/10 that of 2,3,7,8-TCDD as is assumed for toxicity to humans (see Chapter 3). Therefore, the LOAEL values used for comparison to doses of 2,3,7,8-TCDF ingested by large and small mammals are 10 times the LOAEL values for 2,3,7,8-TCDD.

13.2.2 Estimating Exposures to Terrestrial Wildlife

To assess the potential for wildlife exposure to sludge contaminated with 2,3,7,8-TCDD and 2,3,7,8-TCDF, this analysis adopts elements of models used by Sullivan et al. (1987) to estimate the potential 2,3,7,8-TCDD and 2,3,7,8-TCDF exposure to wild birds, and methods employed by Keenan et al. (1989) to estimate the 2,3,7,8-TCDD uptake by wild turkeys and deer. In addition, this analysis also incorporates work by Thiel et al. (1988) and the Ontario Ministry of the Environment (OME 1985) concerning the estimation of the steady-state and nonsteady-state body burden of 2,3,7,8-TCDD and 2,3,7,8-TCDF.

(1) Methodology. The calculations for estimating wildlife exposures proceed in four steps. First, the dose to an individual animal is calculated based on the contaminant concentrations in soil, the 2,3,7,8-TCDD and 2,3,7,8-TCDF uptake rates by prey items, and the amount of each prey item ingested daily. In the second step, the estimated dose is compared to the LOAEL or NOAEL. Next, the steady state body burden for the animal is calculated. For migratory species, a body burden is calculated based on the length of time spent in the treated area. Finally, egg concentrations are estimated based on the body burden of the female.

(a) Calculation for estimating daily dose. The dose of 2,3,7,8-TCDD or 2,3,7,8-TCDF to an animal$_j$ may be calculated as follows:

$$DOSE = (P_a)(TC)\ [\sum 1 \text{ to } i[(C_s)(BCF_i)(FC_{ij})] + (C_s)(FS_j)]\ Ab_g/BW_j$$

where:

Ab_g = gastrointestinal absorption rate of 2,3,7,8-TCDD or 2,3,7,8-TCDF, percent
BW_j = body weight of animal j, kilograms

C_s = concentration of 2,3,7,8-TCDD or 2,3,7,8-TCDF in soil, ng/kg
BCF_i = bioconcentration factor of food source i
FC_{ij} = fraction of animal j's diet that consists of food source i
FS_j = fraction of animal j's diet that consists of soil
P_a = percent of food originating from the land treated area
TC = total daily quantity of food consumed by the animal, kg

In this calculation, the soil concentration of 2,3,7,8-TCDD or 2,3,7,8-TCDF (C_s) is combined with bioconcentration factors (BCFs) of food items to yield the concentration of 2,3,7,8-TCDD or 2,3,7,8-TCDF in these organisms. Birds and small mammals ingest 2,3,7,8-TCDD or 2,3,7,8-TCDF when they prey on these items. Animals may also directly ingest some sludge if they graze on the application site (e.g., deer), or if they dig for prey or burrow directly on the application site. To derive the total amount of 2,3,7,8-TCDD or 2,3,7,8-TCDF ingested daily by an individual of the species, the percent of the diet represented by each contaminated food source is multiplied by the concentration of 2,3,7,8-TCDD or 2,3,7,8-TCDF in that food source and by the total daily quantity of food consumed from the contaminated site. If necessary for consistency in comparison with LOAEL and NOAEL values derived in the literature, this estimated daily intake is adjusted by the bioavailability of 2,3,7,8-TCDD and 2,3,7,8-TCDF from food items.

(b) Comparison of estimated dose to the LOAEL or NOAEL. The estimated daily dose is then compared to the selected benchmark LOAEL or NOAEL, using the following equation:

$$DOSE_{\%LOAEL} = (DOSE/LOAEL) \times 100$$

or

$$DOSE_{\%NOAEL} = (DOSE/NOAEL) \times 100$$

where:

$DOSE_{\%LOAEL}$ = dose to wild animal expressed as a fraction of the LOAEL
$DOSE_{\%NOAEL}$ = dose to wild animal expressed as a fraction of the NOAEL
DOSE = dose to the animal, ng/kg/day
NOAEL = dose at which no adverse effects were observed in laboratory species, ng/kg/day
LOAEL = lowest dose at adverse effects were observed in laboratory species, ng/kg/day

The daily dose of 2,3,7,8-TCDD or 2,3,7,8-TCDF is compared to literature values for doses that cause reproductive effects. The dose predicted for wild species is expressed as a fraction of the lowest dose observed to cause adverse effects (LOAEL), or as a fraction of the dose observed to cause no adverse effects (NOAEL).

(c) Calculation for steady state body burden. In order to determine the concentration of 2,3,7,8-TCDD or 2,3,7,8-TCDF in eggs laid by birds exposed to these contaminants, the body burden resulting from the ingestion of the daily dose must be calculated. If the bird ingests food contaminated with 2,3,7,8-TCDD and 2,3,7,8-TCDF for a sufficient length of time to achieve steady-state, then the body burden is estimated as follows:

$$B_{ss} = 1.443 \text{ (DOSE) } (T_{1/2}) / t$$

where:

B_{ss} = steady-state body burden, ng
DOSE = dose to animal, ng/day
$T_{1/2}$ = half-life of 2,3,7,8-TCDD or 2,3,7,8-TCDF, days
t = time between doses, days

The steady-state body burden is calculated as the dose multiplied by the half-life (in days) divided by the length of time between doses (in days). In this analysis, the dose is ingested daily, so t is equal to one day.

For migratory birds who arrive at the land application site only a few weeks before egg-laying, the body burden may not reach steady-state. For these birds, the body burden is calculated by the equation of Thiel et al. (1988) as follows:

$$B_{ns} = [\text{DOSE} / (0.693/T_{1/2})] [1-(1/2)^n]$$

where:

B_{ns} = nonsteady-state body burden, ng
DOSE = dose to animal, ng/day
$T_{1/2}$ = half-life of 2,3,7,8-TCDD or 2,3,7,8-TCDF
n = (time spent on treated site)/$T_{1/2}$

The larger the ratio n, the closer the body burden will be to the steady-state body burden.

(d) Calculation for estimating egg concentrations. The concentration of 2,3,7,8-TCDD or 2,3,7,8-TCDF in bird eggs is derived using the following equation:

$$C_{egg} = (B_{ns}) \text{ TC} / W_{egg}$$

or,

$$C_{egg} = (B_{ss}) \text{ TC} / W_{egg}$$

where:

B_{ss} = steady-state body burden, ng
B_{ns} = nonsteady-state body burden, ng

C_{egg} = concentration in egg, ng/kg (ppt)
TC = transfer coefficient from mother to egg, expressed as a fraction of body burden
W_{egg} = weight of the egg, kilograms

To predict the concentration of 2,3,7,8-TCDD or 2,3,7,8-TCDF in bird eggs, a transfer rate from the female to eggs is used to estimate the total quantity of 2,3,7,8-TCDD or 2,3,7,8-TCDF in the egg, in ng. The total quantity in the egg is then divided by the weight of the egg to obtain an egg concentration of 2,3,7,8-TCDD or 2,3,7,8-TCDF in ng/kg.

The estimated egg concentrations can then be compared to lowest concentrations observed to cause adverse effects in laboratory studies:

$$C_{egg\%LOAEL} = (C_{egg}/LOAEL_{egg}) \times 100$$

where:

$C_{egg\%LOAEL}$ = concentration in egg expressed as a fraction of the $LOAEL_{egg}$
C_{egg} = concentration in egg, ng/kg (ppt)
$LOAEL_{egg}$ = lowest concentration at which adverse effects were observed in eggs in laboratory, ng/kg (ppt)

The 2,3,7,8-TCDD or 2,3,7,8-TCDF concentration in the egg is compared to a selected literature value for the lowest concentration observed to produce adverse effects in embryos in laboratory studies. The concentration predicted for wild bird species is expressed as a percent of LOAEL for eggs.

(2) Data Used To Estimate Exposures to Terrestrial Wildlife. The data used for estimating wildlife exposures are summarized in Table 13-1. The following subsections describe various factors used in estimating wildlife exposure.

(a) **Selection of species examined.** To select the species of interest, this analysis relied on the expertise of biologists with the Natural Heritage Programs in the seven states of interest, namely, Georgia, Maine, Maryland, Mississippi, Ohio, Pennsylvania, and Wisconsin. In each state, the Natural Heritage experts provided a list of common avian and mammalian species as well as endangered species believed to inhabit regions of the state where land application of paper and pulp mill sludge is practiced. Data on the occurrence of mammals in each state were also obtained from Caras (1967). From these species identified, nine avian species and seven mammalian species were selected for study. They are the following: loggerhead shrike (Lanius ludovicianus), American woodcock (Scoplax rusticola), pine warbler (Dendroica pinus), eastern meadowlark (Sturnella magna), great crested flycatcher (Myiarchus crinitus), tree swallow (Iridoprocne bicolor),

Table 13-1. Assumptions and Parameter Values - Land Application: Wildlife Exposures

Input parameter	Low	Best estimate	High	Notes/explanation/references
Uptake rates for vegetation eaten by wildlife	0.01%	2%	15%	Assumes wildlife eat above-ground plants only. Low: Wipf et al. (1982) Best: Sargeant (1989) (See Appendix A.4) High: Young (1983)
BCF for earthworms	0.2	3.5	10	The best estimate was reported at a site where paper mill sludges were applied. High and low values are range reported in the literature. Martin et al. (1987)
BCF for insects	0.4	1	1.5	This is the approximate range of BCFs for insects at Elgin AFB, Fl. Low is for a composite of soil and plantborne insects, best estimate is for burrow spiders, high is for insect grubs. Young and Cockerham (1985)
BCF for small mammals	1.3	1.4	1.4	These values are for the whole body rather than just the liver. Martin et al. (1987)
BCF for fish from sediment (fish is food source for river otter)	0.0967	0.0967	5	Low and best: Nabholz (1989) High: USEPA (1988)
Duration of Food Sources from Sludge Applied Land:				
Large mammals	0.5	1.0	1.0	Large mammals feed from treated area exclusively in best and high cases. Low is same as for birds. Low river otter % calculated as size of one side of 50 acre treatment site/size of otter's home range. Chapman and Feldhamer (1982)
River otter	0.06	1.0	1.0	
Whole-body elimination rate half life, TCDD:				
Birds	15 days	21 days	31 days	Best estimate: calculated based on information provided on bluebirds in the literature. Low and high estimates are for small vertebrates. Low and High: ONE (1985) Best: Thiel et al. (1988)

Table 13-1. (continued)

Input parameter	Low	Best estimate	High	Notes/explanation/references
Small mammals	15 days	31 days	37 days	Best: data from rat oral; low from hamster oral; high from mouse intraperitoneal. OME (1985)
Large mammals	30 days	60 days	365 days	Best = 2 x data for rat oral; low = 2 x hamster oral; high = monkey. OME (1985)
Transfer of dioxin from hen to eggs: (% of body burden)	3.3%	4.8%	6.2%	Thiel et al. (1988)
Time spent on-site before egg-laying weeks	4	6-8	10	Best: 6-8 weeks depending on species, except logger-headshrike, which is not migratory in its range. Low and high are arbitrary. Bent (1955, 1962, 1963a, 1963b, 1964)
Egg mass		Species-specific		Schonwetter (1960-1983), Thiel et al. (1987), Bent (1964)
Percent of dioxin absorbed from food	0.6	0.7	.95	Low: low end of range for food given by workgroup for non-oily foods. Best: high end of range for non-oily foods. High: high end of range for absorption of oily foods. Boyer (1989) (See Appendix A.2)
Total food consumption for birds	Data from Nice (1939) as cited in Kenaga (1973)	Data from Nice (1939) as cited in Kenaga (1973)	Data from Kendeigh (1960) as cited by Kenaga (1973)	Kenaga (1973)
Total food consumption for mammals		Used data from Davis & Golly (1963) and from Wild Mammals of North America		Where specific species were not included on Davis and Golly table, the ratio of consumption to body weight for a similar animal, and then multiplied by the weight of the animal of interest. Davis and Golly (1963)
Mix of food sources		Species specific data data		Toweill and Tabor (1982) as cited in Chapman and Feldhamer (1982), Hamilton (1979), Bent (1955, 1962, 1963a, 1963b, 1964)

Table 13-1. (continued)

Input parameter	Low	Best estimate	High	Notes/explanation/references
Percent of diet that is soil:				
Eastern meadowlark	0.1%	1%	10%	Southern meadowlarks are described as "fastidious preeners" that ingest soil during preening. No range for soil ingestion given, so range here is arbitrary. Young and Cockerham (1985)
Nine-banded armadillo	1%	10%	20%	Described as often ingesting "large amounts of soil." No range of percentages were given, so range here is arbitrary. Galbreath (1982) as cited in Chapman and Feldhamer (1982)
Eastern mole	0.1%	1%	10%	Beachmouse exposed to soil through burrowing, preening. Mole may have similar exposure. Range given here is arbitrary. Young and Cockerham (1985)
Virginia opossum	1%	7%	10%	Best estimate: analysis of stomach contents for opossum in Pennsylvania revealed 7% sand and stones. Low and high arbitrary. Gardener et al. (1982) as cited in Chapman and Feldhamer (1982)
Fraction of food sources from sludge-applied land:				
Birds	0.5	1.0	1.0	Best and high assume birds feed from treated area only. Low estimate from the literature. Sullivan et al. (1987)
Small mammals	0.5	1.0	1.0	Small mammals assumed to feed completely from treated area. Low estimate same as birds. Sullivan et al. (1987)

American robin (Turdus migratorius), wood thrush (Hylocichla mustelina), and eastern bluebird (Siala sialis sialis). The mammalian species chosen were: nine-banded armadillo (Dasypus novemcinctus), least shrew (Cryptotis parva), eastern mole (Scalopus aquaticus), striped skunk (Mephitis mephitis), virginia opossum (Didelphis virginiana), river otter (Lutra canadensis), and the grey bat (Myotis grisescens). Only species judged to be at risk for exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF were selected. In general, species were selected based on their dietary habits. Those species that ingest significant quantities of prey items that bioconcentrate 2,3,7,8-TCDD and 2,3,7,8-TCDF, such as soil invertebrates, earthworms, insects and small mammals, were thought to be at most risk.

For avian species, the analysis focused on female birds for two reasons: female birds have the potential to transfer a portion of their body burden to eggs, and female birds might receive a higher dose per kilogram body weight than males during the breeding season. To the extent that males may be more sensitive than females to the adverse effects of 2,3,7,8-TCDD or 2,3,7,8-TCDF, this analysis may underestimate risk to more sensitive individuals (adult males or rapidly-growing nestlings) of these species.

Soil-dwelling organisms such as earthworms are directly exposed to contaminated soil. However, Reinecke and Nash (1984) (as cited in Eisler (1986))reported that two species of earthworms (Allolobophora caliginosa and Lumbricus rubellus) were held in soils containing 5 parts per million (ppm) of 2,3,7,8-TCDD and showed no adverse effects. Since a concentration of 5 ppm is 7300 times higher than the highest sludge concentration reported for any mill that currently land-applies sludge, it is assumed that current land application practices would have no adverse effects on the two species of earthworms tested.

Many predatory/scavenger species could also be exposed to 2,3,7,8-TCDD or 2,3,7,8-TCDF through the food chain. Because the land application of sludge is a localized practice, typically covering an areas smaller than a few hundred acres, species with large hunting territories, such as osprey, bald eagles, and herons, are unlikely to obtain a large fraction of their diets from a single sludge land application site. Thus, these types of organisms are not quantitatively evaluated here. However, it is possible that these species may accumulate significant levels of 2,3,7,8-TCDD or 2,3,7,8-TCDF because of their position at the top of the food web; for this reason, these species may warrant further analysis, especially if future land application of sludge is practiced on larger or on multiple contiguous tracts of land. The analysis does estimate exposure to one mammal species with a territory relatively large compared to the land application site: the river otter. This species, which ingests fish that bioconcentrate 2,3,7,8-TCDD and 2,3,7,8-TCDF from river sediments, is considered a threatened species in an area of one state where land application is practiced.

(b) **Soil concentrations.** This analysis uses the soil concentrations at the land application sites during a single year as the basis for the wildlife assessment. The average 2,3,7,8-TCDD and 2,3,7,8-TCDF concentrations over one year for each land application site are summarized in Table 13-2.

(c) **Body weights of animals.** In order to compare the amount ingested by a wild animal to the doses which induce effects in laboratory animals, dose must be expressed in terms of milligrams per kilogram of body weight per day. The body weights of all species are summarized in Table 13-3, along with their daily food consumption levels. The body weights of female birds were obtained from a monograph produced by the Western Bird Banding Association, which lists the average body weights of 686 species of North American birds. The body weights of all mammals except bats and shrews were obtained from Chapman and Feldhamer (1982). Body weights for bats and shrews were obtained from Hamilton (1979).

(d) **Estimating the fraction of food from treated areas.** In this analysis, the "high estimate of risk" and "best estimate of risk" scenarios assume that the species considered obtain all of their food from the treated area. This assumption is derived from the fact that home ranges of most of these species could be encompassed by the sludge treated area. Even those animals with home ranges larger than the treated area are likely to be attracted to the treated area for foraging, since the presence of sludge nutrients may increase the availability of food in the treated area compared to surrounding areas. For the "low estimate of risk," it is assumed for all species except otters that 50 percent of an animal's diet originates in the treated area, which is an assumption consistent with that used by Sullivan et al. (1987). River otters have a much larger home range than any other of the species considered in this analysis. The home range of otters varies from 7 to 15 kilometers (Toweill and Tabor 1982 as cited in Chapman and Feldhamer 1982). Assuming an average site of 20.2 acres would affect 450 meters of an adjacent river, the percent of an otter's range that would be affected would vary from 3 to 6.4 percent. Therefore, for the low estimate of risk, it is assumed that 6 percent of an otter's diet is affected by the treated area.

(e) **Estimating mixes of food sources.** Food sources are assigned to the following categories: earthworms, insects, plant matter, soil, small mammals and fish (river otters only). The food mixes for each species are summarized in Table 13-4. Data on the percent of each type of food consumed by each bird species was obtained from the series of books on the life histories of birds by Bent (1955, 1962, 1963a, 1963b, 1964). The one exception was the food consumption data for the woodcock, because woodcocks are avid consumers of earthworms. Since the bioconcentration of 2,3,7,8-TCDD and 2,3,7,8-TCDF in earthworms is higher than other food sources, this analysis estimates woodcock 2,3,7,8-TCDD

Table 13-2. Characteristics of Land Application Sites and Soil Concentrations Used in Wildlife Analysis

State	Type of application	Application rate (DMT/Ha)	Years land receives sludge	Incorporation depth (cm)	Area receiving sludge (Ha/Year)	Sludge concentration 2,3,7,8-TCDD	Sludge concentration 2,3,7,8-TCDF	Soil 2,3,7,8-TCDD conc.[a] (ppt)	Soil 2,3,7,8-TCDF conc.[a] (ppt)
Georgia	Forest	2,065	1	2.5	81	220	610	181	501
Maine	Forest	40	1	2.5	607	13	55	1	5
Maryland	Mine	224	1	0	27	80	471	80	471
Mississippi	Agriculture	58	70	15	405	681	0	14	0
Ohio	Mine	224	1	0	81	145	795	145	795
Pennsylvania	Agriculture	18	20	15	30	34	10	0.2	0.07
Wisconsin	Forest	40	1	2.5	1,012	109	1,300	9	106

[a]Soil concentrations at the land application site are based on a single year.

Table 13-3. Body Weights and Daily Food Consumption of Animals Affected by Land Application of Sludge

	Body weight, grams	Range of daily food consumption grams, dry weight		Daily food consumption grams, wet weight	Reference
		Nice (1939)	Kendeigh (1969)		
Birds					
Bluebird	31.6	4.522	8.915		
American Robin	77.3	8.84	14.73		
Great Crested Flycatcher	33.5	5.09	9.21		
Loggerhead Shrike	47.4	6.4	11.19		
Eastern Meadowlark	76.0	8.74	14.59		
Tree Swallow	20.1	3.63	6.92		
Pine Warbler	11.9	2.58	5.18		
Woodcock	219.0	--[a]	--[a]		
Wood Thrush	47.4	6.4	11.19		
Mammals					
Nine-Banded Armadillo	4,000			120	Davis and Golly (1963)
Grey Bat	8			8	Hamilton (1979)
Eastern Mole	50			25	Chapman and Feldhamer (1982)
River Otter	9,000			1,000	Chapman and Feldhamer (1982)
Virginia Opossum	3,500			105	Davis and Golly (1963)
Least Shrew	5			5.5	Davis and Golly (1963)
Striped Skunk	2,300			46	Davis and Golly (1963)

[a] Assumed only consumption of earthworms and ignored other food sources.

Table 13-4. Mix of Food Sources for Birds and Mammals

	Food type, percent					
	Earthworms	Insects	Plants	Small mammals	Soil	Fish
Birds						
Bluebird	0	68	32	0	0	0
American Robin	2	98	0	0	0	0
Great Crested Flycatcher	0	93.7	6.3	0	0	0
Loggerhead Shrike	0	72	0	28	0	0
Eastern Meadowlark	0	74	25	0	1	0
Tree Swallow	0	80	20	0	0	0
Pine Warbler	0	95	5	0	0	0
American Woodcock*	100	0	0	0	0	0
Wood Thrush	0	62	38	0	0	0
Mammals						
Nine-Banded Armadillo	0	45	0	45	10	0
Grey Bat	0	100	0	0	0	0
Eastern Mole	34	50	15	0	1	0
Virginia Opossum	8	9	22	30	7	0
Least Shrew	50	50	0	0	0	0
Striped Skunk	0	90	10	0	0	0
River Otter	0	0	0	0	0	100

* Looked only at consumption of earthworms; ignored other food sources.

and 2,3,7,8-TCDF exposure from its consumption of earthworms alone. It is assumed other food sources contribute relatively little to total woodcock 2,3,7,8-TCDD and 2,3,7,8-TCDF exposure. Sheldon (1967) reported that the total daily consumption of earthworms by woodcock is 150 grams.

Data on the mix of food sources for all mammals except shrews were obtained from Chapman and Feldhamer (1982). The data on mix of food sources for shrews were obtained from Hamilton (1979). When data on mix of food sources were reported for more than one area of the country, the data from a state where land application is practiced, or a nearby state, were used.

(f) Estimating the fraction of the diet consisting of soil. Some mammals and birds will ingest soil inadvertently while consuming ground-dwelling prey or while preening or burrowing. Young and Cockerham (1985) reported relatively higher liver concentrations of 2,3,7,8-TCDD for Southern meadowlarks (a subspecies of the eastern meadowlark, one of the bird species selected for detailed study in this analysis) residing around a 2,3,7,8-TCDD-contaminated area at Elgin AFB in Florida. Young and Cockerham (1985) hypothesized that the birds ingest some soil while preening. Based on the Young and Cockerham report, this analysis assumes that the eastern meadowlark, a relative of the Southern meadowlark, ingests a small amount of soil during preening. For this analysis, it is assumed that between 0.1 and 10 percent of the diet consists of soil, with a best estimate of 1 percent.

Young and Cockerham (1985) also postulated that beachmice living in the same area may have elevated liver concentrations of 2,3,7,8-TCDD due to their burrowing and preening behavior. Eastern moles, who are also burrowers, may have similar opportunities for inadvertent soil ingestion. For this analysis, it is assumed that from 0.1 to 10 percent of a mole's diet consists of soil ingested while foraging or burrowing, with a best estimate of 1 percent.

Galbreath (1982) (as cited in Chapman and Feldhamer 1982) reported that armadillos often ingest large amounts of soil, although the percent of the diet consisting of soil was not reported. This analysis assumes that an armadillo's diet could consist of 1 to 20 percent soil, with a best estimate of 10 percent.

Gardener (1982) (as cited in Chapman and Feldhamer 1982) reported that analysis of the stomach contents of virginia opossums found in Pennsylvania contained approximately 7 percent sand and stones. This value is used as an estimate of the percent of the opossum's diet consisting of soil, and 1 and 10 percent are used for the low and high estimates, respectively.

(g) Bioconcentration factors.

- **Earthworms.** The tendency of earthworms to bioconcentrate 2,3,7,8-TCDD has been shown in several studies. Many of the studies that yielded high bioconcentration factors were conducted at sites where soil 2,3,7,8-TCDD concentrations were quite high such as Seveso, Italy; these values were considered inappropriate for use in this analysis, where much lower soil concentrations of 2,3,7,8-TCDD are expected. Reinecke and Nash (1984) reported earthworm concentrations of 2,3,7,8-TCDD of 0.2 to 10 times higher than soil concentrations. The best estimate of the bioconcentration of 2,3,7,8-TCDD and 2,3,7,8-TCDF in earthworms, reported by Martin (1987) at an actual site where paper mill sludges containing 2,3,7,8-TCDD and 2,3,7,8-TCDF had been applied, is 3.5 times higher than the soil concentration. The low and high estimates are 0.2 and 10, respectively.

- **Insects.** Young and Cockerham (1985) reported average concentrations of 2,3,7,8-TCDD in a number of species and families of insects at a 2,3,7,8-TCDD-contaminated site at Elgin Air Force Base in Florida. Comparing the insect concentrations to the average soil concentrations reported for the same area, the bioconcentration factors for insects varies from zero for grasshoppers, to 0.4 for a composite of soil and plant-borne insects, to a high of 1.5 for insect grubs. For insects, a value of 1 is used as the best estimate for the bioconcentration factor, 0.4 and 1.5 are used for the low and high estimates, respectively.

- **Small Mammals.** Martin et al. (1987) reported that the whole body bioconcentration factor for deer mice was about 1.4, and compared this value to the whole body bioconcentration factor of 1.3 reported for field mice taken from the Seveso area. In contrast, Thalken and Young (1983) reported values for beachmice liver tissue that ranged from 6.7 for females to 18 for males. For this analysis, the whole body bioconcentration factors were judged to be more appropriate than liver-only values. Low, best, and high bioconcentration factors are 1.3, 1.4, and 1.4, respectively. It is important to note that these bioconcentration factors were derived from small mammal species that may get little 2,3,7,8-TCDD or 2,3,7,8-TCDF exposure from their diet (i.e., mice eat large quantities of seeds which would not be expected to contain significant amounts of 2,3,7,8-TCDD or 2,3,7,8-TCDF). Small mammals that consume prey that bioconcentrate 2,3,7,8-TCDD and 2,3,7,8-TCDF may have higher levels of 2,3,7,8-TCDD and 2,3,7,8-TCDF in their bodies.

- **Fish.** The fish to sediment ratio used in both the low estimate of risk and best estimate of risk scenarios to estimate the concentration of 2,3,7,8-TCDD in fish consumed by river otters is assumed to be 0.0967 (whole body, wet weight), as derived from a

recent EPA literature review (USEPA 1989b). In the high estimate of risk scenario, the fish to sediment ratio, 5, is obtained from another EPA review of literature (USEPA 1988). Otters are assumed to eat the entire body of the fish. For simplicity, it is assumed that the sediment 2,3,7,8-TCDD and 2,3,7,8-TCDF concentrations near all land application sites are, on average, 1/1000 the 2,3,7,8-TCDD and 2,3,7,8-TCDF concentrations in the soil at the land application site. This value is based on the average sediment to soil ratios calculated in this report for the seven land application sites.

(h) Plant uptake rates. The low, best, and high estimates for the uptake of 2,3,7,8-TCDD or 2,3,7,8-TCDF from soil into plant tissues is 0.01, 2, and 15 percent. The low estimate for the uptake of 2,3,7,8-TCDD or 2,3,7,8-TCDF from soil into plant tissues (0.01 percent) is derived from Wipf (1982). The high estimate of the plant uptake rate is taken from a study by Young (1983). For the best estimate of plant uptake, this analysis uses the value suggested by USEPA (1989a) (see Appendix A) for above-ground plants. Since all of these values were derived from studies on cultivated plants, the use of these values in this analysis is based on the assumption that wild plants take up 2,3,7,8-TCDD and 2,3,7,8-TCDF at the same rate as cultivated crops. Furthermore, the use of this range of estimates assumes that wild animals consume only above-ground crops.

(i) Total food consumption per day. A summary of the data used for total daily food consumption estimates is presented in Table 13-3. The total consumption values for the armadillo, opossum and striped skunk were estimated by applying the food intake/body weight ratio for raccoons (Chapman and Feldhamer 1982) to the body weights of these animals. The total food consumption data for other species were taken directly from the literature, with otters and moles from Chapman and Feldhamer (1982), bats from Hamilton (1979), and least shrew from Davis and Golly (1963).

Data from Nice (1939) and Kendeigh (1969) were used by Kenaga (1973) to predict a regression equation relating the log of body weight to the log of the ratio of food consumption to body weight. The body weights for birds from Dunning (1984) were used in these equations to predict the total daily food consumption, in dry weight. The Nice (1939) data are used for the best estimate, and the Kendeigh (1969) data are used as the high estimate. To use the values derived, dry weight values must be converted to wet weight values. To convert the dry weight values to wet weight, earthworms are assumed to be 83 percent water by weight (French et al. 1957 as cited by Kenaga 1973). No data on the wet weight of other food sources were found; therefore, the percent water of other food sources is arbitrarily assumed to be 50 percent.

Sheldon (1967) reported that the American woodcock consumes 150 grams per day of earthworms. Since earthworms bioconcentrate 2,3,7,8-TCDD and 2,3,7,8-TCDF at a higher rate than any other food source, the exposure

estimate of woodcocks to 2,3,7,8-TCDD and 2,3,7,8-TCDF is based on its consumption of earthworms alone; other food sources are assumed to contribute relatively little to the total dose.

(j) Absorption of 2,3,7,8-TCDD or 2,3,7,8-TCDF from gastrointestinal tract. The absorption of 2,3,7,8-TCDD and 2,3,7,8-TCDF from food sources is needed in order to compare the dose ingested by wild birds to results from laboratory studies where 2,3,7,8-TCDD was delivered to chickens in a corn oil matrix. USFDA (1989) recently reviewed studies on the bioavailability of TCDD ingested in a variety of matrices, and concluded that 60 to 70 percent of 2,3,7,8-TCDD is absorbed by the gastrointestinal tract from non-oily foods, while 85 to 95 percent is absorbed by the gastrointestinal tract from oily foods. For the best estimate, it is assumed that absorption of 2,3,7,8-TCDD and 2,3,7,8-TCDF from all food sources in a wild animal's diet is 70 percent, while values of 60 percent and 95 percent are used to represent the low and high estimates, respectively.

For mammals, no adjustment is necessary, because, in the laboratory studies to which wildlife exposure estimates are compared, researchers administered 2,3,7,8-TCDD to the animals through the diet. This analysis assumes that the 2,3,7,8-TCDD and 2,3,7,8-TCDF absorption rate from the wild species diets is the same as the 2,3,7,8-TCDD and 2,3,7,8-TCDF absorption rate from the laboratory diet.

(k) Estimating body burdens of 2,3,7,8-TCDD and 2,3,7,8-TCDF. Egg concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF are a function of the body burden of 2,3,7,8-TCDD and 2,3,7,8-TCDF in the female laying the eggs. In order to calculate the transfer of 2,3,7,8-TCDD and 2,3,7,8-TCDF from the female bird to her offspring, the body burden of the female bird must be calculated. The equation for calculating steady-state body burden is derived in the Ontario Ministry of the Environment Scientific Criteria Document for PCDDs and PCDFs (OME 1985). However, not all organisms will be exposed to the contaminated area long enough to reach steady state. In fact, of all bird species analyzed here, only the loggerhead shrike is considered nonmigratory. For migratory organisms, a pharmacokinetic model described in Thiel et al. (1988) is used to predict the body burden at the time of reproduction. One input to this model is the length of time exposed to 2,3,7,8-TCDD and 2,3,7,8-TCDF before reproduction. For a migratory bird, it is assumed that the exposure begins upon arrival in the northern portion of the range and continues until the time when the eggs are laid. For the bird species analyzed, this is approximately 6-8 weeks, depending on the species. This value was obtained from calculating the number of weeks between the first spring sightings of the birds in the states where land application is practiced and reported egg-laying dates in these states (Bent 1955, 1962, 1963a, 1963b, 1964).

Another input required for the pharmacokinetic model is the half-life of 2,3,7,8-TCDD and 2,3,7,8-TCDF in wild birds. OME (1985) reported the half-life for whole body elimination of 2,3,7,8-TCDD and 2,3,7,8-TCDF in several species, including rats, mice, hamsters, and monkeys. No data were found for wild birds. However, from an analysis by Thiel et al. (1988), an estimate of the half-life of 2,3,7,8-TCDD in birds was indirectly estimated. Because the 2,3,7,8-TCDD soil concentrations were not reported by Thiel et al. (1988), the average soil concentrations over one year were derived using the sludge concentrations of 2,3,7,8-TCDD applied to the treated area, assuming a 2,3,7,8-TCDD half-life in soil of 10 years, and assuming that the sludge was incorporated with 1 inch of forest floor litter. Information on the dietary habits of bluebirds from Bent (1964) and an estimate of the bluebird's total consumption of food per day from Kenaga (1973) were used to estimate the daily dose of 2,3,7,8-TCDD to the bluebird at the site. In addition, information found in Bent (1964) on the time of arrival and egg dates in Wisconsin, the state where the Thiel et al. (1988) study was conducted, was used to estimate the length of time bluebirds were residing in the treated area before reproducing. Finally, the average weights of bluebirds eggs and the percent of 2,3,7,8-TCDD transferred from the bluebird hen to her eggs was obtained from Thiel et al. (1988). All of the information was entered into the pharmacokinetic model, and the value for the half-life of 2,3,7,8-TCDD was adjusted until the model yielded values that corresponded to the actual 2,3,7,8-TCDD concentration in bluebird eggs reported in Thiel et al. (1988). The value estimated for half-life is 21 days. This value is in good agreement with values reported in OME (1985) for other small vertebrates, such as rats and mice, which range from 17 to 31 days, and is used as the best estimate. For a low and high estimate of the half-life of 2,3,7,8-TCDD in wild birds, data from OME (1985) are used, which reports a range of 2,3,7,8-TCDD half-lives for small vertebrates from 17 to 31 days. Martin et al. (1987) stated that the half-life for the whole body elimination of 2,3,7,8-TCDF is one-eighth the half-life of 2,3,7,8-TCDD. Under this assumption, the estimated half-life for 2,3,7,8-TCDF in wild birds is 2.6 days, and ranges from 2.1 to 3.9 days.

For the estimate of half-life of 2,3,7,8-TCDD in mammals, data reported in OME (1985) are used. For small mammals, a value of 31 days represents the best estimate. This value was observed in rats administered 2,3,7,8-TCDD orally. The low estimate is 15 days, the value observed for hamsters administered 2,3,7,8-TCDD orally, while the high estimate 37 days, the value observed in mice administered with 2,3,7,8-TCDD i.p. For larger mammals (i.e., over 1 kg), the best estimate for half-life of 2,3,7,8-TCDD is 60 days. This value is about twice the half-life observed in rats given 2,3,7,8-TCDD orally. The low estimate is 30 days, twice the hamster value, while the high estimate of 365 days is the half-life of 2,3,7,8-TCDD observed in monkeys. Again, it is assumed that the half-life for the whole body elimination of

2,3,7,8-TCDF is one-eighth the half-life of 2,3,7,8-TCDD (Martin et al. 1987). Under this assumption, the half-life of 2,3,7,8-TCDF in small mammals is 3.9 days (with low and high estimates of 1.9 days and 4.6 days, respectively). The half-life for 2,3,7,8-TCDF in large mammals is 7.5 days (ranging from 3.8 days to 45.6 days).

(l) **Transfer coefficient from the hen to eggs.** The transfer coefficient of 2,3,7,8-TCDD from the hen to the egg was estimated by Thiel et al. (1988) by measuring the 2,3,7,8-TCDD concentration in the body of the bluebird and the concentration in the eggs. Thiel et al. (1988) reported that a mean of 4.8 percent of the female's body burden of 2,3,7,8-TCDD was transferred to eggs, with a range from 3.3. to 6.2 percent. A value of 4.8 percent was derived as the best estimate of transfer rate for 2,3,7,8-TCDD and 2,3,7,8-TCDF, while values of 3.3 percent and 6.2 percent are used for the low and high estimates, respectively.

(m) **Egg weights of birds.** The quantity of 2,3,7,8-TCDD and 2,3,7,8-TCDF transferred from the female bird to the eggs must be divided by the weight of the egg to obtain an estimated egg concentration. Egg weights were reported for bluebirds and tree swallows in Thiel et al. (1988) and for eastern meadowlarks in Bent (1964). All other egg weights were obtained from Schonwetter (1960-1984).

13.2.3 Summary of Results: Terrestrial Wildlife.

The risks to terrestrial wildlife from land application of pulp and paper sludge are presented as best estimates of risk, as well as low and high estimates. For adult birds and bird eggs, these three sets of risk estimates are presented in Table 13-5 and for mammals the estimates are presented in Table 13-6. Table 13-7 presents a summary of the results of the best estimate of risk analysis for birds foraging from land application sites. This table shows the highest estimates of the daily dose (expressed as a percent of the NOAEL) among the seven land application sites assessed in this analysis. The table also indicates the states where the highest values occur. Similarly, Table 13-8 summarizes the risks to bird eggs, while Table 13-9 presents the risks to mammalian species.

The results confirm that those species whose diets consist largely of prey species that bioconcentrate 2,3,7,8-TCDD and 2,3,7,8-TCDF are at the greatest risk from the land application of sludges containing 2,3,7,8-TCDD and 2,3,7,8-TCDF. For example, at all seven sites assessed, the avian species at greatest risk is the American woodcock, a species that consumes relatively large quantities of earthworms. At the land application site with the highest estimated soil concentrations of 2,3,7,8-TCDD, the best estimate of the daily dose of 2,3,7,8-TCDD ingested by this species is 27.6 times the estimated NOAEL; the daily dose of ingested 2,3,7,8-TCDF is 7.6 times the estimated NOAEL. The eggs

Table 13-5. Estimates of Risks to Adult and Hatching Birds from Exposure to 2,3,7,8-TCDD as a Result of Land Application of Pulp and Paper Mill Sludge

Species/state	Sludge conc. (ppt)	Exposure conc. (ppt)	Adult risks[a] (times NOAEL) Low	Best	High	Embryo risks[b] (times LOAEL) Low	Best	High
American Robin								
Georgia	220	181	0.67	4.13	6.57	1.36	10.59	30.16
Maine	13	1	0	0.02	0.04	0.01	0.06	0.17
Maryland	80	80	0.30	1.83	2.90	0.60	4.68	13.33
Mississippi	681	14	0.05	0.33	0.52	0.11	0.84	2.38
Ohio	145	145	0.53	3.31	5.26	1.09	8.49	24.16
Pennsylvania	34	0.2	0	0.01	0.01	0	0.01	0.04
Wisconsin	109	9	0.03	0.21	0.33	0.07	0.53	1.50
Woodcock								
Georgia	220	181	0.68	27.61	78.89	1.36	62.37	358.32
Maine	13	1	0	0.15	0.44	0.01	0.34	1.98
Maryland	80	80	0.30	12.20	34.87	0.60	27.57	158.37
Wisconsin	109	9	0.03	1.37	3.92	0.07	3.10	17.82
Eastern Bluebird								
Maine	13	1	0	0.02	0.03	0	0.05	0.13
Maryland	80	80	0.29	1.70	2.61	0.27	3.72	10.19
Ohio	145	145	0.53	3.09	4.72	0.49	6.75	18.47
Pennsylvania	34	0.2	0	0	0.01	0	0.01	0.03
Wisconsin	109	9	0.03	0.19	0.29	0.03	0.42	1.15
Great Crested Flycatcher								
Georgia	220	181	0.56	3.28	4.93	1.36	6.24	16.80
Maryland	80	80	0.25	1.45	2.18	0.38	2.76	7.43
Mississippi	681	14	0.04	0.26	0.39	0.07	0.49	1.33
Ohio	145	145	0.45	2.63	3.95	0.68	5.00	13.46
Pennsylvania	34	0.2	0	0	0.01	0	0.01	0.02
Loggerhead Shrike[c]								
Maryland	80	80	0.70	2.87	3.78	0.90	3.97	7.88
Mississippi	681	14	0.13	0.51	0.68	0.16	0.71	1.41
Ohio	145	145	1.28	5.20	6.85	1.63	7.20	14.29
Pennsylvania	34	0.2	0	0.01	0.01	0	0.01	0.02
Eastern Meadowlark								
Maryland	80	80	0.15	0.88	1.33	0.29	2.11	5.71
Mississippi	681	14	0.03	0.16	0.24	0.05	0.38	1.02
Ohio	145	145	0.28	1.60	2.42	0.53	3.82	10.34
Pennsylvania	34	0.2	0	0	0	0	0.01	0.02

Table 13-5. (Continued)

Species/state	Sludge conc. (ppt)	Exposure conc. (ppt)	Adult risks[a] (times NOAEL) Low	Best	High	Embryo risks[b] (times LOAEL) Low	Best	High
Tree Swallow								
Georgia	220	181	0.57	3.34	5.05	1.12	8.18	22.19
Maine	13	1	0	0.02	0.03	0.01	0.05	0.12
Maryland	80	80	0.25	1.47	2.23	0.49	3.62	9.81
Ohio	145	145	0.46	2.67	4.05	0.89	6.55	17.77
Wisconsin	109	9	0.03	0.17	0.25	0.06	0.41	1.10
Pine Warbler								
Georgia	220	181	0.82	4.76	7.15	0.92	6.79	18.26
Maine	13	1	0	0.03	0.04	0.01	0.04	0.10
Maryland	80	80	0.36	2.10	3.16	0.41	3.00	8.07
Ohio	145	145	0.65	3.81	5.73	0.74	5.44	14.63
Wisconsin	109	9	0.04	0.24	0.36	0.05	0.34	0.91
Wood Thrush								
Georgia	220	181	0.33	1.94	2.98	0.55	4.05	11.15
Maine	13	1	0	0.01	0.02	0	0.02	0.06
Maryland	80	80	0.15	0.86	1.32	0.24	1.79	4.93
Ohio	145	145	0.27	1.55	2.39	0.44	3.25	8.93
Wisconsin	109	9	0.02	0.10	0.15	0.03	0.20	0.55

a These factors represent the comparison factor between the estimated exposure to birds and the NOAEL for 2,3,7,8-TCDD of 11 ppt for migratory birds and 6 ppt for nonmigratory birds.

b These factors represent the comparison facator between the estimated exposure to bird eggs and the LOAEL for bird eggs for 2,3,7,8-TCDD of 65 ppt.

c The Loggerhead Shrike is considered to be a threatened species in the State of Maryland.

Table 13-6. Estimates of Risks to Mammals from Exposure to 2,3,7,8-TCDD as a Result of Land Application of Pulp and Paper Mill Sludge

Species/state	Sludge conc. (ppt)	Soil conc. (ppt)	Adult risks[a] (times LOAEL) Low	Best	High
Least Shrew					
Georgia	220	181	2.99	44.80	114.48
Maine	13	1	0.02	0.25	0.63
Maryland	80	80	1.32	19.80	50.60
Mississippi	681	14	0.24	3.54	9.04
Ohio	145	145	2.39	35.89	91.71
Pennsylvania	34	0.2	0	0.06	0.14
Wisconsin	109	9	0.15	2.23	5.69
Grey Bat[b]					
Georgia	220	181	3.62	18.10	27.15
Maine	13	1	0.02	0.10	0.15
Maryland	80	80	1.60	8.00	12.00
Mississippi	681	14	0.29	1.43	2.15
Ohio	145	145	2.90	14.50	21.75
Pennsylvania	34	0.2	0	0.02	0.03
Wisconsin	109	9	0.18	0.90	1.35
Eastern Mole					
Georgia	220	181	1.22	15.40	38.50
Maine	13	1	0.01	0.09	0.21
Maryland	80	80	0.54	6.81	17.02
Mississippi	681	14	0.10	1.22	3.04
Ohio	145	145	0.98	12.34	30.85
Pennsylvania	34	0.2	0	0.02	0.05
Wisconsin	109	9	0.06	0.77	1.91
Virginia Opossum					
Georgia	220	181	0.72	2.88	4.69
Maine	13	1	0	0.02	0.03
Maryland	80	80	0.32	1.27	2.07
Mississippi	681	14	0.06	0.23	0.37
Ohio	145	145	0.58	2.31	3.76
Pennsylvania	34	0.2	0	0	0.01
Wisconsin	109	9	0.04	0.14	0.23
Striped Skunk					
Georgia	220	181	0.38	1.92	2.89
Maine	13	1	0	0.01	0.02
Maryland	80	80	0.17	0.85	1.28
Mississippi	681	14	0.03	0.15	0.23
Ohio	145	145	0.31	1.54	2.32
Pennsylvania	34	0.2	0	0	0
Wisconsin	109	9	0.02	0.10	0.14

Table 13-6. (Continued)

Species/state	Sludge conc. (ppt)	Soil conc. (ppt)	Adult risks[a] (times LOAEL) Low	Best	High
Nine-Banded Armadillo					
Mississippi	681	14	0.11	0.31	0.37
River Otter					
Georgia	220	181	0	0	0.06
Maine	13	1	0	0	0
Maryland	80	80	0	0	0.03
Mississippi	681	14	0	0	0
Ohio	145	145	0	0	0.05
Pennsylvania	34	0.2	0	0	0
Wisconsin	109	9	0	0	0

[a] These factors represent the comparison factor between the estimated exposure to mammals and the LOAEL for 2,3,7,8-TCDD of 10 ppt for small mammals and the LOAEL of 1.7 ppt for large mammals. For example, the best estimate of exposure of the Least Shrew to 2,3,7,8-TCDD from sludge applied to land in Georgia is 44.8 times greater than the LOAEL for small mammals (i.e., 10 ppt).

[b] The Grey Bat is considered to be an endangered species in the State of Georgia.

Table 13-7. Summary of Risks to Birds ("Best Estimate")

Species	Comparison of Estimated Dose to NOAEL[a]		
	2,3,7,8-TCDD high dose factor	2,3,7,8-TCDF high dose factor	State where highest value occurs
Eastern Bluebird	3.1	1.7	OH
American Robin	4.1	1.1	GA
Great Crested Flycatcher	3.3	0.9	GA
Loggerhead Shrike	5.2	2.9	OH
Eastern Meadowlark	1.6	0.9	OH
Tree Swallow	3.3	0.9	GA
Pine Warbler	4.8	1.3	GA
Woodcock	27.6	7.6	GA
Wood Thrush	1.9	0.5	GA

[a] These factors represent the comparison factor between the estimated dose to birds and the NOAEL value for 2,3,7,8-TCDD of 11 ppt for migratory birds and 6 ppt for non-migratory birds and the NOAEL value for 2,3,7,8-TCDF of 110 ppt for migratory birds and 60 ppt for non-migratory birds. For example, the estimated exposure level of 2,3,7,8-TCDD in the state of Georgia for the woodcock is 27.6 times greater than the NOAEL for birds.

Table 13-8. Summary of Risks to Bird Eggs ("Best Estimate")

Species	Comparison of Estimated Dose to LOAEL[a]		
	2,3,7,8-TCDD high dose factor	2,3,7,8-TCDF high dose factor	State where highest value occurs
Eastern Bluebird	6.8	0.6	OH
American Robin	10.6	0.4	GA
Great Crested Flycatcher	6.2	0.3	GA
Loggerhead Shrike	7.2	0.5	OH
Eastern Meadowlark	3.8	0.3	OH
Tree Swallow	8.2	0.3	GA
Pine Warbler	6.8	0.3	GA
Woodcock	62.4	2.9	GA
Wood Thrush	4.1	0.2	GA

[a] These factors represent the comparison factor between the estimated dose to bird eggs and the LOAEL value for bird eggs of 65 ppt for 2,3,7,8-TCDD and 650 ppt for 2,3,7,8-TCDF.

Table 13-9. Summary of Risks to Mammals ("Best Estimate")

Species	Comparison of Estimated Dose to LOAEL[a] 2,3,7,8-TCDD high dose factor	2,3,7,8-TCDF high dose factor	State where highest value occurs
Nine-Banded Armadillo[b]	0.3	0	MS
Grey Bat	18.1	5.0	GA
Eastern Mole	15.4	4.3	GA
River Otter	c	c	c
Virginia Opossum	2.9	0.8	GA
Least Shrew	44.8	12.4	GA
Striped Skunk	1.9	0.5	GA

[a]These factors represent the comparison factor between the estimated dose to mammals and the LOAEL value for 2,3,7,8-TCDD of 10 ppt for small mammals and 1.7 ppt for large mammals and the LOAEL value for 2,3,7,8-TCDF of 100 ppt for small mammals and 17 ppt for large mammals.
[b]Armadillo only occurs in Mississippi.
[c]River otter dose less than 0.005 in all states.

of woodcocks residing on this site are estimated to have 2,3,7,8-TCDD concentrations 62.4 times higher than the 2,3,7,8-TCDD LOAEL of 65 ppt and a 2,3,7,8-TCDF concentration that is 2.9 times higher than the 2,3,7,8-TCDF LOAEL of 650 ppt.

Similarly, the mammalian species at greatest risk from land application of 2,3,7,8-TCDD- and 2,3,7,8-TCDF-contaminated sludge is the least shrew. Fifty percent of this species' diet consists of earthworms. This species also consumes large quantities of food relative to its body weight, leading to a greater dose per body weight than other species. At the site with the highest 2,3,7,8-TCDD concentrations, the estimated daily dose of 2,3,7,8-TCDD ingested by the shrew is 44.8 times higher than the 2,3,7,8-TCDD LOAEL for small mammals.

The wildlife risk assessment results also show that species whose diets consist of only moderate amounts of prey species that bioconcentrate 2,3,7,8-TCDD and 2,3,7,8-TCDF may exceed toxicity thresholds if the concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF are sufficiently high. For example, at the site with the highest 2,3,7,8-TCDD and 2,3,7,8-TCDF concentrations, all avian species assumed to reside there exceed the 2,3,7,8-TCDD NOAEL for adult birds. The eggs of these species are estimated to have concentrations of 2,3,7,8-TCDD that exceed the 2,3,7,8-TCDD LOAEL for eggs. Furthermore, all of the mammals at this site, except the otter and armadillo, also exceed the 2,3,7,8-TCDD LOAEL.

The wildlife risk assessment results imply that individual members of certain wildlife species are at risk for reproductive and other effects from the land application of pulp and paper mill sludges containing 2,3,7,8-TCDD and 2,3,7,8-TCDF. This result assumes that wild species are at least as sensitive to the effects of 2,3,7,8-TCDD and 2,3,7,8-TCDF as laboratory species. Adverse effects on individuals may be important if the individuals affected are members of species that are endangered or threatened. Table 13-10 presents results of the preliminary search for endangered and threatened species found in seven counties where the eight pulp and paper mills are located that apply pulp and paper mill sludge to land.

This assessment does not attempt to quantify the effects of 2,3,7,8-TCDD and 2,3,7,8-TCDF on populations or ecosystems. However, the results of assessment show that at certain land application sites, the reproductive capability of individuals of certain terrestrial species may be affected, assuming that wild species are at least as sensitive to the effects of 2,3,7,8-TCDD and 2,3,7,8-TCDF as laboratory species. Effects on the reproductive capability of a sufficient number of individual members of a species may lead to overall population effects for that species in that area.

13.3 Aquatic Life Risk Assessment

13.3.1 Risks to Fish

Currently, sufficient data are not available concerning the chronic effect of 2,3,7,8-TCDD and 2,3,7,8-TCDF on aquatic life to derive

Table 13-10. Results of Preliminary Search[a] for Endangered (E) and Threatened (T) Species Found in the Counties[b] Where Pulp and Paper Mills Are Located that Apply Dioxin- and Furan-Contaminated Pulp and Paper Mill Sludge to Land

Endangered and threatened species	States with soil application						
	GA	ME	MD	MS	OH	PA	WI
Mammals							
Indiana bat (E)			P		P	P	
West Indian Manatee (E)	K						
Birds							
Bald Eagle (E)	K	K					K
Piping Plover (ED)	K	K					
Wood Stork (E)	K						
Red-Cockaded Woodpecker (E)				K			
Reptiles							
Eastern Indigo Snake (T)				P			
Gopher Tortoise (T)				K			
Kemp's Ridley Sea Turtle (E)	K						
Leatherback Sea Turtle (E)	P						
Loggerhead Sea Turtle (T)	K						
Fish							
Shortnose Sturgeon		K					
Invertebrates							
Iowa Pleistocene Snail (terrestial) (E)							P
Plants							
Harperalla (E)			K				
Small Whorled Pogonia (E)		K					

[a] Based on information dated October 26, 1989.

[b] Pulp mill sites:
- Camden County, Georgia
- Cumberland County, Maine
- Alleghany County, Maryland
- Perry County, Mississippi
- Ross County, Ohio
- Wyoming County, Pennsylvania
- Wood County, Wisconsin

P - Possibly present in county
K - Known to be present in county

national water quality or sediment criteria for these contaminants. However, several studies have been conducted that provide some information concerning the long-term effects of 2,3,7,8-TCDD and 2,3,7,8-TCDF on fish, that is, from subacute exposures longer than short-term acute tests, but not full-life cycle chronic tests. EPA/OTS has developed estimated chronic toxicity values for 2,3,7,8-TCDD and 2,3,7,8-TCDF based on these existing exposure studies. Potential impacts on fish were determined by comparing estimated in-stream concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF to these estimated chronic toxicity values: 0.038 pg/l for 2,3,7,8-TCDD and 0.41 pg/l for 2,3,7,8-TCDF. Site-specific water column contaminant concentrations were calculated by USEPA (1990a) using the simple dilution exposure assessment approach and low (7Q10) receiving stream flow conditions.

Water column concentrations of 2,3,7,8-TCDD immediately downstream from 80 out of 90 mills evaluated (89 percent) were estimated to exceed the estimated chronic toxicity value of 0.038 pg/l. Seventy-four mills (82 percent) exceeded the estimated chronic toxicity value of 0.41 pg/l for 2,3,7,8-TCDF.

The 7Q10 is used as a design flow for stressed aquatic systems; however, use of 7Q10 receiving water flow rates does not necessarily result in the extreme worst-case scenario for aquatic life impacts. 7Q10 is defined as the lowest consecutive seven-day average flow over a 10-year period. Streamflows less than or equal to the 7Q10 flow (expressed as a daily flow) can occur multiple times within a given year for periods of 1 to several days. It is possible that even brief exposures (i.e., less than seven days) to high concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF can result in toxic effects to aquatic organisms such as migrating fish, and such effects may occur after an appreciable delay following only brief exposures.

Taking into account the above assumptions, simplifications, and limitations concerning the risks to fish from effluent discharges of 2,3,7,8-TCDD and 2,3,7,8-TCDF, the results of this assessment indicate that the levels of 2,3,7,8-TCDD and 2,3,7,8-TCDF contamination in the water column resulting from surface water effluent discharges from many chlorine-bleaching pulp and paper mills could be exerting significant adverse effects on fish.

13.3.2 Risks to Aquatic Plants and Herbivores

Physical properties of TCDD and TCDF, such as low water solubility and high log P, are associated with chemicals which are expected to sorb to organic material. The sorption of 2,3,7,8-TCDD and 2,3,7,8-TCDF to suspended matter, plants, and sedments in an aquatic environment may pose a potential risk to plants, herbivores and benthic organisms. No toxic effects on plants were identified for 2,3,7,8-TCDD. While active uptake via systemic root absorption is not expected to yield much 2,3,7,8-TCDD

in plants, 2,3,7,8-TCDD is likely to sorb to organic surfaces of plants immediately downstream of pulp mills. The amounts of 2,3,7,8-TCDD and 2,3,7,8-TCDF that would adsorb to plants has not been estimated, nor have samples been taken for measurement.

The question of risks to herbivores, however, is not unimportant. Dermal exposure to dioxins and consumption of 2,3,7,8-TCDD-contaminated plants both represent likely routes of exposure which might produce lethal or sublethal effects. If exposure levels in plants and dermal uptake can be measured or estimated, potential adverse effects on aquatic herbivores should be considered further.

13.3.3 Risks to Benthic Organisms

Sediments downstream of effluent discharges from pulp mills are predicted to be contaminated by dioxins and furans. If the half-life in sediments are similar to the 10 to 15 years in soils for 2,3,7,8-TCDD, the dioxin- and furan-contaminated sediments may pose a problem for many years. The extent of the contaminated sediments produced from pulp mill discharges were not determined by monitoring or modelling. At the predicted soil degradation rates, in ten years 2,3,7,8-TCDD levels in sediments would be more than 7 times higher than the annual deposition levels. Risks to benthic species cannot be determined at this time, because sediment toxicity data on 2,3,7,8-TCDD are not available for benthic species. Also, concentrations of 2,3,7,8-TCDD and 2,3,7,8-TCDF in sediments are not available for the 104 pulp mills.

However, 2,3,7,8-TCDD concentrations have been measured in the sediments in the Fox River, Wisconsin; the mean 2,3,7,8-TCDD level for 13 sites was 3.1 pg/g (ppt) and ranged from undetected at 1.4 pg/g to 7.4 pg/g (Ankley et al., in press). The 2,3,7,8-TCDF levels at the same sites average 7.6 times higher than 2,3,7,8-TCDD and ranged from 0.1 to 61.1 pg/g. The highest 2,3,7,8-TCDD and 2,3,7,8-TCDF concentrations were found at the site of a pulp mill. The numbers of benthic fauna were significantly reduced at the pulp mill site compared to the control site. The average number of chironomids and oligochaetes at the pulp mill site were significantly lower than the controls. Numbers of organisms in both taxa were less than 1 percent of the control levels. The numbers of benthic fauna were also significantly reduced at all 9 sites downstream of the pulp mill. Other pollutants measured at the sites included penta-, hexa-, hepta-, and octa-chlorinated forms of dioxin and furans.

The contaminated sediments can also be expected to provide a reservoir from which dioxins and furans will enter the food web and be bioaccumulated by benthic organisms and bottom feeders, such as carp, catfish, and other edible fish species. Dioxins and furans are expected to be transferred from one trophic level to another. Toxic effects might be anticipated in those 2,3,7,8-TCDD-sensitive species throughout the

food web, especially in the top carnivores such as salmon, bass, pike, greyling, and fish-eating birds and mammals, including humans.

13.3.4 Risks to Fish-Eating Birds and Mammals

The New York Department of Environmental Conservation (NYDEC 1987) reviewed non-cancer toxicity data for dioxins for piscivorous mammals and birds and arrived at a dietary criterion for consumption of fish. They determined that wildlife feeding primarily on fish with 2,3,7,8-TCDD body burden concentrations of greater than 3 ppt were at risk. Comparison of the 3 ppt toxicity value with measured 2,3,7,8-TCDD concentrations in whole fish (Table 13-11) sampled near pulp mills in the National Bioaccumulation Study indicates that 66 percent (57/86) of the fish samples exceeded that threshold toxicity value. Over 38 percent of the fish sampled (33/86) contained twice the 2,3,7,8-TCDD level compared with the criterion. Distribution of whole fish contaminated with more than 3 ppt 2,3,7,8-TCDD include 21 states from Florida to Minnesota and from Maine to California. The fish with low 2,3,7,8-TCDD levels (i.e., less than 3 ppt) were usually collected from large bodies of water such as lakes and the Puget Sound area. The mean measured 2,3,7,8-TCDD concentration in whole fish is about 7 ppt. Since typically only a single sample of whole fish was analyzed from each site, the range and distribution of 2,3,7,8-TCDD concentrations in the fish at these sites cannot be determined.

Measured 2,3,7,8-TCDD levels in whole fish from several areas appear high enough to pose an unacceptable risk to resident wildlife species even with large feeding ranges. For example, in the area around the first 12 pulp mills in Table 13-11, wildlife (e.g., a bald eagle) would only have to obtain about one-tenth of its dietary food intake from the receiving waters to be at risk.

13.4 References

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Table 13-11. Distribution of Dioxin Concentrations in Whole Fish Sampled in the National Bioaccumulation Study

2,3,7,8-TCDD concentration (ppt)	Fish species	State	Water body
117.89	Carp	Louisiana	Wham Brake
107.02	Blue catfish	South Carolina	Sampit River
75.70	Sucker	North Carolina	Pigeon River
67.18	Carp	Wisconsin	Wisconsin River
58.21	White sucker	Maryland	N. Br. Potomac River
40.96	Sucker	Maine	Androscoggin River
34.40	Catfish	Mississippi	Escatawpa River
33.86	Blue catfish	Arkansas	Arkansas River
32.69	White sucker	Minnesota	Rainy River
30.04	Carp	Alabama	Coosa River
28.66	Carp	Alabama	Alabama River
24.04	Carp	Florida	Elevenmile Creek
24.01	Spot	Georgia	Turtle River
22.07	Carp	North Carolina	Pigeon River
21.01	Carp	Michigan	Menominee River
16.60	Sm. buffalo	Alabama	Chicasaw River
16.08	Sucker	Maine	Androscoggin River
15.31	Carp	South Carolina	Catawbee River
14.75	Channel catfish	Ohio	Scioto River
13.69	Channel catfish	Louisiana	Bayou Anacoco
13.19	Bowfin	Florida	Fenholloway River
9.10	Carp	South Carolina	Wateree River
8.58	Carp	Ohio	Scioto River
8.54	Carp	Wisconsin	Peshtigo River
7.97	White sucker	Maine	Penobscot River
7.87	Sucker	Massachusetts	Millers River
7.82	Sucker	New Hampshire	Androscoggin River
6.76	Shorthead redhorse	Virginia	James River
6.40	White sucker	Maine	Kennebec River
6.35	Sucker	California	Sacramento River
6.00	Carp	Mississippi	Mississippi River
5.79	White sucker	Pennsylvania	Clarion River
5.23	Sucker	Washington	Columbia River
5.20	White sucker	Maine	Presumoscot River
5.12	Sucker	Washington	Columbia River
5.02	White carp	Wisconsin	Wisconsin River
4.88	Spotted sucker	Georgia	Altamana River
4.75	Carp	Kentucky	Mississippi River
4.73	Carp	Arkansas	Mississippi River
4.50	Carp	Georgia	Savannah River
4.42	Carpsucker	Kentucky	Ohio River

Table 13-11. (continued)

2,3,7,8-TCDD concentration (ppt)	Fish species	State	Water body
4.30	Carp	Alabama	Tombigbee River
4.17	Sucker	Arkansas	Red River
3.97	Carp	Tennessee	Hiwassee River
3.92	Carp	Wisconsin	Lake Superior Ashland Harbor
3.85	Carp	Michigan	Escanaba River
3.80	Carp	Alabama	Tombigbee River
3.80	Carp	Alabama	Alabama River
3.62	Carp	Arkansas	Ouachita River
3.50	Hardhead catfish	Florida	St. Josephs Bay
3.47	Sucker	California	San Joaquin River
3.46	Carp	Michigan	Muskegon River
3.13	Hardhead catfish	Florida	St. Andrew Bay
3.10	Carp	Alabama	Tombigbee River
2.78	Sucker	Oregon	Columbia River
2.40 nd	Carp	Wisconsin	Wisconsin River
2.01	Redhorse sucker	North Carolina	Neuse River
1.79	Flathead catfish	Louisiana	Mississippi River
1.71	White sucker	Pennsylvania	Hoiter Creek
1.69	Carp	Georgia	Flint River/ Lake Blackshear
1.58	Catfish	Texas	Lake Sam Rayburn
1.57	St. flounder	Washington	Steamboat Slough
1.51	St. flounder	Washington	Commencement Bay
1.40 nd	Largescale sucker	Montana	Clark Fork River
1.20	Goldfish	New York	Hudson River
1.20 nd	Carp	Tennessee	Holston River
1.20 nd	Red drum	Georgia	North River
1.10 nd	Catfish	Virginia	Pamunkey River
1.11 nd	Atlanta salmon	Washington	Port Angeles Bay
1.10 nd	Flathead sole	Alaska	Ward Cove
1.00 nd	St. flounder	Washington	Grays Harbor
0.90 nd	Flathead sole	Alaska	Silver Bay
0.76	Sucker	Oregon	Willamette River
0.70	Carp	Texas	Sulfur River
0.67 nd	White sucker	Maine	St. Croix River
0.59	Redhorse sucker	Pennsylvania	Susquehanna River
0.55	Quillback carp	Alabama	Conecun River
0.50	White sucker	Minnesota	St. Louis River
0.46	Sucker	Idaho	Snake River
0.45	St. flounder	Washington	Grays Harbor
0.41	Sm. buffalo	Texas	Neches River

nd = Not detected.

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APPENDIX A

Bioavailability

TABLE OF CONTENTS

		Page No.
A.1	ASSESSMENT OF BIOAVAILABILITY VIA DERMAL EXPOSURES	A-1
A.1.1	Introduction	A-1
A.1.2	Migration from Solid Matrices	A-2
A.1.2.1	Liquid Mediated Extraction	A-2
A.1.2.2	Dry Transfer from Paper	A-4
A.1.2.3	Exposure to PCDD/F Contaminated Soil and Sludge	A-6
A.1.3	Percutaneous Absorption	A-8
A.1.3.1	Dermal Absorption Studies	A-8
A.1.3.2	Parameters for Exposure Assessment	A-9
A.1.3.3	Factors Affecting Percutaneous Absorption ..	A-11
A.1.3.4	Future Experiments	A-11
A.1.4	Discussion	A-11
A.1.5	References	A-12
A.2	BIOAVAILABILITY OF INGESTED 2,3,7,8-TCDD	A-14
A.2.1	Gastrointestinal Absorption of 2,3,7,8-TCDD in a Volunteer	A-14
A.2.2	Gastrointestinal Absorption of 2,3,7,8-TCDD in Experimental Animals	A-15
A.2.2.1	2,3,7,8-TCDD in Vegetable Oil, in Other Solvents, or in the Diet	A-15
A.2.2.2	2,3,7,8-TCDD in Soil	A-16
A.2.2.3	2,3,7,8-TCDD and Related Substances in Soot	A-20
A.2.2.4	2,3,7,8-TCDD and Related Substances in Fly-Ash	A-21
A.2.3	Bioavailability of Ingested 2,3,4,7,8-PeCDF (pentachlorodibenzofuran)	A-22
A.2.4	Bioavailability of Ingested OCDD (octachlorodibenzo-p-dioxin)	A-22
A.2.5	Evaluation of Bioavailability in Humans Based on Animal Data	A-24
A.2.5.1	Estimated Bioavailability of 2,3,7,8-TCDD from Foods	A-24
A.2.5.2	Estimated Bioavailability of 2,3,7,8-TCDD from Soil	A-24

TABLE OF CONTENTS (continued)

		Page No.
A.2.5.3	Estimated Bioavailability of 2,3,7,8-TCDD from Paper	A-27
A.2.5.4	Estimated Bioavailability of 2,3,7,8-TCDD from Sludge	A-28
A.2.5.5	Estimated Bioavailability of 2,3,7,8-TCDD from Water	A-28
A.2.6	Conclusions	A-28
A.2.7	References	A-28
A.3	BIOAVAILABILITY OF INHALED VAPORS AND PARTICLES CONTAINING 2,3,7,8-TCDD AND 2,3,7,8-TCDF	A-33
A.3.1	Introduction	A-33
A.3.2	Inhalation of Vapors and Bioavailability	A-34
A.3.3	Particulate Inhalation and Deposition	A-34
A.3.4	Bioavailability of Particulate-Bound 2,3,7,8-TCDD and 2,3,7,8-TCDF	A-37
A.4	UPTAKE IN TERRESTRIAL PLANTS	A-37
A.4.1	Root Crops	A-38
A.4.2	Aboveground Crops	A-42
A.4.3	References	A-45

LIST OF TABLES

		Page No.
Table A-1.	Paper:Solvent Equilibrium Partition Coefficients (Kp)	A-3
Table A-2.	Some Properties of TCDD and TRIS	A-5
Table A-3.	In Vivo Dermal Absorption of PCDD and PCDF in Rats ..	A-7
Table A-4.	Bioavailability of Ingested Dioxins and Related Compounds in Vegetable Oils or in the Diet	A-25
Table A-5.	Bioavailability of Ingested Dioxins and Related Compounds in Soils, Soot and Fly-Ash, and Other Substances	A-26
Table A-6.	Bioavailability of Ingested 2,3,7,8-TCDD from Various Media	A-30
Table A-7.	Percent of Total Dust in Different Aerodynamic Diameter (da) Size Fractions	A-36
Table A-8.	Root-to-Soil Ratios for 2,3,7,8-TCDD and PCBs	A-39

Appendix A

Bioavailability

A.1 ASSESSMENT OF BIOAVAILABILITY VIA DERMAL EXPOSURES

A.1.1 Introduction

The purpose of this section is to provide a set of common assumptions for use in assessing the human dermal exposure to polychlorinated dibenzo-p-dioxins (PCDD) and polychlorinated dibenzofurans (PCDF). This section was compiled from:

> Babich M, Adams M, Cinalli C, Galloway D, Hoang K, Huang S, Rogers P. 1989. Common Assumptions for the Assessment of Human Dermal Exposure to Polychlorinated Dibenzo-p-Dioxins and Dibenzofurans. Interagency Dioxin-in-Paper Workgroup, Dermal Bioavailability Workgroup. December 12, 1989.

This integrated risk assessment assesses exposure from a variety of sources such as communications paper, personal care products, contaminated soil, and paper mill wastes. Exposure may occur by a diversity of scenarios, ranging from occupational exposure at paper mills to the use of disposable diapers made of bleached fluff pulp. However, all of these scenarios may be divided into three general categories: (1) liquid mediated extraction from pulp or paper products; (2) dry transfer from paper products; and (3) exposure to contaminated soil or paper mill sludge. Scenario 3 could have been treated as a special case of scenario 1, because exposure is frequently to wet material; however, soil and sludge are treated separately from paper products, in part because of the types and format of the data available.

For the purpose of this multiple source (i.e., multimedia) risk assessment, dermal exposure will be treated as a two-step process: (1) migration or extraction of PCDD/F from the appropriate matrix (e.g., soil, sludge, or paper) onto the skin, followed by (2) percutaneous absorption. Moreover, dermal absorption studies have been done with only a few vehicles (mainly volatile solvents) and only one solid matrix (soil). It would be unrealistic either to conduct dermal absorption studies or to expect to find published studies using matrices and/or vehicles specific for every exposure scenario. On the other hand, there have been studies of the extraction of dioxin from pulp and paper using several different solvents. Therefore, Step 1, migration/extraction, will be treated as scenario specific, whereas Step 2, percutaneous absorption, will be treated as common to all exposure scenarios.

A.1.2 Migration from Solid Matrices

A.1.2.1 Liquid Mediated Extraction

Liquid mediated extraction of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) and 2,3,7,8-tetrachlorodibenzofuran (TCDF) has been characterized by paper:solvent equilibrium partition coefficients (Kp) (NCASI 1987a), defined by:

$$Kp = [TCDD]_{paper}/[TCDD]_{solvent} \quad (A\text{-}1)$$

where $[TCDD]_{paper}$ and $[TCDD]_{solvent}$ represent the equilibrium concentrations of TCDD (or TCDF) in the paper and aqueous phase, respectively. Some of the Kp values in Table A-1 were determined with ground bond paper, at room temperature, and with 24 h for equilibration (NCASI 1987a). Kp values for synthetic urine and isotonic saline at 32° were determined with soft-wood fluff pulp (NCASI 1989). The ethanol Kp was determined with bleached paperboard strips, after 96 h at 49°C (NCASI 1987a). In some cases, 2,3,7,8-TCDD was not detected in the solvent phase. Thus, Kp values for 2,3,7,8-TCDD are lower limits, calculated from the limits of detection in the solvent.

The time course of extraction was reported for synthetic urine (NCASI 1989) and 8 percent ethanol (NCASI 1987b). Equilibrium apparently was reached within 2 h with urine and at 4-to-12 h with 8 percent ethanol. That ethanol required more time to reach equilibrium may be due to the fact that paperboard strips were used as the solid phase, whereas fluff pulp was used with urine. It appears reasonable to assume that equilibrium is reached within 2 h with aqueous solvents under most conditions.

Kp values may be used to estimate the concentration of TCDD/F in a liquid medium in contact with the skin. This concentration may be regarded as an upper limit, since equilibrium conditions are not necessarily expected to occur during the course of exposure. This concentration may be combined with a measure of percutaneous dermal absorption to estimate exposure.

(1) Factors affecting liquid mediated extraction. The factors which are expected to affect the rate or extent of extraction include temperature, solvent, and time. Kp is expected to decrease at higher temperatures, at least with aqueous solvents. (A smaller Kp means more PCDD/F is extracted into the liquid phase). The rate of extraction is expected to increase at higher temperatures. For example, at room temperature and allowing 24 h for equilibration, the paper:water Kp for TCDF was estimated to be 29,000 (NCASI 1987a). Thus, ~1/29,000 or only 0.003 percent of TCDF was extracted from the paper into the liquid phase. In contrast, up to 100 percent of TCDF was extracted from coffee filters in a few minutes, with water at 90°C (NCASI 1988).

Table A-1. Paper: Solvent Equilibrium Partition Coefficients (Kp)

Solvent	Solid phase	Temp. (°C)	Time (h)	Kp TCDD	Kp TCDF
Water[a]	Ground bond paper	21	24	>=13,000	29,000
Isotonic saline[a]	Ground bond paper	21	24	>=19,000	63,000
Isotonic saline[b]	Softwood fluff pulp	32	24	14,300	5,300
Synthetic urine[a]	Ground bond paper	21	24	>=14,000	48,000
Synthetic urine[b]	Softwood fluff pulp	32	2-to-24	14,300	6,300
8% Ethanol[a]	Paperboard strips	49	96	>= 2,000	2,000

[a]NCASI 1987a
[b]NCASI 1989

Extraction is also dependent on the solvent. Extraction from pulp or paper is favored with solvents which can swell the paper fibers, such as alcohols (NCASI 1987b). Thus, 95 percent alcohol was a better extractant than benzene for quantitative 2,3,7,8-TCDD extraction (NCASI 1987b). Extraction by aqueous media is decreased at higher ionic strength. For example, the Kp for TCDF at 21°C is 29,000 with water and 63,000 with isotonic saline (Table A-1).

A.1.2.2 Dry Transfer from Paper

There are no empirical data relating to the "dry transfer" of PCDD/F from paper to skin, such as that which might occur during contact with communications paper. In the absence of appropriate data, previous risk assessments have relied upon various assumptions regarding the possible mechanisms and rates of dry transfer.

(1) Previous risk assessments. In a risk assessment prepared for the U.S. Environmental Protection Agency (EPA), Arthur D. Little, Inc. assumed that dry transfer is actually mediated by skin oil (ADL 1987). In other words, "dry transfer" is fundamentally similar to liquid mediated extraction, but with skin oil as the liquid phase. Because exposure to communications paper is brief (15 sec), the liquid phase penetrates only 1 percent of the depth of the product. It was further assumed that extraction of TCDD by skin oil is 100 percent efficient and that 0.1 percent of the liquid phase remains on the skin. Thus, the fraction of TCDD transferred to the skin in a 15 sec exposure would be:

$$0.01 \times 1 \times 0.001 = 1 \times 10^{-5} \qquad \text{(A-2)}$$

In the U.S. Paper Industry risk assessment, it was assumed that paper is roughly equivalent to soil, except that sheets of paper allow less opportunity for skin contact than granular soil (NCASI 1987a). Thus, the bioavailability of TCDD from sheet paper was assumed to be 50 percent of the bioavailability from soil.

(2) TRIS flame retardant as a model for TCDD. In the absence of data on the unmediated migration of TCDD from paper, the migration of the flame retardant tris-(2,3,dibromopropyl)phosphate (TRIS) from dry cloth will be used as a surrogate (Ulsamer et al. 1978). Like 2,3,7,8-TCDD, TRIS is a nonvolatile, hydrophobic compound (Table A-2). Thus, it is reasonable to employ TRIS as a surrogate for TCDD, but the appropriateness of polyester cloth as a surrogate for paper is of concern. The basis weight of the cloth (~0.01 g cm^{-2}) is roughly equivalent to that of bond paper. However, the abilities of these two matrices as barriers to diffusion or to adsorb TCDD or TRIS are likely to differ. Thus, it is not possible to be certain whether the TRIS cloth model would adequately estimate exposure to TCDD from paper.

Table A-2. Some Properties of TCDD and TRIS

	TCDD[a]	TRIS[b]
Molecular weight	321.97	697.7
Vapor pressure, mm Hg @ 25°C	1.4×10^{-9}	1.9×10^{-4}
Physical state at room temperature	solid	viscous liquid
Melting/freezing point	305°C	5.5°C
Soluble/miscible in:	o-dichlorobenzene chlorobenzene benzene $CHCl_3$ acetone	CCl_4 $CHCl_3$ CH_2Cl_2
Water solubility	19.3 ng/l	insoluble

[a]Neal and Basu 1989
[b]IARC 1979

From the data of Ulsamer et al. (1978), it may be calculated that 4.63 percent of the total amount of radiolabeled TRIS in a dry cloth (applied at a concentration of 160 ug/cm^2) was transferred to rabbit skin after 96 h (Ulsamer et al. 1978). Thus, the rate of transfer was ~0.0005 h^{-1}, assuming that the amount transferred is approximately linear with time during this time period, which is a reasonable assumption, because <10 percent of the initial TRIS dose was transferred after 96 h.

For purposes of this exposure assessment, it will be assumed that TCDD (or TCDF) is transferred from dry paper to skin at the same rate of 0.0005 h^{-1}. Based on this methodology, the fraction of TCDD transferred to the skin in 15 sec would be:

$$0.0005\ h^{-1} \times 15/3600\ h = 2 \times 10^{-6} \qquad (A\text{-}3)$$

which is one-fifth the amount estimated in the ADL (1987) risk assessment. The amount of TCDD transferred to the skin may be multiplied by the fraction of TCDD absorbed to estimate dormal exposure.

A.1.2.3 Exposure to PCDD/F Contaminated Soil and Sludge

Dermal absorption of 2,3,7,8-TCDD in soil has been measured in vivo by direct exposure of rats to soil-bound TCDD, using either dry soil (Shu et al. 1988) or soil-water paste (Poiger and Schlatter 1980). The percentage of soil bound TCDD found in the liver ranged from 0.05 percent to 2.2 percent with wet soil and from 0.65 percent to 1 percent with dry soil (Table A-3). The extent of release of 2,3,7,8-TCDD from soil was estimated by comparing the dermal absorption of soil-bound 2,3,7,8-TCDD with 2,3,7,8-TCDD dissolved in methanol (Poiger and Schlatter 1980). By this methodology, the release of soil-bound TCDD can be estimated to range from 0.3 percent to 15 percent, based on a 24 h exposure.

For the purpose of this exposure assessment, it will be assumed that 1 percent of soil bound TCDD (or PCDD/F) is available for dermal absorption. For sludge, since the physical structure of sludge presumably would allow easier access to TCDD, the high end value of 15 percent release of TCDD is recommended. These factors can be combined with either the rate or fraction of percutaneous absorption to estimate dermal exposure. For comparison, the U.S. Environmental Protection Agency (EPA), Office of Research and Development (ORD) has proposed using 0.5 percent as the overall dermal exposure (i.e., including migration and dermal absorption) of TCDD from contaminated soil (USEPA 1988).

Since oral bioavailability of soil-bound 2,3,7,8-TCDD varies greatly among the different types of soil, this phenomenon may apply to dermal bioavailiability as well. Oral bioavailability of TCDD is reduced in soil with high organic content and by increasing the soil residence time,

Table A-3. In Vivo Dermal Absorption of PCDD and PCDF in Rats

Chemical	Applied dose ug	% Applied dose absorbed	Exposure duration h	Vehicle	Reference
TCDD	0.026	14.8[a]	24	methanol	Poiger & Schlatter 1980
	0.026	1.4		petroleum jelly	
	0.35	9.3		PEG 1500	
	0.35	14.1		PEG-water	
	0.026	-0.05		soil-water	
	0.35	1.7		soil-water	
	1.3	2.2		soil-water	
	0.026	<0.05		activated carbon	
	1.3	<0.05		activated carbon	
TCDD	0.028	0.62[a]	4	soil	Shu et al. 1988
		1.00	24		
TCDD	0.011	38.3[b]	72	acetone	Brewster et al. 1989
	0.072	40.3			
	0.72	27.4			
	7.2	17.8			
	36	19.1			
	72	17.3			
TCDF	7	48.8[b]	72	acetone	Brewster et al. 1989
	34	17.9			
	69	11.3			

[a] Based on liver level.
[b] Based on body burden.

whereas bioavailibility is enhanced by the presence of oil (Poiger and Schlatter 1980; Umbreit et al. 1986, 1988). Furthermore, dermal absorption was undetected when TCDD was applied as an activated carbon-water paste (Poiger and Schlatter 1980) (see Table A-3). However, the presence of used motor oil (up to 2 percent) did not significantly affect dermal absorption of soil-bound TCDD (Shu et al. 1988). While the presence of oil appears to enhance gastrointestinal absorption, use of mineral oil or petroleum jelly as vehicles reduced the dermal absorption of TCDD relative to methanol or acetone, respectively (Poiger and Schlatter 1980; Weber et al. 1989).

The Exposure Assessment Group of EPA/ORD through a contractor is conducting dermal absorption studies with 2,3,7,8-TCDD-contaminated soil. In addition to studying the effects of different soil types, different concentration levels, and the duration of contact, the study also involves in vivo (rats) and in vitro (human cadaver skin) studies of interspecies variation. In this study, total tissue distribution, not just liver levels, will be analyzed. The laboratory experiments should have been completed during the summer of 1989.

A.1.3 Percutaneous Absorption

A.1.3.1 Dermal Absorption Studies

(1) Animal studies. Several laboratories have studied the in vivo percutaneous absorption of 2,3,7,8-TCDD in rats (Banks et al. 1989; Brewster et al. 1989; Poiger and Schlatter 1980; Shu et al. 1988), while one laboratory has also studied 2,3,7,8-TCDF, 1,2,3,7,8-penta-chlorodibenzofuran (1-PeCDF), and 2,3,4,7,8-pentachlorodibenzofuran (4-PeCDF) as well (Banks et al. 1989; Brewster et al. 1989). Most studies have been done by the "finite dose" technique, in which the penetrant (i.e., PCDD/F) was applied to the skin in a volatile solvent, either acetone or methanol, and the solvent was allowed to evaporate. Nonvolatile vehicles (polyethylene glycol, polyethylene glycol-water, and petroleum jelly) have also been studied (Poiger and Schlatter 1980), while two laboratories studied TCDD-contaminated soil (Poiger and Schlatter 1980; Shu et al. 1988). In these studies, absorption was reported as either the fraction of the applied dose found in the liver (Poiger and Schlatter 1980) or as a more complete tissue distribution (Banks et al. 1989; Brewster et al. 1989) at 24 to 72 h post exposure. For the most part, the time course of absorption was not studied, although Shu et al. (1988) reported liver levels were reported at both 4 and 24 h following exposure to TCDD contaminated soil. The effect of aging of organisms on degree of absorption was also studied by Banks et al. (1989).

The results from these studies (Table A-3) show that up to 40 percent of TCDD and 48 percent of TCDF were absorbed by 72 h after initiation of

exposure (Brewster et al. 1989). In addition, the percent dermal absorption (i.e., the percentage of the applied dose which is absorbed) increased as the applied dose decreased, as is generally the case (Scheuplein and Ross 1974). This is important, because the TCDD levels to which humans are expected to be exposed are much lower than the levels used in these laboratory studies.

The only other species studied in vivo is the rhesus monkey. Dermal absorption of 1-PeCDF was studied concomitantly with oral absorption of 4-PeCDF in a rhesus monkey which had survived a previous intravenous 4-PeCDF exposure at a dose which killed two of three animals (Brewster et al. 1988). This study is not appropriate for purposes of a risk assessment, because it involved only one animal and the animal had been previously exposed.

(2) In vitro studies with human skin. One laboratory has reported preliminary data on the penetration of human skin by TCDD in vitro (Weber et al. 1989). Experiments were done using both acetone (i.e., finite dose technique) and mineral oil as vehicles and using both intact and "damaged" skin. The distribution of TCDD in stratum corneum, epidermis, and dermis were reported at times from 30 min to 17 h post exposure.

Interpretation of these in vitro studies is complicated by the distribution of 2,3,7,8-TCDD among the stratum corneum (SC), epidermis, and dermis. Only the portion of TCDD which migrates to the dermis is generally expected to be available for systemic absorption (Weber et al. 1989). However, one might also expect TCDD in the epidermis to be absorbed, because the SC is the primary barrier to percutaneous absorption. Some of the TCDD in the SC may also be absorbed since the SC has been reported to act as a reservoir for certain penetrants (Stoughton 1989). Therefore, in interpreting these in vitro data, we will assume that TCDD in both the dermis and epidermis will be absorbed systemically, but that TCDD in the SC will not be absorbed.

A.1.3.2 Parameters for Exposure Assessment

Measures of dermal exposure include the rate of absorption and the extent of absorption at a given time. Either measure may be required, depending on the exposure model used and the exposure data which are available. Thus, estimates of both will be presented. The rate of absorption will be given as the transfer coefficient, defined by:

$$J = kA \qquad \text{(A-4)}$$

where,

J is the flux (ug cm^{-2} h^{-1}), k is the transfer coefficient (h^{-1}), and A is the specific dose (ug cm^{-2}) (Scheuplein and Ross 1974). The

extent of absorption will be expressed as the percent of the applied dose which is absorbed by 24 h post exposure. Parameters for dermal absorption will be based on "finite dose" experiments (i.e., experiments where volatile solvents such as methanol or acetone were used as vehicles).

Data obtained from human studies are preferred for use in assessing human exposure and risk. In addition, the dermal penetration data with human skin have the advantage that the time course of 2,3,7,8-TCDD absorption was determined over a range of times (0.5 to 17 h) which is particularly relevant to human exposure scenarios. The primary disadvantage of the human data is that they were obtained *in vitro*. However, *in vitro* percutaneous absorption studies have been shown to correlate well with *in vivo* studies (Feldman and Maibach 1970; Franz 1978), including studies with hydrophobic compounds (Bronaugh and Franz 1986; Bronaugh and Stewart 1984, 1986; Cruzan et al. 1986). The primary disadvantage of the *in vivo* studies with rats is that dermal absorption was generally determined at one time point only. In addition, the applicability of rat data to human exposure assessment is uncertain, because human skin is generally considered to be less permeable than rat skin, based on studies with various penetrants, but not TCDD (Bartek et al. 1972; Wester and Maibach 1983). In consideration of the advantages and disadvantages of both kinds of studies, the human data will be used in assessing human exposure.

Based on the *in vitro* data with human skin (Weber et al. 1989), it is estimated that 2,3,7,8-TCDD was absorbed at an average rate of approximately 0.012 h^{-1} over the time period from 0.5 h to 17 h. This estimate is based on the assumption that TCDD in both the dermis and epidermis is available for systemic absorption. However, it has been assumed by others that only TCDD in the dermis is available for absorption, and that TCDD was absorbed at a 10-fold lower rate (Weber et al. 1989).

On average, 18.5 percent of TCDD was absorbed by 17 h post exposure (Weber et al. 1989). Assuming first order kinetics, the fraction (F) of the applied dose which would be absorbed at a given time is:

$$F = 1 - e^{-kt}. \qquad \text{(A-5)}$$

Thus, with a transfer coefficient k of 0.012 h^{-1}, the fraction of 2,3,7,8-TCDD absorbed at 24 h post exposure is ~0.25. Thus, it may be assumed that TCDD is absorbed at a rate of 0.012 h^{-1} and that 25 percent of the applied dose is absorbed by 24 h post exposure.

For comparison, Table A-3 shows that 14.8 percent of TCDD was found in the livers of rats at 24 h post exposure (Poiger and Schlatter 1980). Assuming that liver accounts for roughly half of the body burden (Brewster et al. 1989), ~29 percent of TCDD was absorbed at 24 h, in good agreement with the value of 25 percent estimated from the human *in vitro* studies.

Dermal absorptions of several other PCDD and PCDF isomers in rats have also been studied by Brewster et al. (1989). At a dose of 5 ug cm^{-2}, the extent of absorption of 72 h post exposure decreased in the order: TCDF (49 percent) > 4-PeCDF (34 percent) > 1-PeCDF (25 percent) > TCDD (18 percent).

A.1.3.3 Factors Affecting Percutaneous Absorption

Numerous factors are known to affect the rate or extent of percutaneous absorption (Wester and Maibach 1983). Percutaneous absorption is highly dependent on the vehicle (Bronaugh and Franz 1986). Dermal absorption is expected to decrease when 2,3,7,8-TCDD is applied with hydrophobic vehicles, due to its hydrophobicity. The extent of absorption of 2,3,7,8-TCDD decreased 10-fold with a petroleum jelly vehicle, relative to solvent deposition from methanol (Poiger and Schlatter 1980). The rate of 2,3,7,8-TCDD absorption in vitro also decreased 10-fold with a mineral oil vehicle, as compared to solvent deposition from acetone (Weber et al. 1989). However, dermal absorption is generally increased by skin hydration, occlusion of the application site, and the presence of diseased or damaged skin (Wester and Maibach 1983; Bronaugh et al. 1986). Percutaneous absorption also depends on the anatomical site of application. For example, the scrotum may be 40-fold more permeable than the forearm (Wester and Maibach 1983). Dermal absorption may occur more rapidly in preterm infants, because the SC is not fully developed (Wester and Maibach 1983), while absorption of 2,3,7,8-TCDD in rats was reported to decrease with age (Banks et al. 1989). Finally, the extent of absorption is expected to increase as the surface of exposed skin increases (Noonan and Wester 1980).

A.1.3.4 Future Experiments

NCASI has agreed to fund additional research on percutaneous absorption of TCDD. This work will be done in vitro with human skin and will involve the use of aqueous vehicles, including synthetic urine (Bond 1989).

A.1.4 Discussion

Dermal exposure to PCDD and PCDF may be affected by many factors, for example, the matrix, vehicle, exposure duration, and temperature. Thus, dermal exposure is highly dependent on the exposure scenario. The effect of these variables and others on the common assumptions discussed must be considered in any exposure assessment. In addition, the validity of these assumptions should be reevaluated in light of any new data which become available.

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A.2 BIOAVAILABILITY OF INGESTED 2,3,7,8-TCDD

This section was compiled, with the author's approval, from:

Boyer I. 1989. Bioavailability of Ingested 2,3,7,8-TCDD and Related Substances. U.S. Food and Drug Administration. Draft report submitted to U.S. EPA, Office of Toxic Substances, June 22, 1989.

A.2.1 Gastrointestinal Absorption of 2,3,7,8-TCDD in a Volunteer

After an overnight fast, a healthly male volunteer ingested 105 ng of [1,6-^{3}H] 2,3,7,8-TCDD (1.14 ng TCDD/kg b.w.) dissolved in 6 ml of corn oil (Poiger and Schlatter 1986). About 11.5 percent of the administered ^{3}H-activity was excreted in the feces during the first three days after exposure, followed by a considerable decrease in the rate of ^{3}H elimination; 3.5 percent of the dose was excreted from day 7 to day 125 post-exposure. Of the ^{3}H-activity excreted during the first three days, most was probably from non-absorbed TCDD. Poiger and Schlatter (1986) suggested that the almost complete absorption (88.5 percent) of 2,3,7,8-TCDD observed in this study was probably due to the use of corn oil as

the vehicle, as well as to the fasted state of the subject, and lower absorption may be observed when the 2,3,7,8-TCDD is ingested in food or other carriers. The results also showed that very low doses of 2,3,7,8-TCDD can be highly absorbed in the human gastrointestinal tract.

A.2.2 Gastrointestinal Absorption of 2,3,7,8-TCDD in Experimental Animals

A.2.2.1 2,3,7,8-TCDD in Vegetable Oil, In Other Solvents, or in the Diet

Male and female Sprague-Dawley rats exposed to a single oral dose of 1.0 ug ^{14}C-labelled 2,3,7,8-TCDD/kg body weight (b.w.) in a mixture of acetone and corn oil (1:25, v:v) absorbed about 66 percent to 93 percent (mean ± SD = 84 ± 11 percent) of the total dose (Rose et al. 1976). The mean absorption was 78 ± 13 percent for males and 90 ± 3 percent for females. After repeated oral dosing at 0.1 or 1.0 ug/kg b.w./day, Monday through Friday for 7 weeks, the calculated absorption was 86 ± 12 percent; the mean absorption was 87 ± 6 percent for males and 91 ± 1 percent for females at 0.1 mg/kg b.w./day, and 73 ± 10 percent for males and 93 ± 2 percent for females at 1 ug/kg b.w./day.

Male Sprague-Dawley rats were exposed to a single dose of 50 ug [^{14}C]TCDD/kg b.w. in corn oil by stomach tube (Allen et al. 1975). Approximately 25 percent of the dose was eliminated with the feces within the first 3 days. About 1 to 2 percent/day was excreted during the subsequent 18 days. During the first three days, the fecal content probably represented mainly un-absorbed 2,3,7,8-TCDD. Thus, more than 75 percent of the administered dose was absorbed.

Twenty-four hours after male Syrian Golden hamsters were orally exposed to a single 650 ug/kg dose of [1,6-^{3}H]-labelled 2,3,7,8-TCDD in olive oil, 74 ± 23 percent (mean ± SD) of the ^{3}H-activity remained unexcreted (Olson et al. 1980). Thus, approximately 74 percent of the dose was absorbed.

Male Sprague-Dawley rats were administered 50 ug [^{14}C]TCDD/kg b.w. in acetone:corn oil (1:9, v:v) by gavage (Piper et al. 1973). Almost 30 percent of the administered dose was eliminated in the feces during the first 48 hours. Most of this probably represented un-absorbed 2,3,7,8-TCDD. About 1 to 2 percent of the ^{14}C-activity was excreted each day in the feces over the remaining 19 days. The results suggest that about 70 percent of the dose was absorbed.

An unspecified amount of ^{14}C-labelled 2,3,7,8-TCDD was administered to guinea pigs by gavage, and "only half of the dose was absorbed" (Nolan et al. 1979). However, the vehicle and the method used for calculating the percentage absorbed were not given in the report.

From 54 to 67 percent of the dose was absorbed when ^{14}C-labelled 2,3,7,8-TCDD was administered to male and female Sprague-Dawley rats in the diet to provide 0.5 or 1.4 ug TCDD/kg b.w./day for 42 days (Fries and Marrow 1975). The estimates were obtained by comparing total retention at steady state ($R_{SX\%}$), calculated from actual total retention data and assuming a single compartment and first-order rates, with the steady state total retention predicted assuming 100 percent G.I. absorption ($R_{S100\%}$). $R_{SX\%}$ ranged from 9.4 to 11.6, expressed as multiples of the daily intake, while $R_{S100\%}$ ranged from 17.2 for females to 21.7 for males. Thus, absorption was 54 to 55 percent for males and 55 to 67 percent for females.

Male ICR/Ha Swiss mice were exposed to a single oral dose of 135 ug [^{14}C]TCDD/kg b.w. in 1 percent ethanol (95 percent)/10 percent Tween 80/89 percent saline (Koshakji et al. 1984). About 71 percent of the administered ^{14}C-activity was excreted in the feces during the first 24 hours, and an additional 1 percent during the next 10 days. Koshakji et al. (1984) suggested that the [^{14}C]-activity excreted in the first 24 hours after exposure represents un-absorbed 2,3,7,8-TCDD. Thus, approximately 29 percent of the ingested dose had been absorbed.

A.2.2.2 2,3,7,8-TCDD in Soil

Soil containing 81 ± 8 ppb 2,3,7,8-TCDD from a "highly contaminated" area in Seveso, Italy, was suspended in 10 ml of water and administered by gavage to albino male rabbits to provide 80 ng TCDD/day (30.8 ng/kg b.w./day) for 7 days (Bonaccorsi et al. 1984). For comparison, other rabbits were treated with 80 ng TCDD/day in alcohol/water (1:1). The level of 2,3,7,8-TCDD in the rabbit liver after the 7-day treatment with Seveso soil was 0.88 ± 0.28 ppb, compared to 2.7 ± 0.5 ppb for treatment with the 50 percent alcohol mixture. Thus, the bioavailability of 2,3,7,8-TCDD from the Seveso soil appeared to be 32 percent (99 percent confidence interval 5 to 60 percent) of the bioavailability from the 50 percent alcohol mixture.

In comparison, 2,3,7,8-TCDD-free Seveso soil, with added 2,3,7,8-TCDD ("lab-contaminated soil") to provide 80 ng TCDD/day, yielded liver retention of 56 percent compared with that observed from exposure to the 50 percent alcohol mixture (Bonnaccorsi et al. 1984). Also, rats exposed to 40 ng TCDD/day in lab-contaminated soil exhibited only 74 percent of the liver retention observed in rats exposed to 40 ng TCDD/day either in the 50 percent alcohol mixture or in the acetone/vegetable oil (1:6) mixture. Rats exposed to 20 ng TCDD/day from the lab-contaminated soil exhibited liver retention equal to that observed in rats exposed to 20 ng/day in the acetone/vegetable oil mixture.

Guinea pigs were exposed orally to various samples of soil from either the Times Beach or the Minker Stout site, containing 770 and 880 ppb

2,3,7,8-TCDD, respectively, to provide exposures of 1, 3, or 10 ug TCDD/kg b.w. (McConnell et al. 1984). These soils also contained 40 to 80 ppb dibenzofurans and 2 to 4 ppm PCBs. Control guinea pigs received soil samples in which 2,3,7,8-TCDD, PCBs, or PCDFs were not detected. Another group of guinea pigs received 2,3,7,8-TCDD in corn oil at 0, 1, or 3 ug TCDD/kg b.w. (McConnell et al. 1984). The guinea pigs were observed for 30 days after treatment with the single doses. The estimated LD_{50}s were 1.75 ug/kg b.w. for 2,3,7,8-TCDD in corn oil, 7.15 ug/kg b.w. for Times Beach soil, and 5.50 ug/kg b.w. for the Minker Stout site soil. Bioavailability was not calculated from these results. However, an examination of the hepatic 2,3,7,8-TCDD content in the 2,3,7,8-TCDD-exposed guinea pigs revealed highly efficient absorption of 2,3,7,8-TCDD from the soil samples.

Female Sprague-Dawley rats were exposed to a single oral dose of either 2,3,7,8-TCDD in corn oil or TCDD-contaminated soil from the Minker Stout site (McConnell et al. 1984). Induction of aryl hydrocarbon hydroxylase (AHH) was measured in the rat livers 6 days after treatment. The doses ranged from 0.04 to 5.0 ug TCDD/kg b.w. AHH induction in rats receiving 2,3,7,8-TCDD in corn oil was similar to induction in rats exposed to contaminated soil, when comparing equivalent doses of 2,3,7,8-TCDD. In rats treated with the soil, the AHH activity was about 51 to 114 percent (overall mean = 84 percent) of the activity observed in rats treated with 2,3,7,8-TCDD in corn oil (97 percent at 5 ug/kg b.w.; 51 percent at 1 ug/kg b.w.; 74 percent at 0.2 ug/kg b.w.; 114 percent at 0.04 ug/kg b.w.). Less than 2 percent of the inductive effect of the contaminated soil could be accounted for by the dibenzofurans present, and not more than 0.2 percent by the PCBs. Thus, the TCDD in the Minker Stout site soil was nearly as potent an inducer of AHH activity as TCDD in corn oil, suggesting high bioavailability from the soil.

Guinea pigs were exposed to single oral doses of 2,3,7,8-TCDD in soils (10 percent suspensions in 5 percent gum acacia) from heavily contaminated sites in Newark, New Jersey, including a 2,4,5-T manufacturing site and a metal salvage site (Umbreit et al. 1986a). More than 50 different dibenzodioxins and dibenzofurans were detected in the soil samples. The negative control was a decontaminated soil sample from the manufacturing site. The positive controls were 2,3,7,8-TCDD in a 1:9 mixture of acetone and corn oil, and 2,3,7,8-TCDD placed on decontaminated soil 1 hour before dosing ("recontaminated soil").

The soil from the 2,4,5-T manufacturing site was substantially less toxic than equivalent doses of 2,3,7,8-TCDD in corn oil (Umbreit et al. 1986a). The 2,3,7,8-TCDD in either corn oil or recontaminated soil, both providing 6 ug TCDD/kg b.w., proved highly toxic. These positive control treatments produced death with typical signs of 2,3,7,8-TCDD toxicity in over half the treated animals (6 ug/kg b.w.). In contrast, guinea pigs treated with soil from the manufacturing site, at up to 12 ug/kg b.w.,

showed some signs of toxicity, but none that could be attributed to 2,3,7,8-TCDD ("TCDD syndrome"); all survived 60 days, except for those that died as a result of gavage error. Thus, the lethal dose for 2,3,7,8-TCDD in the manufacturing site soil was clearly greater than 12 ug/kg b.w.

Accordingly, at 60 days, liver TCDD content was 18,000 ppt in the rats treated with recontaminated soil containing 6 ug TCDD/kg b.w., compared to 90 ppt in rats treated with manufacturing site soil containing 12 ug/kg b.w. (Umbreit et al. 1986a). Other compounds (including other dibenzo-dioxins and dibenzofurans) in the contaminated soil from the manufacturing site did not appear to alter the toxicity of the 2,3,7,8-TCDD in the soil, by inhibiting 2,3,7,8-TCDD absorption in the G.I. tract or by competing with 2,3,7,8-TCDD at the receptor level. Thus, the relatively low toxicity and low liver content of 2,3,7,8-TCDD in the rats treated with the manufacturing site soil were attributed to low bioavailability.

In a separate experiment, soil from the metal salvage site was administered to rats to provide 0.32 ug TCDD/kg b.w. (Umbreit et al. 1986a). Neither the contaminated soil nor the negative control (decontaminated soil) caused any signs of toxicity. However, the liver TCDD-content was 230 ppt after treatment with metal salvage site soil (0.32 ug TCDD/kg b.w.), compared to a TCDD liver content of 90 ppt after treatment with manufacturing site soil (12 ug TCDD/kg b.w.). Thus, although the TCDD content of the metal salvage site soil was lower, the bioavailability of 2,3,7,8-TCDD in this soil was substantially greater than in the manufacturing site soil.

In another study, Umbreit et al. (1986b, 1988b) reported that 8 of 14 guinea pigs treated with Times Beach soil at 10 ug TCDD/kg b.w. died of TCDD intoxication within the first 20 days (average time-to-death = 14.3 ± 3.2 days). In comparison, 19 of 20 positive control (recontaminated soil) guinea pigs died within 20 days (average time-to-death = 9.8 ± 3.2 days). In contrast, the manufacturing site soil (at 10 ug TCDD/kg b.w.) produced only a single death from apparent TCDD intoxication out of the 18 guinea pigs treated, and that one guinea pig died 57 days after the initiation of treatment.

Liver-TCDD contents were measured in male and female Hartley guinea pigs for up to 60 days after exposure to a single dose of contaminated soil from Times Beach, and the measurements were compared to the liver contents after exposure to the manufacturing site soil (Umbreit et al. 1988a). Soils from both sources are contaminated with several chlorinated dioxins and numerous other compounds. On the basis of liver analyses, the bioavailability of 2,3,7,8-TCDD in the Times Beach soil (10 ug TCDD/kg b.w.) was approximately 29.5 percent, and the bioavailability of 2,3,7,8-TCDD in the manufacturing site soil (10 ug/kg b.w.) was about 1.6 percent, of the bioavailability from recontaminated soil (6 ug TCDD/kg b.w.). The liver TCDD-contents were as follows: recontaminated soil, 56,000 ppt; Times Beach soil, 27,500 ppt; 2,4,5-T manufacturing site soil, 1,500 ppt.

When an animal fed PCBs undergoes weight loss as a result of illness or restricted food intake, the fat is metabolized and PCBs in the fat are transferred to the liver. This process typically produces a decrease in the PCB concentration in adipose tissue, and a corresponding increase in the liver PCB concentration. By analogy, a toxic quantity of Times Beach soil (at 10 ug TCDD/kg b.w.) would be expected to produce a higher liver TCDD-content than a non-toxic quantity of the manufacturing site soil (also at 10 ug TCDD/kg b.w.), simply because of the transfer of TCDD from adipose tissue to liver in those animals succumbing to TCDD toxicity. When male Sprague-Dawley rats were exposed orally to 50 ug of radiolabeled TCDD/kg b.w. in corn oil (approximately twice the LD_{50}) and observed for up to 21 days post-treatment, the rats lost weight and exhibited a decided depletion of depot fat (Allen et al. 1975). However, the concentration of radioactivity in the adipose tissue remained constant, and the concentration in the liver showed a 62 percent decrease. Thus, the redistribution of TCDD at toxic doses may not be analogous to the redistribution of PCBs.

Umbreit et al. (1986a) suggested there are several possible explanations for the observed differences in soil-TCDD bioavailability. For example, the bioavailability of dioxin may depend on the nature of the soil and matrix material. The soil at the manufacturing site contained asphalt and concrete as well as coarse sand-soil fill. Umbreit et al. (1986a) suggested that the "carbonaceous" materials in the asphalt and tar could enhance binding to the matrix and thus reduce bioavailability. Accordingly, much less than 0.07 percent of a 14.7 ng dose administered to rats by gavage was retained in hepatic tissues when the 2,3,7,8-TCDD was mixed in an aqueous suspension of activated carbon (25 percent w/w) and stored for 15 to 20 hours at room temperature before dosing. In contrast, 36.7 $\pm$ 1.2 percent was retained in hepatic tissues when the dose was administered in 50 percent ethanol. These results suggest that absorption was almost completely eliminated by the presence of activated carbon.

Likewise, Silkworth et al. (1982) and Kaminsky et al. (1985) reported that the 42-day LD_{50} values for female Hartley guinea pigs were 2.5 ug/kg b.w. for 2,3,7,8-TCDD administered by gavage in corn oil, and 19 ug/kg b.w. for 2,3,7,8-TCDD in an aqueous 0.75 percent methyl cellulose mixture, a 7.6-fold decrease in toxicity associated with the presence of the organic methyl cellulose carbon.

Also, bioavailability may be affected by differences in the application of the 2,3,7,8-TCDD to the soil and by the residence time of 2,3,7,8-TCDD in the soil (McConnell et al. 1984). The soil at the 2,4,5-T site was contaminated over decades from a generally aqueous medium by leaks in the manufacturing stream and spills. Furthermore, this site was open to the environment, and solvent materials may have enhanced the percolation into the soil and thus facilitate the binding to soil components. In

contrast, the Times Beach site was contaminated by spraying waste oil, contaminated with 2,3,7,8-TCDD and other chlorinated dibenzo-p-dioxins, directly onto a sandy loam-soil to control road dust (McConnell et al. 1984). The presence of the waste oil and the absence of certain other types of material may largely account for the relatively high bioavailability of 2,3,7,8-TCDD from the Times Beach soil.

The comparative extractability data also support the hypothesis that the differences in bioavailability between Times Beach and Newark soils are a function of soil binding. 2,3,7,8-TCDD is weakly sorbed onto Times Beach soil as evidenced by the ease with which it is extracted. In contrast, 2,3,7,8-TCDD is very difficult to extract from the Newark 2,4,5-T manufacturing site soil, indicating that it is tightly sorbed onto this soil. Umbreit et al. (1986a) reported that Soxhlet extraction of the Times Beach soil yielded a similar quantity of 2,3,7,8-TCDD (950 ppb) as compared to the solvent extraction of this soil (770 ppb), as reported by McConnell et al. (1984). In contrast, the Soxhlet-extractable 2,3,7,8-TCDD in the 2,4,5-T manufacturing site soil was 2200 to 2280 ppb, and only a small fraction, ">2.5 ppb," was extractable by the solvent extraction methodology used by McConnell et al. (1984).

In addition, oral exposures of female Sprague-Dawley rats to uncontaminated Seveso soil spiked with 2,3,7,8-TCDD produced lower hepatic levels of 2,3,7,8-TCDD than exposures to 2,3,7,8-TCDD in 50 percent ethanol (Poiger and Schlatter 1980). Comparison of the percentages of the doses found in the liver indicates that 2,3,7,8-TCDD absorption from the spiked soil was 68 to 73 percent (doses = 14.7 to 22.9 ng) of the absorption from the solvent vehicle. Aging the spiked soil for 8 days at 30 to 40°C following the addition of 2,3,7,8-TCDD decreased the uptake into the liver to 44 percent of the uptake of 2,3,7,8-TCDD from the 50 percent ethanol solution. This observation is consistent with reports showing that 2,3,7,8-TCDD from environmental soil (naturally aged) was generally less bioavailable than 2,3,7,8-TCDD freshly added to clean samples of these soils. Likewise, Philippi et al. (1981) and Huetter and Philippi (1982) have shown that radiolabeled 2,3,7,8-TCDD added to soil becomes progressively more resistant to extraction with time.

A.2.2.3 2,3,7,8-TCDD and Related Substances in Soot

Kaminski et al. (1985) and Silkworth et al. (1982) have examined the toxicity of soot containing dibenzodioxins and dibenzofurans from a polychlorinated biphenyl (transformer) fire. Hartley guinea pigs were exposed to one of the following in a single oral dose: 1) soot in an aqueous 0.75 percent methylcellulose vehicle, or 2) Soxhlet benzene extract of the soot in the aqueous 0.75 percent methylcellulose vehicle. The soot contained approximately 1.2 ppm 2,3,7,8-TCDD, 48 ppm 2,3,7,8-TCDF, and 5,000 ppm PCBs, in addition to many other chlorinated dibenzodioxin and dibenzofuran congeners. 2,3,7,8-TCDF was estimated to be the most hazardous toxic component of the soot because of its high toxicity to

guinea pigs and the relatively large quantities present. The 42-day LD_{50} of the soot in the aqueous methylcellulose vehicle was 410 mg soot/kg b.w. (95 percent confidence limits, 281 and 604), a dose which would provide approximately 0.5 ug TCDD/kg b.w. (confidence limits, 0.3 and 0.7), 20 ug TCDF/kg b.w. (confidence limits, 13 and 29), and 2,050 ug PCBs/kg b.w. (confidence limits, 1,404 and 3,020). In comparison, the extract of the soot in the aqueous methylcellulose vehicle yielded a 42-day LD_{50} of 327 mg soot equivalent/kg b.w. (95 percent confidence limits, 183 and 583), a dose which would provide approximately 0.4 ug TCDD/kg b.w. (confidence limits, 0.2 and 0.7), 16 ug TCDF/kg b.w. (confidence limits, 9 and 28), and 1,635 ug PCBs/kg b.w. (confidence limits, 915 and 2,915). Thus, in the 0.75 percent aqueous methylcellulose suspension, the carbon-like matrix of the soot had little, if any, effect on the oral toxicity of the substances in the soot.

A.2.2.4 2,3,7,8-TCDD and Related Substances in Fly-Ash

Van den Berg et al. (1985) demonstrated that 2,3,7,8-TCDD and related substances were bioavailable from fly-ash mixed 25 percent w/w with standard laboratory diet consumed by guinea pigs over 31 to 95 days. Approximately 0.9 to 3.7 percent of the total 2,3,7,8-TCDD and approximately 2.2 to 4.7 percent of the total 2,3,7,8-TCDF were taken up into the livers of the guinea pigs. Liver contents in the guinea pigs ranged from 0.5 percent of the total 1,2,3,4,7,8-HxCDD + 1,2,3,6,7,8-HxCDD (hexachlorinated dibenzo-dioxins) to 11.3 percent of the 1,2,3,7,8-PnCDN (pentachlorinated dibenzofuran).

Van den Berg et al. (1983) fed Wistar rats for 19 days with a diet mixed with one of the following: 1) fly-ash containing dioxins and furans, 2) a Soxhlet/ toluene extract of the fly-ash, or 3) an extract of the fly-ash purified by means of column chromatography. The rats receiving food mixed with the purified fly-ash extract retained at least 3 to 5 times as much polychlorinated dibenzodioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) in their livers as those rats fed the fly-ash itself. For PnCDD, HxCDF, and HxCDD isomers, the liver concentrations were 10 to 20 times higher for food mixed with purified extract vs. the fly-ash itself. Liver retention in rats fed food mixed with the crude fly-ash extract was somewhat reduced compared to that in rats fed food containing the purified extract. However, the doses of PCDDs and PCDFs in the fly-ash were from 76 to 151 percent of the doses administered by mixing the crude extract with the food, and from 51 to 112 percent of the doses administered by mixing the purified extract with the food. Correcting for these differences suggests that the bioavailability of TCDD from the fly-ash itself was 70 to 75 percent as high as the bioavailability from the crude extract mixed with the food, and 58 to 75 percent as high as the purified extract mixed with the food.

A.2.3 Bioavailability of Ingested 2,3,4,7,8-PeCDF (pentachlorodibenzofuran)

Apparently, no data are available for feeding studies with 2,3,7,8-tetrachlorodibenzofuran.

Male Fischer rats were orally exposed to 34, 170, or 340 ug 2,3,4,7,8-PeCDF/kg b.w. by gavage (Brewster et al. 1987). The 2,3,4,7,8-PeCDF was dissolved in acetone/corn oil, and the acetone was removed by evaporation prior to administration. Approximately 30 percent of the dose was excreted within 3 days, independent of the magnitude of the dose. For rats exposed to 34 ug PeCDF/kg b.w. by intravenous (i.v.) injection, about 3 percent of the 2,3,4,7,8-PeCDF was eliminated in the feces within 3 days. The results showed that more than 70 percent of the oral dose was absorbed.

A.2.4 Bioavailability of Ingested OCDD (octachlorodibenzo-p-dioxin)

Toxicity studies of OCDD have not demonstrated lethality at g/kg b.w. dosages in rats or mice, and OCDD has been classified as essentially non-toxic by some investigators (Norback et al. 1975, Williams et al. 1972, Schwertz et al. 1973). However, several investigators have observed tissue accumulation following repeated exposure to OCDD (Birnbaum and Couture 1988, Norback et al. 1975, Williams et al. 1972, Couture et al. 1988), and Schwertz et al. (1973) reported that OCDD was embryotoxic at 500 mg/kg b.w./day.

OCDD was administered at 100 ug in 1 ml corn oil to male Sprague-Dawley rats by gastric intubation (Norback et al. 1975). The OCDD had been bomarded with neutrons to produce 12.6 pg SCDD ([^{35}S] thiohepta-chlorodibenzo-p-dioxin) to serve as a marker. About 0.56 percent of the total dose was present in the tissues after 21 days of continuous treatment, suggesting a low rate of gastrointestinal absorption.

[^{14}C]OCDD was dissolved in o-dichlorobenzene (DCB), and male Fischer 344 rats were exposed by gavage to 50, 500, or 5000 ug OCDD/kg b.w. (Birnbaum and Couture 1988). The low dose (50 ug/kg b.w.) was administered in the same vehicle (DCB/Emulphor 620, 1:1) and volume (0.2 ml/kg b.w.) as for rats exposed to this dose by i.v. injection. The middle dose (500 ug/kg b.w.) was administered in 1 ml DCB:corn oil (1:1)/kg b.w., or in 1 ml corn oil/kg b.w. (Birnbaum and Couture 1988). The highest dose (5000 ug/kg b.w.) was given in 1 ml corn oil/kg b.w. For repeated exposure, the rats were treated orally once a day, 1, 2, 4, 7, or 10 times, and sacrificed 3 days after the last exposure.

Birnbaum and Couture (1988) reported that more than 85 percent of the radioactivity was eliminated with the feces within 3 days after oral treatment with 50 or 500 ug OCDD/kg b.w. (in DCB:corn oil). More than

95 percent of the dose appeared in the feces within 3 days after exposure to 500 and 5000 ug/kg b.w. (in corn oil). In contrast, only 13 percent of the 50 ug OCDD/kg b.w. administered i.v. was excreted in the feces, and less than 0.2 percent in the urine, within the first 3 days. By day 56 post-exposure, the rate of excretion in the feces was reduced to 0.04 percent/day. Thus, OCDD is poorly absorbed after oral exposure.

Birnbaum and Couture (1988) also reported that repeated doses of OCDD resulted in increased body burden proportional to the number of doses administered. However, as the oral dose was increased, proportionally less OCDD was found in the tissues. At 500 ug/kg b.w. (in DCB:corn oil), approximately one-quarter to one-half as much of the dose was found in the liver as after 50 ug/kg b.w. (in DCB:corn oil). When the dose was increased to 5000 ug/kg b.w., the proportion of OCDD retained in the liver decreased further, such that a 10-fold increase in the dose (from 500 ug/kg b.w. to 5000 ug/kg b.w., in corn oil) resulted in less than a 3-fold increase in hepatic OCDD concentrations and no increase in blood or skin concentrations. The results suggest that absorption is nonlinear at exposure levels between 0.5 and 5 mg/kg b.w.

[^{14}C] OCDD was dissolved in acetone/corn oil, and the acetone was evaporated (Couture et al. 1988). Rats were exposed to 50 ug OCDD/kg b.w. by gastric intubation. The rats were dosed once a day, 5 days per week, for a total of 10, 20, 40, or 65 times. The liver was the major depot, accounting for 97 percent of the total body burden after 65 doses, with no evidence of saturation of any of the depots. Some alterations in the liver were observed, such as fatty vacuolization, which may be an early indicator of potentially severe liver damage. Also, some hematologic changes were noted. However, no severe toxicity was reported. Couture et al. (1988) estimated that approximately 10 percent of the total administered dose was absorbed, on the basis of OCDD liver retention. This estimate is supported by the observation that absorbed OCDD is persistent in the body; Birnbaum and Couture (1988) calculated that the whole-body half-life of OCDD was at least 70 days, after exposing rats to 50 ug OCDD/kg b.w. by i.v. injection.

At a dose of 11.5 ug [^{14}C]OCDD/kg b.w. in corn oil, the concentration of OCDD in the liver at the higher dose volume (5 ml/kg b.w.) was more than 6-fold higher than at the lower dose volume (1 ml/kg b.w.) 3 days after exposure (Couture et al. 1988). Thus, the low gastrointestinal absorption of OCDD is probably a function of its extreme insolubility. As the concentration of OCDD is increased in corn oil, the OCDD is probably present more in suspension than in solution, a factor which may account for reduced G.I. absorption at higher concentrations.

A.2.5 Evaluation of Bioavailability in Humans Based on Animal Data

A.2.5.1 Estimated Bioavailability of 2,3,7,8-TCDD from Foods

One would expect slow but nearly complete absorption of pure 2,3,7,8-TCDD from the gastrointestinal tract based on its high lipid solubility and low solubility in water. Accordingly, the estimates of bioavailability presented in Table A-4 indicate that 2,3,7,8-TCDD is readily absorbed from vegetable oils or laboratory diet. Almost complete absorption (88.5 percent) of 2,3,7,8-TCDD was noted in a human volunteer who consumed radiolabelled 2,3,7,8-TCDD in corn oil after an overnight fast (Poiger and Schlatter 1986). Similarly, absorption of 2,3,7,8-TCDD in various vegetable oil mixtures ranged from 70 to 93 $\pm$ 2 percent in rats and hamsters (Rose et al. 1976, Allen et al. 1975, Piper et al. 1973). These results suggest that 85 to 95 percent absorption is a reasonable estimate of 2,3,7,8-TCDD bioavailability from fatty or oily foods, especially milk, fish, and meats.

Fries and Marrow (1975) reported that rats absorbed 54 to 67 percent of the 2,3,7,8-TCDD mixed into their unpelleted standard laboratory diet (Table A-4). The decreased bioavailability of 2,3,7,8-TCDD in the diet is consistent with other studies showing lower 2,3,7,8-TCDD bioavailability in various experimental vehicles compared to the bioavailability in corn oil (Koshakji et al. 1984, Silkworth et al. 1982, Nolan et al. 1979) (Table A-4). Thus, 60 to 70 percent absorption appears to be a reasonable estimate of 2,3,7,8-TCDD bioavailability from foods other than those with high fat or oil content.

A.2.5.2 Estimated Bioavailability of 2,3,7,8-TCDD from Soil

The estimates presented in Table A-5 indicate that 2,3,7,8-TCDD added to non-contaminated Seveso soil shortly before ingestion ("spiked soil") was only 56 to 74 percent as bioavailable as 2,3,7,8-TCDD in solvent vehicles (vegetable oil or 50 percent ethanol), with 95 percent confidence limits ranging from 32 to 95 percent (Poiger and Schlatter 1980, Bonaccorsi et al. 1984) (Table A-5). Furthermore, the 2,3,7,8-TCDD in the spiked Seveso soil at the lowest dose tested appeared to be as bioavailable as an equivalent dose of 2,3,7,8-TCDD in acetone/vegetable oil vehicle (Bonaccorsi et al. 1984) (Table A-5). These results attest to the potential for high TCDD-bioavailability from soils.

On the other hand, the bioavailability of 2,3,7,8-TCDD from an environmentally-contaminated 2,4,5-T manufacturing site soil was reduced to 2 percent of the bioavailability from the spiked sample of non-contaminated manufacturing site soil (Umbreit et al. 1988a) (Table A-5). These results demonstrate the potential for marked decreases in bioavailability as a result of soil-TCDD interactions. Reduced bioavailability of 2,3,7,8-TCDD in environmentally contaminated soil compared to 2,3,7,8-

Table A-4. Bioavailability of Ingested Dioxins and Related Compounds in Vegetable Oils or in the Diet

Species	Substance	Dose (ug/kg)	Vehicle/ carrier	Bioavailability (%)	Reference
Man	2,3,7,8-TCDD	0.00114	corn oil (6 ml)	88.5	Poiger and Schlatter (1986)
Rat	2,3,7,8-TCDD	1.0 0.1/day or 1.0/day[a,b]	acetone:corn oil (1:25)	84±11[c](66-93)[d] 86±12[c](73±10-93±2)[e]	Rose et al. (1976)
Rat	2,3,7,8-TCDD	50	corn oil	>75	Allen et al. (1975)
Hamster	2,3,7,8-TCDD	650	olive oil	74±23[c]	Olson et al. (1980)
Rat	2,3,7,8-TCDD	50	acetone:corn oil (1:9)	70	Piper et al. (1973)
Guinea pig	2,3,7,8-TCDD	NR[f]	NR[f]	50	Nolan et al. (1979)
Rat	2,3,7,8-TCDD	0.5 or 1.4/day for 42 days	diet	54-67	Fries and Marrow (1975)
Mouse	2,3,7,8-TCDD	135	1% ethanol/10% Tween 80/89% saline	29	Koshakji et al. (1984)
Rat	2,3,4,7,8-PeCDF	34, 170, or 340	corn oil	>70 for each dose	Brewster et al. (1987)
Rat	OCDD	50 or 500	DCB:corn oil	<15	Birnbaum and Couture (1986)
Rat	OCDD	500 or 5,000	corn oil	<5	Birnbaum and Couture (1986)
Rat	OCDD	50/day, 10-65 times[a]	corn oil	10	Couture et al. (1988)

[a] Monday through Friday.
[b] For 7 weeks.
[c] Mean ± SD, all rats.
[d] Range for individual rats.
[e] Range of means for groups of rats.
[f] Not reported.

Table A-5. Bioavailability of Ingested Dioxins and Related Compounds in Soils, Soot and Fly-Ash, and Other Substances

Species	Substance	Dose (ug/kg)	Vehicle/ carrier	Bioavailability (%)	Relative to	Reference
BASIS FOR COMPARISON - Toxicity (doses = estimated LD_{50}s):						
Guinea pig	TCDD	5.50	Minker Stout soil	32	LD_{50} = 1.75 ug/kg in corn oil	McConnell et al. (1984)
		7.15	Times Beach soil	24		
Guinea pig	Soot	327,000[a]	0.75% aqueous methylcellulose	80 (30-207)[b]	Soxhlet extract of the soot in 0.75% aqueous methylcellulose	Kaminski et al. (1985)
Guinea pig	TCDD	19	0.75% aqueous methylcellulose	13	LD_{50} = 2.5 ug/kg in corn oil	Silkworth et al. (1982)
BASIS FOR COMPARISON - Liver TCDD-concentration:						
Guinea pig	TCDD	10	Times Beach soil	30	Non-contaminated manufacturing site soil with added TCDD (6 ug/kg)	Umbreit et al. (1988a)
			Manufacturing site soil	2		
Rabbit	TCDD	0.031/day[c]	Contaminated Seveso soil	32(5-60)[d]	Same dose in: 50% ethanol	Bonaccorsi et al. (1984)
		0.031/day[c]	Spiked Seveso soil	56(32-81)[d]	50% ethanol	
		0.015/day[c]	Spiked Seveso soil	74(47-95)[d]	50% ethanol or veg. oil[e]	
		0.008/day[c]	Spiked Seveso soil	100	veg. oil[e]	
Rat	TCDD	0.04-0.06	Spiked Seveso soil	68-73	Same dose in 50% ethanol	Poiger & Schlatter (1980)
Rat	TCDD	3.2	Purified extract of fly-ash mixed with food	58-75	Ingesting the fly-ash itself (1.6 ug/kg)	van den Berg et al. (1983)
Rat	TCDD	0.04	Aqueous activated-carbon	<2	Same dose in 50% ethanol	Poiger & Schlatter (1980)

[a] Provides 0.5 ug TCDD/kg, 20 ug TCDF/kg, and 2,050 ug PCBs/kg.
[b] Mean (95% confidence interval).
[c] For 7 days.
[d] Mean (99% confidence interval).
[e] Veg. oil = acetone/vegetable oil (1:6).

TCDD in freshly spiked soil is consistent with reports that aging the spiked soil reduces the TCDD bioavailability (Poiger and Schlatter 1980). Presumably, 2,3,7,8-TCDD becomes more tightly associated with the soil over time (Philippi et al. 1981, Huetter and Philippi 1982) by adsorbing onto soil components which act in a manner analogous to activated carbon or aqueous methylcellulose (Kaminski et al. 1985, Poiger and Schlatter 1980) (Table A-5), thus reducing the bioavailability.

The 2,3,7,8-TCDD in environmentally contaminated soil samples from the Seveso, Times Beach, and Minker Stout sites was from 24 to 32 percent as bioavailable as TCDD in corn oil, or in spiked manufacturing site soil, with 99 percent confidence limits of 5 and 60 percent reported for the Seveso soil (McConnell et al. 1984, Umbreit et al. 1988a, Bonaccorsi et al. 1984) (Table A-5). Thus, in the absence of site-specific information concerning bioavailability, or solvent extractability, to which bioavailability may be correlated (Umbreit et al. 1986a), 45 to 55 percent absorption appears to represent a reasonable estimate of 2,3,7,8-TCDD bioavailability from environmentally contaminated soil.

A.2.5.3 Estimated Bioavailabiliy of 2,3,7,8-TCDD from Paper

Workers employed in the manufacturing of paper products may inhale dioxin-contaminated paper dust that would be deposited in the airways and, through mucocilliary action, can gain entry to the gastrointestinal tract. Apparently, no information is available which specifically addresses the bioavailability of 2,3,7,8-TCDD from ingested paper dust.

However, it has been argued that the bioavailability from paper dust can be estimated from the observed bioavailability from other matrices, such as soil, fly ash, or soot (NCASI 1988a), and that soil serves as the best surrogate for paper dust because the primary source of the organic components of soil, namely decaying plant (cellulosic) materials, would be expected to be the most similar to the organic components of paper. On this basis, NCASI (1988a) estimated that the bioavailability of 2,3,7,8-TCDD from soil was 57 percent, based on a review of the available scientific literature, and proposed 50 percent bioavailability for 2,3,7,8-TCDD from paper. Fifty percent bioavailability from paper dust is suggested to be an over-estimate of the actual bioavailability because 1) paper dust has a higher "organic" content than soil, and 2) the much lower doses expected from paper dust may be associated with lower bioavailability than the higher doses tested in the soil studies.

However, Poiger and Schlatter (1986) showed that even extremely low doses of 2,3,7,8-TCDD can be highly absorbed in the human G.I. tract. Moreover, the animal data suggest that decreases in the doses of 2,3,7,8-TCDD added to non-contaminated soil can exhibit increases in the bioavailability. In addition, although there may be an inverse relationship between 2,3,7,8-TCDD bioavailability and the presence of certain sub-

stances in the soil, such as charcoal, tar, and other "carbonaceous" materials, it seems unlikely that these "carbonaceous" materials can be equated with "organic" material, such as the cellulose in paper. Likewise, Kaminski et al. (1985) reported that a 0.75 percent aqueous methylcellulose suspension reduced the bioavailability of 2,3,7,8-TCDD compared to 2,3,7,8-TCDD in corn oil, but the aqueous methylcellulose suspension is probably not comparable to the cellulose matrix of paper.

On the other hand, Umbreit et al. (1986a) proposed that a direct relationship exists between the solvent extractability of 2,3,7,8-TCDD from soil and the bioavailablity of 2,3,7,8-TCDD from the soil. By analogy, the degree to which 2,3,7,8-TCDD can be extracted from paper dust may be related to its bioabailability from the paper matrix.

NCASI (1987) reported that greater amounts of 2,3,7,8-TCDD and 2,3,7,8-TCDF were extracted from a non-barrier food packaging paper in a 50 percent ethanol solution compared with the amounts originally thought to be present in the packaging material, suggesting highly efficient extraction. Alcohol solutions apparently swell paper pulp fibers, as does water itself (Browning 1963, Kress and Bialkowsky 1931, McKenzie 1957). As the paper fiber swells, the intermolecular bonds break, the degree of order within the fiber diminshes, the surface area elevates, and the number of available inter- and intra-molecular bonding sites increases. The ethanol forms hydrogen-bonds with hydroxy groups in the swollen fiber, creating a more lipophilic environment at the fiber. The NCASI (1987) report suggests that the process probably facilitates desorption and subsequent diffusion of PCDD/PCDF occluded within the fiber, providing a reasonable explanation for the relatively efficient extraction of TCDD and TCDF from paper into 50 percent ethanol. This phenomenon may also account, to some extent, for observations of relatively high migration of 2,3,7,8-TCDD and 2,3,7,8-TCDF from paper coffee filters to the coffee brewed using these filters, with up to 62 percent migration for 2,3,7,8-TCDD and up to 79 percent or more for 2,3,7,8-TCDF (NCASI 1988b).

In comparison, bile salts in the intestines combine with lipids to form micelles (water soluble complexes) from which the lipids can be more easily absorbed (the hydrotropic effect). The bile salts reduce surface tension and, in conjunction with fatty acids and glycerides, emulsify fats and oils prior to their digestion and absorption. Water in the G.I. tract can swell ingested paper, providing the digestive juices with increased accessibility to the paper matrix. In turn, the emulsion in the intestines may present a more lipophilic environment to the swelled paper matrix. Thus, by mimicking the action of 50 percent ethanol, the fluids in the G.I. tract may support a relatively high rate of migration of TCDD and TCDF from the paper to a highly absorbable fraction of the intestinal fluids.

The foregoing argument suggests that the bioavailability of 2,3,7,8-TCDD from ingested paper may be similar to the bioavailability from foods with low fat or oil content. Thus, in the absence of adequate data, 60 to 70 percent absorption appears to be a reasonable estimate of 2,3,7,8-TCDD bioavailability from ingested paper.

A.2.5.4 Estimated Bioavailability of 2,3,7,8-TCDD from Sludge

No information is available which specifically addresses the bioavailability of 2,3,7,8-TCDD from ingested sludge. However, one may suppose that in the absence of oily substances in the sludge, the bioavailability of 2,3,7,8-TCDD from the sludge might be less than that from solvent vehicles, such as corn oil, or from fatty or oily foods. Instead, the bioavailability of 2,3,7,8-TCDD in sludge might be best compared with the bioavailability in foods with relatively low fat or oil content. Thus, in the absence of adequate data, 60 to 70 percent absorption appears to be a reasonable estimate of 2,3,7,8-TCDD bioavailability from sludge.

A.2.5.5 Estimated Bioavailability of 2,3,7,8-TCDD from Water

No information is available which specifically addresses the bioavailability of 2,3,7,8-TCDD in drinking water. However, one may suppose that the 2,3,7,8-TCDD in drinking water will be present at the maximum solubility of 2,3,7,8-TCDD in water, and that the solubilized 2,3,7,8-TCDD is readily absorbed in the G.I. tract. Thus, in the absence of adequate data, 100 percent absorption appears to be a reasonable assumption for 2,3,7,8-TCDD bioavailability from drinking water.

A.2.6. Conclusions

Table A-6 presents the bioavailability estimates for ingested 2,3,7,8-TCDD from various media, based on the information presented or assumptions made in the foregoing discussion.

A.2.7. References

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Table A-6. Bioavailability of Ingested 2,3,7,8-TCDD from Various Media

Medium	Bioavailability (% of oral dose)
Drinking water	100
Fatty or oily foods (e.g., milk, fish, meats)	85-95
Other foods	60-70
Paper dust	60-70
Sludge	60-70
Soil	45-55

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Umbreit TH, Hesse EJ, Gallo MA. 1988b. Bioavailability and cytochrome P-450 induction from 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) contaminated soils from Times Beach, Missouri, and Newark, New Jersey. Drug and Chemical Toxicol. 11: 405-418.

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A.3 BIOAVAILABILITY OF INHALED VAPORS AND PARTICLES CONTAINING 2,3,7,8-TCDD and 2,3,7,8-TCDF

A.3.1 Introduction

The purpose of this section is to provide a set of common assumptions that can be used for assessing bioavailability of inhaled 2,3,7,8-TCDD and 2,3,7,8-TCDF. This section was compiled from:

NCASI. National Council of the Paper Industry for Air and Stream Improvement. 1988. Risks Associated with Dioxin Exposure Through Inhalation of Paper Dust in the Workplace. Technical Bulletin No. 537. January, 1988.

and

DiCarlo F. 1989. Dioxins in Paper Products: Bioavailability by Inhalation. Memorandum from F. J. DiCarlo (EPA, Office of Toxic Substances) to G. Schweer (EPA, Office of Toxic Substances). June 16, 1989.

and

Griffin S. 1989. Inhalation Exposure to Dioxin Particles and Vapors. Memorandum from S. Griffin (EPA, Office of Solid Waste) to G. Schweer (EPA, Office of Toxic Substances). July 27, 1989.

and

Abt Associates. 1989. Multimedia Exposure Assessment for Re-use and Disposal of Sludge from the Pulp and Paper Industry and Disposal of Paper Products. Final Draft Report. Prepared for EPA, Office of Toxic Substances. September 29, 1989. Sect. 2.4.4, pp. 154-171.

The bioavailability of inhaled chemicals is dependent on the physical and chemical characteristics of the inhaled chemical and on the anatomy and physiology of the respiratory tract. The deposition and retention of inhaled chemicals are a function of the anatomic features of the respiratory tract including the alveolar surface area, the structure and spatial relationships of conducting airways leading into the alveoli, and the overall lung volume. Particle deposition and absorption are also a function of the respiratory rate and tidal volume. Inhaled gases, vapors, and aerosols are different, however, and can be absorbed throughout the respiratory tract, primarily by diffusion. Gases and vapors are generally absorbed rapidly from the respiratory tract, although the solubility of the gas or vapor in water is an important characteristic in determining the relative rate of transport from the gaseous phase into the liquid phase lining the respiratory tract.

A.3.2 Inhalation of Vapors and Bioavailability

No toxicokinetic data are available for assessing the bioavailability of inhaled dioxin vapors. However, it may be assumed that, due to their high lipophilicity, there would be 100 percent absorption of the 2,3,7,8-TCDD or 2,3,7,8-TCDF vapors entering the respiratory tract (DiCarlo 1989, Griffin, 1989).

A.3.3 Particulate Inhalation and Deposition

The probability of inspiration of a particle is a function of its size. This relationship indicates that the larger the aerodynamic diameter, the lower the "efficiency" of inhalation (probability of inhalation into the alveolar spaces). Once a particle is inhaled, its ultimate fate is again a function of its size (NCASI 1988). Four different modes of

deposition may occur. First, a particle may deposit in the head and neck regions of the airways. This deposition is termed extra-thoracic deposition (ETD). Second, particles which pass through the head and neck airways may deposit in the thoracic regions of the lung, the trachea to the terminal bronchi. This deposition is tracheobronchial deposition (TD). Third, particles which pass through the tracheobronchial airways may deposit in the respiration regions of the lung, the alveolar or gas-exchange regions. This is respiratory deposition (RD). Fourth, some particles may not deposit in any region of the lung and are exhaled (NCASI 1988).

The particulate deposition pattern and rates for nose breathing are different than for mouth breathing. Particles entering through the nose have ETD which is divided between the anterior (front) portion of the nose and the main nasal passages. With nose breathing, complete deposition can be expected for particles larger than 4 μ, and of these particles deposited, 80 percent impact in the anterior nasal passages and 20 percent impact in the main nasal passages (NCASI 1988).

The deposition pattern for particles up to 100 μ can be calculated for mouth breathing. Particle-size normalized deposition for particles of (a) da < 2 μ, (b) 2 μ < da < 10 μ and (c) 10 μ > da having combined ETD + TD or RD have been calculated and are presented in Table A-7.

In a similar manner, the deposition pattern for nose breathing has also been calculated (Table A-7). For nose breathing, ETD, TD, or RD were calculated to determine the particle-size normalized deposition for particles (a) da < 2 μ, (b) 2 μ < da < 10 μ and (c) 10 μ > da.

Just as the site of particle deposition varies with particle size and the type of breathing (mouth vs. nose), the fate of the particle varies with the site of deposition. For mouth breathing, particles having ETD impact in the mouth or throat and are swallowed, resulting in an ingestion route of exposure. Particles having TD are cleared from the lung via the muco-ciliary escalator (the mechanism of clearing mucus from the lung) into the throat, and are ultimately swallowed. Particles having RD could have either of three fates. They either (1) remain deposited in the alveoli (the gas-exchange region of the lung) or are phagocytized (engulfed) by macrophages (cells of the immune system) and (2) enter the lymphatic system or (3) are cleared via the mucociliary escalator and are swallowed.

For nose breathing, particles having TD are ingested and those with RD have pulmonary deposition, the same fate as for mouth breathing. However, the fate of particles having ETD is different. ETD in the main nasal passages results in ingestion of these particles. ETD in anterior regions of the nose results in particles being trapped in nasal mucus and being

Table A-7. Size-Weighted Average Particulate Deposition

Size range (u)	Average size (u)	ETD + TD (%)	ETD (%)	TD (%)	RD (%)
Mouth Breathing					
10-100	55	52.4			0
2-10	6	69.6			12.2
0-2	1	18.7			53.1
Nose Breathing					
10-100	55		52.4	0	0
2-10	6		78.7	2.17	2.36
0-2	1		43.9	6.93	47.9

Source: NCASI (1988).

cleared by mechanical clearing mucus from the nose (nose wiping, blowing the nose, or sneezing). Little systemic absorption of toxicants is expected from deposition in the anterior portions of the nasal passages because of the thickness of the mucus, the limited blood perfusion, and short residence time, about 12 hours. Thus, any nose breathing during the period of exposure to particulates decreases the ingestion of particulates and the subsequent dioxin exposure.

A.3.4 Bioavailability of Particulate-Bound 2,3,7,8-TCDD and 2,3,7,8-TCDF

The bioavailability of 2,3,7,8-TCDD and 2,3,7,8-TCDF via particulate inhalation is a function of the pathway of uptake, nasal deposition, ingestion or pulmonary deposition, and also the relative rate of adsorption into the body. However, there is no experimental data on bioavailability of dioxin from paper dust (or any cellulosic matrix) by any route of exposure nor is there any data for bioavailability from sludge or soil inhaled from the air.

Based on the limited information available, DiCarlo (1989) utilized two simplifying assumptions for estimating the bioavailability of dioxins from inhaled particles. The first is that dioxins on the inhaled smaller particles (up to 10 μ in diameter) are almost completely absorbed (i.e., nearly 100 percent uptake). This assumption can be justified because the smaller particles in this range will reach the alveoli where they may either remain for long periods of time (allowing complete absorption of dioxins) or undergo engulfment by macrophages followed by transport to the pharynx where they can be swallowed with sputum. The larger particles in the 2 μ < da < 10 μ range will be deposited in the tracheobronchiolar region of the lung, then carried to the pharynx by the mucociliary escalator and swallowed.

The second simplifying assumption utilized by DiCarlo (1989) was that about 25 percent of the dioxins in the larger particles (>10 μ) are absorbed. This assumption can be justified because the particles will be deposited in the nasopharyngeal region from which about half will be exhaled or mechanically removed from the body by sneezing or nose blowing and about half of the particles may be swallowed; about half of the PCDDs and PCDFs on these ingested particles will then be absorbed.

A.4 UPTAKE IN TERRESTRIAL PLANTS

The purpose of this section is to provide plant uptake ratios for 2,3,7,8-TCDD for the edible portions of various types of plants. This section was compiled from:

Sargeant A. 1989. Dioxin Uptake in Terrestrial Plants. U.S. EPA,

Office of Research and Development. Memorandum to C. Cinalli, EPA, Office of Toxic Substances, June 21, 1989.

Aboveground crops and root crops are treated separately, since the literature suggests that uptake factors differ. Although it also appears that uptake into reproductive structures (fruits, seeds) is lower than in other aboveground parts, the available data are not sufficient to quantify this difference. Therefore, all aboveground portions are treated as one group.

A.4.1 Root Crops

A range of root-to-soil ratios can be estimated for fleshy root crops based on the literature for 2,3,7,8-TCDD, PCBs, and pesticides. The root-to-soil ratios described below are expressed as the wet-weight concentration of 2,3,7,8-TCDD in the crop divided by the dry-weight concentration of 2,3,7,8-TCDD in the soil.

It is assumed that the root vegetables will be scrubbed but not peeled before consumption. This is an important assumption, since many researchers have found higher concentrations of hydrophobic organic chemicals in the peels of root crops (Iwata et al. 1974, Sacchi et al. 1986, Wipf et al. 1982). A summary of the estimated root-to-soil ratios is presented in Table A-8.

As can be seen in Table A-8, the root-to-soil ratios ranged approximately three orders of magnitude, from 0.003 to 3. The range of ratios can be reduced somewhat by considering data on outer tissues separately from data on the whole root. For outer tissues or peels, the range of values is from 0.2 to 2, with root-to-soil point estimate of 2. It should be noted, however, that peel-to-soil ratios reported by Wipf et al. (1982) are well below this range.

For whole roots, the range of values was from 0.01 to 1, with a root-to-soil point estimate of 0.5. This range includes the higher end of some reported ratios (Wipf et al. 1982), but the value of 2.83, reported by Moza et al. (1979) for uptake of 2,4',5-trichlorobiphenyl, is not included in the range. 2,4',5-trichlorobiphenyl has a lower K_{ow} than 2,3,7,8-TCDD, while the other PCB congener included in the same study, 2,2',4,4',6-pentachlorobiphenyl, has a K_{ow} more comparable to 2,3,7,8-TCDD, was found to be taken up to a lesser extent than the 2,4',5-trichlorobiphenyl congener, and was included in the range of values used.

Root-to-soil ratios will depend on characteristics of both the soil and root. Because these characteristics were not quantitatively considered, the root-to-soil ratios presented may over- or underestimate uptake occurring in the field for the following reasons:

Table A-8. Root-to-Soil Ratios for 2,3,7,8-TCDD and PCBs

System	Chemical	Plant	Root:soil ratio[a] Inner root	Whole root	Outer root[b]	Reference
Pots	TCDD	Carrots	0.0015 to 0.0075	0.003 to 0.015	0.01 to 0.05	Wipf et al. 1982
Pots	TCDD	Maize	NA[c]	NA	0.98 to 1.8	Facchetti et al. 1986
Field	TCDD	Beans	NA	NA	1.5 to 2.4	
		Carrots	0.69	0.14	1.43	Cocucci et al. 1979
		Potatoes	0.35	0.019	0.19	
		Onions	0.56	0.56	0.55	
		Narcissus	0.42	0.43	0.49	
Field	PCBs	Carrots	NA	0.03 to 0.5	NA	Iwata & Gunther 1976
Pots	PCBs	Carrots	NA	0.92 to 2.83	NA	Moza et al. 1979
	PCBs	Sugar beets	NA	0.061 to 0.16	NA	
Hydroponic	TCDD[d]	Barley	NA	0.015 to 0.24	0.11 to 1.78	Briggs et al. 1982

[a] Ratios are expressed as concentration per wet weight of root divided by concentration per dry weight of soil.

[b] Ratios calculated for peels, outer portions of fleshy roots, and fibrous roots are included under this heading.

[c] NA = not available.

[d] Ratio for TCDD was calculated based on a correlation derived using pesticides.

- Attributes of the roots that may influence uptake will include their oil content and surface-to-volume ratio. Ratios calculated from studies using fibrous roots, because of their high surface-to-volume ratio, were assumed to be more reflective of the peels of fleshy roots.

- Roots grown in hydroponic systems were assumed to adequately represent roots grown in soil.

- Soil attributes that will influence uptake include organic carbon content, water content, and particle size distribution. However, many investigators did not report information concerning these soil parameters.

Limited data were available describing uptake of 2,3,7,8-TCDD by root crops. To expand the number of studies on which to evaluate root-to-soil ratios, experiments using PCBs and a correlation based on pesticides were included in the analysis. Not all data in the literature were reported as wet weight concentration in tissue divided by dry weight concentration in soil. When necessary, the ratios were converted to comparable units using standard assumptions for water content of soil. In addition, an attempt was made to use the data on peels and on inner tissues to calculate ratios for the whole root. This was done by assuming the peel accounts for 14 percent of the weight of whole carrots (Iwata et al. 1974) and 10 percent of the weight of other vegetables such as potatoes and sugar beets (USDA 1975). Finally, because of their higher surface-to-volume ratios, the root-to-soil ratios calculated for fibrous roots were assumed to be most comparable with the outer tissues of fleshy roots.

Wipf et al. (1982) reported 2,3,7,8-TCDD concentrations in carrots grown in contaminated soil. Uptake factors ranged from 0.0015 to 0.0075 for the peeled carrots, and 0.01 to 0.05 in peels. The organic carbon content of the soil was not reported. Using the assumption of Iwata et al. (1974) that the peel accounts for 14 percent of the weight of the whole carrot, whole root-to-soil ratios would range from 0.003 to 0.015.

Somewhat higher concentrations of 2,3,7,8-TCDD have been found by Fachetti et al. (1986) in the roots of maize and soybeans than in the surrounding soil. It should be noted that root-to-soil ratios were much higher in "blank" pots. Because these higher root-to-soil ratios may be a result of analytical limitations, only data from the experimental pots were used to this analysis. Root-to-soil ratios from these pots ranged from 0.98 to 2.4.

Concentrations of 2,3,7,8-TCDD in vegetables grown in the Seveso area of Italy have been reported by Cocucci et al. (1978). Outer tissues of carrots, potatoes, and onions did not consistently contain higher residues than inner tissues. This study should be treated with some caution,

because the results as described in the text do not agree with the data provided in their table. The ratios were calculated based on the data presented in the table. The ratios were reported as fresh weight of soil, so the ratios were converted to dry weight by assuming that the soil was at field capacity, and hence 17 percent water. Root-to-soil ratios for 2,3,7,8-TCDD for inner tissues were 0.69, 0.35, 0.56, and 0.42 for carrots, potatoes, and onions, and narcissus, respectively, and the corresponding ratios for outer tissues were 1.43, 0.19, 0.55, and 0.49, respectively. These data were used to calculate a ratio for whole roots by assuming that the outer tissues comprised 10 percent of the weight of the whole root. The resulting whole-root-to-soil ratios were 0.76, 0.33, 0.56, and 0.43 for carrots, potatoes, onions, and narcissus, respectively.

Iwata and Gunther (1976) grew carrots in PCB-contaminated field plots having very low organic carbon content (0.35 percent). The whole-root-to-soil ratios ranged from 0.03 to 0.50. Although specific congeners were not identified, the uptake factors decreased with increasing chlorination.

Moza et al. (1979) grew carrots and sugar beets in outdoor boxes containing either 2,4'5-trichlorobiphenyl (TCB) or 2,2',4,4',6-pentachlorobiphenyl (PeCB). Whole root-to-soil ratios for the TCD ranged from 0.16 for sugar beets to 2.83 for carrots. Uptake of the PeCB was lower; root-to-soil ratios ranged from 0.061 for sugar beets to 0.92 for carrots. Moza et al. (1979) suggested that uptake into carrots would be expected to be greater than sugar beets because of the higher oil content of carrots.

Briggs et al. (1982) studied a variety of pesticides in a hydroponic system using barley, and the uptake of organic chemicals into barley roots from solution was very dependent on the octanol:water partition coefficient (K_{ow}) of the chemical. The wet-weight root concentration factor (RCF) was defined as the concentration of the chemical in the root divided by the concentration of chemical in solution surrounding the root, and was related to K_{ow} by the following equation:

$$\log (RCF - 0.82) = 0.77 \log K_{ow} - 1.52$$

where

RCF = root:soil solution partition coefficient (mg/kg root divided by mg/kg solution)
K_{ow} = chemical-specific octanol:water partition coefficient.

According to this equation, only those chemicals that are dissolved in solution in the interstitial spaces between soil particles are available for uptake by plants. The concentration of chemicals in the soil solution can be calculated (for equilibrium conditions) using a simple partition model:

$$Csol = \frac{Cs}{(K_{oc})\ (f_{oc})}$$

where

Csol = concentration of chemical in soil solution (mg/l)
Cs = concentration of chemical in soil (mg/kg)
K_{oc} = organic carbon partition coefficient
f_{oc} = fraction organic carbon in soil.

Combining these two equations, a root:soil partition coefficient (uptake factor or UF) can be calculated using the following formula:

$$UF = \frac{RCF}{(K_{oc})(f_{oc})} \qquad \text{(A-8)}$$

In order to solve this equation, values are needed for K_{ow}, K_{oc}, and f_{oc}. It was assumed that log K_{ow} of 2,3,7,8-TCDD ranged from 6.15 (Schroy et al. 1985) to 7.02 (Burkhard and Kuehl 1986), log K_{oc} was 5.68 (Schroy et al. 1985), and soil organic carbon content ranged from 0.9 to 3 percent (Brady 1974).

Using these variables, the estimated uptake factor for 2,3,7,8-TCDD ranges from 0.11 to 1.78. There are some important limitations in using this information for estimating uptake of 2,3,7,8-TCDD by root crops. First, barley roots are fibrous and may not accurately represent uptake into fleshy roots, such as carrots or potatoes. Second, the chemicals studied were mostly organic pesticides, none of which were as hydrophobic as 2,3,7,8-TCDD. Finally, it must also be assumed that roots grown hydroponically adequately represent roots grown in soil.

If it is assumed that the uptake factor applies only to the peel of root vegetables, and that the peel is 14 percent of the whole carrot weight, estimated uptake factors for the whole carrot would range from 0.015 to 0.25. The lower end of this range corresponds to soils having high organic content, and the upper corresponds to soils of low organic content.

A.4.2 Aboveground Crops

USEPA (1988) uses a preliminary estimate of 2 percent for the plant-to-soil ratio for 2,3,7,8-TCDD for aboveground crops, based primarily on the data presented in Sacchi et al. (1986) and Wipf et al. (1982). There

does not appear to be any additional recent work to support modification of this factor. However, this factor could under- or overestimate uptake in aboveground portions of plants for the following reasons:

- Some studies have found plant-to-soil ratios much higher than 2 percent (although most of these were connected with the Seveso accident), so the 2 percent factor may underestimate concentrations.

- 2,3,7,8-TCDD was not detected (detection limit 1 ppt; soil level ~10,000 ppt) in edible aboveground portions (fruits and grain) of plants (Wipf et al. 1982), which suggests that a 2 percent ratio may overestimate actual concentrations.

- In contrast, Sacchi et al. (1986) found significant 2,3,7,8-TCDD uptake in aboveground portions of maize and soybeans, which suggests that the compound may indeed be translocated, but will partition unevenly between leaves, fruits, and grain.

- Many workers are convinced that volatilization is a significant fate of 2,3,7,8-TCDD in the soil environment. Because the compound is very lipophilic, it is possible that 2,3,7,8-TCDD volatilizing from the soil could adsorb onto plant cuticles and perhaps enter leaves via stomates. Most experiments were not designed to distinguish between translocation and volatilization as separate aboveground contamination pathways.

- Another source of uncertainty comes from the use of radio labeled 2,3,7,8-TCDD. Stock solutions are typically contaminated with unknown compounds at a rate of about 1 percent. If the unknown compounds happen to be substances that are readily taken up and translocated, assays that rely on scintillation counting alone will not provide an accurate measure of the specific distribution of 2,3,7,8-TCDD in the plant. It is possible to validate scintillation with additional GC-MS analysis, but this procedure was not always included in experiments.

- Hydroponic studies, while having advantages, may complicate analysis because of the difficulty of keeping 2,3,7,8-TCDD in solution. The chemical is so hydrophobic that it sorbs onto the surfaces of plants and containers in a matter of minutes after being added to the growth solution.

- Finally, it appears that 2,3,7,8-TCDD becomes more strongly adsorbed to soil particles with time. Therefore, studies using soils spiked in the laboratory with 2,3,7,8-TCDD probably overestimate the amount of the soil-to-plant partitioning that would occur under field conditions.

In experiments with maize and soybeans, Sacchi et al. (1986) reported that plants grown in soil accumulated increasing amounts of 2,3,7,8-TCDD with increases in both time and soil concentration, with uptake ratios of about 2 percent. In addition, plants grown hydroponically seemed to take up TCDD via the transpirational stream, based on the observation that plants kept in the dark and treated with transpiration inhibitors did not takeup 2,3,7,8-TCDD. Unfortunately, workers at EPA's Corvallis laboratory have been unable as yet to reproduce the hydroponic portion of this study, possibly casting some doubt on the soil portion of the study.

Vegetation growing near the ICMESA chemical plant in Seveso, Italy, where a large quantity of dioxin was accidentally released, has been analyzed by Cocucci et al. (1978). 2,3,7,8-TCDD concentrations were highest in twigs, then cork, then leaves, and lowest in fruit. These data suggest transpirational flow to, and subsequent elimination from, newly-formed organs. Other possible explanations for the observed distribution (e.g., dust deposition on plant surfaces) were not discussed.

Maize and soybeans grown in pots have been analyzed by Facchetti et al. (1986). Based on the fact that TCDD was volatile at ambient temperatures, Facchetti et al. (1986) theorized that TCDD contamination present in aboveground portions of plants was the result of volatilization from contaminated soil.

One early study by Isensee and Jones (1971) includes oats and soybeans grown in soil and a variety of spiked solutions, including TCDD at 0.06 ppm in a benzene carrier. TCDD uptake from solution reached its maximum in 24 hr and gradually decreased with time. It was theorized that movement, volatilization from tissue, and translocation back to roots could all account for this removal. With regard to uptake from soil, tissue content decreased as age of the plant increased; this was attributed to tissue dilution. On potential limitation of this work is that "Tween," a surfactant, was used to keep the 2,3,7,8-TCDD in solution. This compound decreases the ability of 2,3,7,8-TCDD to bind to plant roots and promotes a great deal of bacterial growth, which may also affect 2,3,7,8-TCDD behavior in solution.

Another early study by Helling et al. (1973) concluded that plant uptake and translocation were "highly unlikely." However, the analytical detection limits (about 1 ppb) may not have been low enough to detect uptake.

Another early study by Kearney et al. (1973) concluded that TCDD is immobile in soils, not readily taken up by plants, persistent in soils, subject to photodegradation, and slowly degraded in soils to polar metabolities. Foliar applications were conducted, and there was no translocation from leaves. Very little was lost from soybean leaves, but loss was gradual from oat leaves.

Jensen et al. (1983) conducted a study to determine the potential impacts of 2,4,5-T used on rice crops. No 2,3,7,8-TCDD was found in rice grain (detection limits were 2-7 ppt), either in treated rice or in rice purchased in locations throughout the United States.

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APPENDIX B

Summary Information on Chlorinated Chemicals Other than PCDDs and PCDFs (OCOs) Identified in Pulp Mill Effluents, Sludges, and Pulps

TABLE OF CONTENTS

	Page No.
B.1 Introduction	B-1
B.2 Master List of Other Chlorinated Organic Chemicals (OCOs) Identified in Pulp Mill Effluent	B-1
B.3 Screening-Level Human Health Data	B-2
B.4 Screening-Level Environmental Hazards from Chlorinated Organic Chemicals Other than PCDDs and PCDFs Released by Pulp/Paper Mills	B-3
B.4.1 Hazards to Aquatic and Terrestrial Organisms Posed by Other Chlorinated Organic Chemicals (OCOs)	B-4
B.4.2 Risks Posed by the Release of Effluents Containing Other Chlorinted Organic Chemicals (OCOs)	B-4
B.4.3 Conclusions	B-4
B.5 Screening Level Human and Aquatic Exposure to Other Chlorinated Organic Chemicals (OCOs) in Treated Pulp/Paper Mill Effluent	B-5
B.6 Temporal Data - Ten-Month, Bi-Daily OCO Concentrations in Pulp Mill Effluent	B-6
B.7 Activities and Regulatory Approaches of Various Governmental Agencies Regarding Other Chlorinated Organic Chemicals Released into the Environment from Pulp/Paper Mills	B-6
B.7.1 Summary of the Office of Water Regulations and Standards' (OWRS) Regulatory Approach	B-7
B.7.2 Summary of the Office of Air Quality Planning and Standards (OAQPS) Activities Concerning Emissions of Chloroform from Pulp/Paper Mills	B-8
B.7.3 Summary of the Office of Toxic Substances' Toxic Release Inventory (TRI) Data Regarding OCO Emissions from Pulp/Paper Mills	B-8
B.7.4 Summary of European and Canadian Regulatory Approaches	B-9
B.8 Conclusions and Recommendations	B-10
B.9 References	B-11

LIST OF TABLES

		Page No.
Table B-1.	Master List of the Chlorinated Compounds Other Than 2,3,7,8-TCDD/F Found in Pulp Mill Effluents	B-14
Table B-2.	Other Chlorinated Organic Chemicals Cancer Risk	B-20
Table B-3.	Other Chlorinated Organic Chemicals Drinking Water Health Advisories	B-21
Table B-4.	Other Chlorinated Organic Chemicals Reference Doses	B-22
Table B-5.	Species List	B-23
Table B-6.	Toxicity of Other Chlorinated Organics from Bleached Wood Products to Aquatic Species	B-24
Table B-7.	Ranking by Acute Toxicity to Aquatic Organisms	B-31
Table B-8.	Acute Toxicity of Selected Chlorinated Organic Chemicals (OCO) to Terrestrial Organisms	B-32
Table B-9.	Summary of Treated Effluent Chemical Concentrations	B-33
Table B-10.	Statistical Analysis of Temporal Changes in Chemical Concentrations in Pulp Mill Effluent	B-35
Table B-11.	Regulatory Schedule for OCOs in Some Canadian Provinces and European Countries	B-36

LIST OF FIGURES

		Page No.
Figure B-1.	10th% Low Flow Stream Conc vs. Eff Conc	B-37
Figure B-2.	50th% Low Flow Stream Conc vs Eff. Conc	B-37
Figure B-3.	Average LADDs at 10th % Mean Flow	B-38
Figure B-4.	Average LADDs at 50th % Mean Flow	B-38
Figure B-5.	Temporal Changes in 3,4,5-Trichlorocatechol Conc. .	B-39
Figure B-6.	Temporal Changes in Pentachlorophenol Conc.	B-40

APPENDIX B

B.1 Introduction

Risks from exposure to 2,3,7,8-TCDD and 2,3,7,8-TCDF have been the primary focus of this Integrated Exposure/Risk Assessment. Prior to the discovery of PCDDs and PCDFs in the effluents and products of the pulp and paper industry, other chlorinated organic compounds (OCOs), such as chloroform and chlorinated phenols, had already been identified (Suntio et al. 1988; Sodergren et al. 1988; USEPA 1988; USEPA 1987). Even though these chemicals may be present individually at relatively low concentrations in pulp mill effluents, sludges, and pulps, the cumulative effect of PCDDs, PCDFs, and the OCOs on exposed populations (human, aquatic life, and wildlife) could be significant. In order to enable informed risk management decisions with regard to the pulp and paper industry, it is important to understand at least on a qualitative basis, the identity, quantity, impacts, and risks of other chlorinated organic chemicals present in pulp/paper mill wastes and products.

The purpose of this appendix is to present screening level analysis of OCOs using monitoring data obtained from industry and the literature and, also, to describe various governmental action regarding OCOs released by the pulp and paper industry. The screening level analysis includes the compilation of readily available information on the properties and toxicity of the individual chemicals and generic exposure estimates for those chemicals where treated effluent data are available.

This appendix is structured to first provide in section B-2, a master list of the other chlorinated organic chemicals (Table B.1) that have been found in pulp mill wastes/products. Section B-3 presents screening level human health data and section B-4 contains a discussion and presentation of the readily available data on the aquatic and terrestrial toxicity of the OCOs. Screening level estimates of stream concentrations and drinking water exposure are given in section B-5. The graphs presented in Section B.6 show how certain chemical concentrations varied during a ten month study of treated pulp mill effluent. Section B.7 presents a summary of governmental activities on OCOs released by the pulp and paper industry. Lastly, conclusions and recommendations concerning OCOs and the pulp and paper mill industry are made.

B.2 Master List of Other Chlorinated Organic (OCO) Chemicals Identified in Pulp Mill Effluent

Table B-1 is a master list of chlorinated compounds other than PCDDs/PCDFs which have been found in pulp mill wastewater. No information was readily available on OCOs in pulp or wastewater sludge.

Of the 263 chemicals/chemical families on the master list, treated effluent concentration data are available for 31, human health effect information is readily available for 23, aquatic toxicity data are readily available for 45, and terrestrial toxicity data are readily available for 10. The last column, "Data Code," lists the readily available information for each chemical. The master list is partitioned into acidic, phenolic, and neutral categories for which there were 64, 63, and 130 listings, respectively. There are six entries of general chemical families (i.e., chlorinated phenols, etc.) at the end of the neutrals category.

B.3 Screening Level Human Health Data

A CAS number search of the IRIS data base in May 1989 showed that there are human health data for 23 of the chemicals on the master list. The IRIS data base lists all chemicals that EPA has reviewed and given an official agency evaluated reference dose, cancer slope factor, or other toxicity evaluation. The 23 chemicals with health data listed in IRIS are identified in Table B-1 by an "H" in the "Data Code" column.

Table B-2 shows the cancer risk associated with these chemicals. The definitions for the various cancer classification codes can be found in the Guidelines for Carcinogen Risk Assessment published by EPA. The slope factor, q_1^*, is taken as the 95% upper-bound of the potency of the chemical in inducing cancer at low doses, where the upper confidence limit for the extra risk calculated at low doses is always linear. The unit risk for an air or water pollutant is defined as the lifetime cancer risk occurring in a hypothetical population in which all individuals are continuously exposed from birth throughout their lifetimes (70 years) to a concentration of 1 $\mu g/m^3$ of the agent in the air they breath, or to 1 $\mu g/l$ in the water they drink. The unit risk calculation assumes the individual in the hypothetical population weighs 70 kg, drinks 2 liters of water per day, and breaths 20 cubic meters of air per day.

Table B-3 lists values for drinking water standards and health advisories. Maximum contamination limits (MCLs) are enforceable standards that must be met in finished drinking water supplies. Several of the values in this table are only proposed values and are not considered as final rulings. Water quality criteria for the protection of human health can be used by the states to develop waste load allocations for surface water dischargers. There are also ambient water quality criteria for the protection of aquatic life (not listed here). The water quality criteria values given here are revised values that will not be published until May 1990. These values are still undergoing review and they may change when finally published.

The drinking water health advisories represent guidance levels for drinking water and were developed from data describing noncarcinogenic toxicity endpoints. The values for the one-day, ten-day, and longer-term

exposure periods do not consider other exposures such as food and air, and therefore assume that 100 percent of the individual exposure comes from drinking water. The lifetime value is calculated only for adults but does consider other sources of exposures. The Drinking Water Equivalent Level (DWEL) adjusts the oral RfD for a 70 kg adult who drinks 2 liters of water a day. The DWEL assumes that 100 percent of an individual's exposure to that contaminant comes from their drinking water.

Table B-4 lists the available Reference Doses (RfD). Reference doses are based on noncarcinogenic toxicity endpoints. The RfD is a peer-reviewed estimate of the time-weighted average daily exposure to the human population that is likely to be without appreciable risk of deleterious results during a lifetime. The Uncertainty Factor (UF) consists of divisors of multiples of 10 and is assigned according to guidelines. Each uncertainty factor represents a specific area of uncertainty inherent in the available data; for example, these factors take into account the differences in responsiveness between animals and humans, and between individuals in the exposed population.

B.4 Screening Level Environmental Hazards from Chlorinated Organic Chemicals Other than PCDDs and PCDFs Released by Pulp/Paper Mills

(NOTE: This subsection was extracted from a memorandum from Robert E. Morcock, Health and Environmental Review Division, U.S. Environmental Protection, to Thomas M. Murray, Exposure Evaluation Division, U.S. Environmental Protection Agency, dated September 29, 1989.)

The ACQUIRE data base (USEPA 1984) (an aquatic information retrieval system) and the RTECS (Registry of Toxic Effects of Chemical Substances) were used in developing information on hazards posed by OCO chemicals. In general, data were based on studies conducted in the United States. However, two pentachlorophenol studies incorporated results from foreign studies on amphibians. Of the U.S. studies, only records with reliability codes of "1" ("meets all criteria") or "2" ("meets some criteria") were examined. Studies with rating "1" have followed all established laboratory procedures. A rating of "2" represents studies that are generally satisfactory, but one variable may not have been reported or measured (e.g., water chemistry variables are not reported or toxicant concentration was unmeasured). The codes were established by the EPA Environmental Research Laboratory in Duluth. In addition, the potential impact of nine arbitrarily selected chemicals on terrestrial organisms was examined.

All species were considered to be relevant indicators and the lowest concentration at which effects were reported was used. Ranking by toxicity was based on acute and/or chronic studies on thirty-two species. Table B-5 lists species reported in all of the available studies.

B.4.1 Hazards to Aquatic and Terrestrial Organisms Posed by Other Chlorinated Organic Chemicals (OCOs)

Table B-6 gives a summary of available data on the toxicity of other chlorinated organic chemicals to aquatic organisms. The chemicals listed in Table B-6 are limited to those with LC_{50} data in ACQUIRE (USEPA 1984). Of the chemicals examined, two exhibit little toxicity: trichloroacetic acid and tetrachloro-acetic acid (2,000 mg/l) (Tadken and Lewis 1983). Phenols were the most toxic (LC_{50}); pentachlorophenol, 0.023 mg/L and tetrachlorophenol, 0.14 to 0.17 mg/L. These data are summarized and ranked by toxicity in Table B-7.

Because of time constraints, the potential impacts of the chemicals on terrestrial organisms were only briefly evaluated. Like heavy metals, chlorinated organic chemicals are persistent in the soil. Uptake of chlorinated organic contaminants by plant roots may be an important biological exposure pathway and must be considered when evaluating risks from these compounds.

Table B-8 summarizes terrestrial toxicity data of ten selected chemicals.

B.4.2 Risks Posed by the Release of Effluents Containing Other Chlorinated Organic Chemicals (OCOs)

At present, available data regarding composition of complex effluents are insufficient in quantity and quality to enable their use in the development of quantitative risk assessments. In addition, synergistic effects have not been assessed. Finally, it is not practical (within the constraints of this project) to develop risk assessments for all of the chemicals covered in this appendix. Nevertheless, it is expected that there will be unacceptable risks to aquatic and terrestrial organisms where environmental concentrations approach toxic levels reported in this subsection. Table B-9 compares the available data on treated-effluent chemical concentration to the LC_{50}. The upper range of pentachlorophenol and chloroform treated-effluent chemical concentrations exceed the LC_{50} when fate is not considered.

As indicated in Table B-7, twenty-five chemicals exhibited LC_{50} values below 10 mg/L, eight between 10 and 100 mg/L, and five between 220 mg/L and 2,000 mg/L.

B.4.3 Conclusions

Information on environmental concentration and potential exposure is currently too sparse to make quantitative risk assessments, although potential impacts on both aquatic and terrestrial species conceivably may occur. More than 10 percent of the chemicals on the original list of 263

and 65 percent of the chemicals for which data were available, would have adverse effects if environmental concentrations were as low as 10 mg/L.

B.5 Screening Level Human and Aquatic Exposure to Other Chlorinated Organic Compounds (OCOs) in Treated Pulp and Paper Mill Effluents

In order to provide a qualitative sense of exposures to chemicals for which treated effluent data are available, a generic exposure scenario was developed for the pulp and paper mill industry. This scenario consists of treated effluent being discharged into a "generic" river thus exposing aquatic life directly and exposing humans through ingestion of contaminated drinking water. The generic approach has given an "order-of-magnitude" impression of potential exposures, which are depicted graphically in Figures B-1 through B-4. These figures can be used along with the data on concentration ranges of the treated effluent presented in Table B-9 to estimate stream concentrations of chemicals of interest under low and mean flow conditions. It is important to note that fate and transport processes (i.e., volatilization, biodegradation, adsorption, etc.) are not considered in this analysis. Therefore, this exercise provides only a screening level estimate. Also, it should be noted that the data provided in Table B-9 is dated and may or may not reflect actual effluent concentrations for each of the chemicals listed.

Generic exposure assessments are developed from stream flow and effluent flow data from numerous facilities within the same Standard Industrial Classification (SIC) code. For the purpose of this assessment, stream and effluent flows for 63 pulp and paper mills within SIC code 2611 were obtained from the GAGE and IFD files in EPA's STORET system. These data were used to generate a statistical distribution of the stream dilution factors under mean and low (7-Q-10) flow conditions. The 50th percentile and 10th percentile mean flow dilution factors were 358 and 3, respectively. Stream concentrations were then estimated by dividing the treated effluent concentration by the dilution factor for the flow condition of interest. The 10th and 50th percentile dilution factors were used to estimate exposure. The 10th percentile stream flow dilution factor was used to represent a reasonable worst case and the 50th percentile stream flow dilution factor represents the median dilution factor.

Low stream flow (i.e., 7-Q-10) dilution factors were used to estimate potential aquatic toxicity impacts (see Figures B-1 and B-2). Low flow stream data are typically used to calculate acute toxicity impacts to aquatic life since the exposure to the fish will be the highest. Mean stream flow dilution factors were used to estimate lifetime average daily exposure (LADE) to humans from drinking contaminated water. Again, 10th and 50th percentile stream flow dilution factors represent worst case and median exposures (see Figures B-3 and B-4). Estimates of drinking water

exposure are based on the assumption that a person consumes two liters of contaminated drinking water 365 days per year for 70 years.

Figures B-1 through B-4 were based on the highest treated effluent data for all chemicals (excluding chloroform, which was found at much higher levels than all other compounds and is expected to volatilize rapidly from water to air). These figures for aquatic and human drinking water exposure values are generic interpretations (e.g., non-chemical specific) of the data, since any chemical put into a stream will be equally diluted because the stream flow will be the same. Due to time limitations, fate and environmental transport properties for each of the chemicals listed in Table B-9 (other than dilution) could not be considered. Processes such as volatilization, sorption to sediment, solubility of the chemical in water, as well as others could considerably influence the concentrations of OCOs in surface water, sediment, and air.

B.6 Temporal Data - Ten-Month, Bi-Daily OCO Concentrations in Pulp Mill Effluent

In one study supplied to EPA by NCASI (NCASI 1985a), concentration data for 18 different chemicals were collected every other day for a 10-month period. Data for two of these chemicals are plotted in Figures B-5 and B-6 for illustration. Table B-10 shows the statistical analysis for the data. These data show the minimum and maximum concentrations vary by a factor of 2 to a factor of 60.

B.7 Activities and Regulatory Approaches of Various Governmental Agencies Regarding Other Chlorinated Organic Chemicals Released into the Environment from Pulp/Paper Mills

The pulp and paper industry has been under investigation by many government agencies both domestic and foreign in regards to their production and release of OCOs. Within the U.S. EPA, the Office of Water Regulations and Standards (OWRS) is conducting a thorough investigation of the pulp and paper industry to support the revision of the effluent limitations guidelines and standards for the pulp, paper, and paperboard industrial point source category, the Office of Air Quality Planning and Standards (OAQPS) has identified the pulp and paper industry as the largest emitter of chloroform, and the Office of Solid Waste is considering listing paper mill sludge as a hazardous waste based on the presence of dioxins.

Canadian officials have been studying the release of OCOs for some time and much of the monitoring data presented in this appendix was obtained from scientific papers published by Canadian researchers. Overseas, West Germany, Finland, and Sweden have regulated or announced intentions to regulate the pulp and paper industry on the basis of TOX (Total Organic Halogens) or AOX (Adsorbable Organic Halogens) per ton of air dried bleached pulp produced.

B.7.1 Summary of the Office of Water Regulations and Standards' (OWRS) Regulatory Activities

On January 2, 1990, the Office of Water Regulations and Standards issued a 304(m) notice announcing that the existing effluent limitations guidelines and standards for the pulp, paper, and paperboard industrial point source category will be revised. OWRS has established a Pulp and Paper Guidelines Review Workgroup and has devised a Development Plan and Schedule. The Development Plan includes the generation of data to support the revision of the effluent limitations and to investigate pollution prevention technology options to reduce the water discharges of conventional, toxic, and nonconventional pollutants. These pollutant categories include the OCOs as defined in this appendix. Therefore, the water release of OCOs by pulp and paper mill effluent will be regulated according to the new standards.

OWRS believes reduction in toxic and nonconventional pollutant loads from pulp and paper facilities may be best accomplished through alternative process technology rather than wastewater treatment. Furthermore, OWRS believes that process changes (e.g., use of chlorine dioxide and oxygenation instead of chlorine and hypochlorite to bleach the pulp and paper) should reduce air emissions, lower levels of toxics in solid waste, and reduce consumer exposure to pollutants in pulp and paper products. It is anticipated that through short and long term field sampling, completion of a Pulp, Paper, and Paperboard Industry Survey questionnaire, and an analysis of in-plant process changes, options will be identified to reduce the formation of dioxin and other pollutants of concern in pulp and paper manufacturing operations.

During rulemaking development, OWRS anticipates that data for more than 200 chemicals will be collected. Thus far, OCOs are the main pollutants identified for regulation. In addition to or instead of developing chemical-specific limitations and standards, OWRS is exploring the use of an alternate control parameter, such as TOX (Total Organic Halogens) or AOX (Adsorbable Organic Halogens). Some of the regulatory options approaches OWRS may choose to examine include: (1) limitations based on modifications to the process, (2) numerical limitations for a limited list of toxic pollutants (i.e., TCDD/TCDF, volatiles, OCOs, etc.), (3) regulation of toxics through the use of numerical limitations for surrogate parameters (i.e., TSS (Total Suspended Solids), TOX, and (4) whole effluent toxicity limits. OWRS anticipates the options selection for BPT (Best Practical Control Technology Currently Available) will be proposed by July 1993 (Red Border review completed by March 1993). The BAT (Best Available Technology Economically Achievable)/NSPS (New Source Performance Standards)/PSES (Pretreatment Standards for Existing Sources)/PSNS (Pretreatment Standards for New Sources) will be developed for the bleached kraft and sulfite mills only. Red border review will be complete by May 1995 and BAT/NSPS/PSES/PSNS will be promulgated by June 30, 1995.

B.7.2 Summary of the Office of Air Quality Planning and Standards (OAQPS) Activities Concerning Emissions of Chloroform from Pulp/Paper Mills

In 1985, a chloroform exposure and risk assessment was performed by OAQPS as part of a process initiated in 1984 to determine if chloroform should be listed as a Hazardous Air Pollutant under Section 112 of the Clean Air Act (USEPA 1985). Of the 11 major source categories for chloroform releases assessed in the report, the pulp and paper industry was identified as the largest category source of chloroform emissions. It was estimated to account for about 25 percent of the approximate 14,700,000 metric tons of chloroform emitted annually nationwide from all sources. The second highest emitter was estimated to be drinking water treatment facilities.

The maximum individual cancer risk estimated for the pulp and paper industry by this assessment was 1.1×10^{-2} and the annual aggregate cancer incidence was reported to be 0.76 cases/year. As the result of this assessment, a Notice of Intent to List chloroform under section 112 of the Clean Air Act was published in 1986. The pulp and paper industry raised concerns in 1986 regarding the modeling of chloroform emissions from wastewater ponds (lagoons) used in the assessment.

Since this initial assessment was performed, additional data have been obtained and a new model has been developed for estimating chloroform emissions from lagoons. OAQPS requested that pulp and paper plants submit new emissions data including data on process vent emissions of chloroform which had not been modelled previously. These data are currently being analyzed.

The new Clean Air Act is anticipated to be signed into law by Congress during 1990. Under the new law, OAQPS must promulgate emission standards for ten source categories within two years of enactment. Of all source categories for which emissions standards must be promulgated, the worst 25 percent must be done within four years (Cinalli 1989). The pulp and paper industry is highly likely to be included in the top 25 percent. Therefore, chloroform emissions from this industry are likely to be regulated within two to four years. During the process of regulating source categories, co-pollutants on the "Hazardous Air Pollutant List" must be considered as well; therefore, the emissions of other chlorinated compounds on the List may be regulated along with chloroform.

B.7.3. Summary of the Office of Toxic Substances Toxic Release Inventory (TRI) Data Regarding OCO Emissions from Pulp/Paper Mills

Under section 313 of the Superfund Amendments and Reauthorization Act (SARA) of 1986, all facilities must report releases of certain toxic chemicals to the EPA, Office of Toxic Substances. A list of approximately

300 toxic chemicals is defined under SARA Title III. Of the chemicals identified in Section B.2 of this appendix, 20 chemicals are on the SARA Title III list. A search of the Toxic Release Inventory (TRI) database was performed in November, 1989 for these 20 chemicals being released or transferred from facilities in SIC codes 2611 (pulp mills), 2621 (paper mills), and 2631 (paperboard mills). Only two chemicals, chloroform and 1,1,1-trichloroethane, were reported to be released from these facilities.

Chloroform releases to air, water, and land from pulp mills were prevalent and, in some cases, considerable (especially releases to air). Sixty-eight of the 76 facilities reporting chloroform releases are in the 104 Mill Study. The largest reported releases in 1987 to air, land and water according to the TRI database are as follows:

Largest single air release: 1,700,000 pounds per year
Releasing facility: Westvaco-Bleached Board (Covington, VA)

Largest single water release: 140,000 pounds per year
Releasing facility: Simpson Paper (Eureka, CA)

Largest single land release: 31,337 pounds per year
Releasing facility: Stone Container Corp. (Snowflake, Az)

Many other facilities reported large releases to all media; however, the largest estimated releases across the industry were to air. Considering the volatility of chloroform, the reported water releases will also ultimately result in air releases.

Additionally, two facilities from the 104 mill study reported releases to air of 1,1,1-trichloroethane on their 1987 Toxic Release Report. International Paper of Mobile, Alabama, reported releasing 16,000 pounds and the Mead Corporation in Chillicothe, Ohio reported releasing 499 pounds.

No other OCOs were reported released by the 104 mills. This is not surprising since the reportable quantity was 50,000 pounds per year for 1987. Also the SARA section 313 list contains less than 8 percent of the OCO chemicals identified in Table B.1.

B.7.4 Summary of European and Canadian Regulatory Approaches

Other countries have regulated or announced intentions to regulate the pulp and paper industry on the basis of TOCL (Total Organic Chlorine) or AOX (Adsorbable Organic Halogens) per ton of air dried bleached pulp produced. Table B-11 lists regulatory schedules for some Canadian provinces and European countries (Clodey 1989).

Due to the complexities of the composition of bleached mill wastes, many other countries have adopted generic measures to determine chlori-

nated organic compounds in various media. The following parameters have been suggested for water and effluents: ATOCL (Total Organically Bound Chlorine), AOX, and TOX (Total Organic Halogens). For sediments and tissues, the following parameters have been suggested: EOX (Extractable Organic Halogen), EOCL (Extractable Organic Chlorine), and EPOCL (Extractable Persistent Organic Chlorine). Use of the AOX method of analysis is becoming more widespread due to its ease of use, high precision, and low interference by inorganic chloride and ambient humic substances.

Pre-regulatory monitoring conducted by the Ontario Ministry of the Environment detected approximately 200 organic compounds in various wastestreams at a variety of pulp and paper mill (Ontario Ministry of the Environment 1989). Three Canadian provinces are establishing regulations for allowable AOX levels between 1.5 to 2.5 kg/ton air-dried pulp (see Table B-11).

The Swedish Pulp and Paper Research Institute (STFI) has positively identified 315 individual compounds in wastestreams from pulp bleaching operations and in whole mill effluent (Sodergren et al. 1988). Adverse biological effects of pulp and paper mill wastes on several Baltic Sea species has been documented by the Swedish Environmental Protection Board's Environment Cellulose Project. Based on these and other studies, the Swedish government has taken a protective approach and has limited the amount of OCOs released by regulating AOX to the lowest levels technologically possible.

B.8 Conclusions and Recommendations

Although there is a copious amount of information on chlorinated organic chemicals present in pulp mill effluent, the available information is not adequate to permit accurate assessments of human or environmental risk to the other chlorinated organics. In addition, no data are available on OCO concentrations in either sludge or pulp; thus, no attempt is made in this analysis to assess potential risks to humans or terrestrial organisms from exposure to sludge or pulp/paper. The estimates presented in this analysis are screening-level only and should only be used as a qualitative screening tool.

Some general observations can be made, however, on the OCO data. First, there are species of chemicals that show significant toxicity at low levels to aquatic life. The most toxic species are the chlorinated phenols. These compounds are typically found in concentrations from 4 to 15 μg/l in the treated effluents. At low stream flow conditions, this may result in concentrations that are within an order of magnitude of LC_{50} levels. Second, in nearly all cases, insufficient site-specific data exist to identify plants that may be discharging OCO chemicals at high levels. In addition, the available data are somewhat dated and therefore may not necessarily reflect changes in effluent

concentrations due to recent changes in bleaching processes. Third, concentrations of OCO chemicals in treated effluents are generally much higher than concentrations of PCDD and PCDF. OCO concentrations in treated effluents are typically two to three orders of magnitude higher than PCDD and PCDF concentrations. Fourth, it should be noted that paper mill effluents are complex mixtures. They contain not only PCDD and PCDF but also a wide range of organic compounds. This wide array of compounds may interact, thus further complicating the analysis of paper mill effluents. Furthermore, the effluent concentration of chloroform reported on Table B-9 exceeds the water criteria for human water and fish consumption as listed on Table B-3. Finally, as illustrated on Table B-9, pentachlorophenol and chloroform exceed the LC_{50}; therefore, additional sampling and analysis may be warranted on OCO compounds in paper mill effluents. This is warranted since, at this screening level, qualitative analysis indicates that potential risks from OCO chemicals may be significant.

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Table B.1
Master List of the Chlorinated
Compounds Other Than 2,3,7,8-TCDD/F
Found in Pulp Mill Effluents

CHEMICAL NAME	CAS No.	Chemical Formula	(C + CL)[a] no. N	(C)[b] no. C	M.W.[c]	Data[d] Code
Acidic Compounds						
Chloroacetic acid	79-11-8	C2H3O2CL	3	2	94.5	P
Dichloroacetic acid	79-43-6	C2H2O2CL2	4	2	128.95	E,P
Trichloroacetic acid	76-03-9	C2HO2CL3	5	2	163.4	E,P
Dichloropropenoic acid		C3H2O2CL2	5	3	140.95	Pm
2,3-Dichloropropenoic acid	13167-36-7					Pm
3,3-Dichloropropenoic acid	1561-20-2					Pm
Trichloropropenoic acid	2257-35-4	C3HO2CL3	6	3	175.4	Pm
Trichlorobutenoic acid		C4H3CL3O2	7	4	189.42	Pm
2,4,4-Trichlorobutenoic acid	19313-98-5					Pm
3,4,4-Trichlorobutenoic acid	21422-57-1					Pm
4,4,4-Trichlorobutenoic acid	22230-93-9					Pm
3,4,4-Trichloro-3-butenoic acid	21422-57-1					Pm
4,4,4-Trichloro-2-butenoic acid	2230-93-9					Pm
Tetrachlorobutenoic acid	98091-33-7	C4H2CL4O2	8	4	223.87	Pm
3,4,4,4-Tetrachloro-2-butenoic acid	98982-93-8					Pm
2,3,4,4-Tetrachloro-2-butenoic acid	76144-02-0					Pm
2,3,4,4-Tetrachloro-3-butenoic acid	2892-54-8					Pm
Dichlorostearic acid	31136-63-4	C18H34O2CL2	20	18	353.39	Pm
9,10-dichlorostearic acid	5629-48-1					E,D,Pm
Chloromalonic acid	600-33-9	C3H3O4CL	4	3	138.51	Pm
Chlorofumaric acid	617-42-5	C4H3O4CL	5	4	150.52	P,Pm
Chloromaleic acid	617-43-6	C4H3O4CL	5	4	150.52	P,Pm
Dichloromaleic acid	608-42-4	C4H2O4CL2	6	4	184.96	P,Pm
3-Chloromuconic acid	20665-95-6	C6H5O4CL	7	6	176.56	Pm
Chloro-3,4-dihydroxybenzoic acid		C7H5O4CL	8	7	188.57	Pm
2-Chloro-3,4-dihydroxybenzoic acid	87932-50-1					Pm
5-Chloro-3,4-dihydroxybenzoic acid	87932-49-8					Pm
6-Chloro-3,4-dihydroxybenzoic acid	87932-51-2					Pm
Dichloro-3,4-dihydroxybenzoic acid (3 isomers)	69846-51-8	C7H4O4CL2	9	7	223.01	Pm
2,5-Dichloro-3,4-dihydroxybenzoic acid	108098-45-9					Pm
Trichloro-3,4-dihydroxybenzoic acid	99185-96-5	C7H3O4CL3	10	7	223.02	Pm
Dichloro-4-hydroxybenzoic acid		C7H4O3CL2	9	7	207.02	Pm
2,5-Dichloro-4-hydroxybenzoic acid	35456-33-4					Pm
2,3-Dichloro-4-hydroxybenzoic acid	66584-09-6					Pm
3,5-Dichloro-4-hydroxybenzoic acid	3336-41-2					Pm
Chlorovanillic acid	62936-23-6	C8H7O4CL	9	8	202.59	Pm
Dichlorovanillic acid	79186-92-4	C8H6O4CL2	10	8	237.04	
2,3-Dichlorovanillic acid	108544-97-4					
Trichlorovanillic acid	9328-59-0	C8H5O4CL3	11	8	271.48	
Dichlorosyringic acid	20624-95-8	C9H8O5CL2	11	9	267.06	
Chloromethoxyprotocatechuic acid		C8H7O5CL	9	8		
Chloro-2-thiophenic acid		C5H3O2CLS	6	5		
3-Chloro-2-thiophenic acid	59337-89-2					
4-Chloro-2-thiophenic acid	59614-95-8					
5-Chloro-2-thiophenic acid	24065-33-6					
Dichloro-2-thiophenic acid		C5H2O2CL2S	7	5		
2,3-Dichloro-2-thiophenic acid	31166-29-7					
3,4-Dichloro-2-thiophenic acid	61209-02-7					
3,5-Dichloro-2-thiophenic acid	89166-94-9					

Table B.1 (cont.)

CHEMICAL NAME	CAS No.	Chemical Formula	(C+Cl)[a] no. N	(C)[b] no. C	M.W. [c]	Data [d] Code
Chlorodehydroabietic acid	57055-38-6	C20H28O2CL	21	20	335.88	E,Pm
12-Chlorodehydroabietic acid		C20H28O2CL	21	20	335.88	D,Pm
14-Chlorodehydroabietic acid		C20H28O2CL	21	20	335.88	D,Pm
Dichlorodehydroabietic acid	57055-39-7	C20H27O2CL2	22	20	370.32	E,D,Pm
12,14-Dichlorodehydroabietic acid	65281-77-6	C20H27O2CL2	22	20	370.32	Pm
Neoabietic acid	471-77-2					D,Pm
Trichloro-2-oxo-3-pentenoic acid isomer 1		C5H3O3CL3	8	5	217.44	Pm
Trichloro-2-oxo-3-pentenoic acid isomer 2		C5H3O3CL3	8	5	217.44	Pm
5,5,5-Trichloro-2-oxo-3-pentenoic acid	99165-97-6	C5H3O3CL3	8	5	217.44	Pm
3,4,5,5-Tetrachloro-2-oxo-3-pentenoic acid	99165-94-3	C5H2O3CL4	9	5	251.88	Pm
Tetrachloro-2-oxo-3-pentenoic isomer 1		C5H2O3CL4	9	5	251.88	Pm
Tetrachloro-2-oxo-3-pentenoic isomer 2		C5H2O3CL4	9	5	251.88	Pm
3,4,5,5,5-Pentachloro-2-oxo-3-pentenoic acid	99165-95-4	C5HO3CL5	10	5	286.33	Pm
Chlorothiophendicarboxylic acid	99165-93-2	C6H3O4SCL	7	6	206.6	Pm
5-Chlorothiophendicarboxylic acid	36157-45-6					Pm
Neutral Compounds						
Chloro-1-(2-hydroxy)-isopropyl-4-methyl benzene						Pm
Dichloro-1-(2-hydroxy)-isopropyl-4-methyl benzene (5 isomers)						Pm
1,3-Dichloro-2-propanol	96-23-1	C3H6OCL2	5	3	128.99	E,Pm
1,1,3-Trichloro-2-propanol	39979-44-7	C3H4OCL2	5	3	163.43	Pm
Chloroacetaldehyde	107-20-0	C2H3OCL	3	2	78.5	Pm
Dichloroacetaldehyde	79-02-7	C2H2OCL2	4	2	112.94	Pm
Trichloroacetaldehyde	75-87-6	C2HOCL3	5	2	147.39	H,P,Pm
2-Chloropropenal	683-51-2	C3H3OCL	4	3	90.52	Pm
Chlorobutenal		C4H5OCL	5	4	104.54	Pm
Dichlorobutenal		C4H4OCL2	6	4	138.99	Pm
4-Chloro-2-butenal	24443-10-5					Pm
2-Chloro-2-butenal	53175-28-3					Pm
Chlorobenzaldehyde	35913-09-8	C7H5OCL	8	7	140.57	Pm
Dichlorobenzaldehyde	31155-09-6	C7H4OCL2	9	7	175.02	Pm
Chloroacetone	78-95-5	C3H5OCL	4	3	92.53	P,Pm
Dichloroacetone		C3H4OCL2	5	3	126.97	Pm
1,1-Dichloroacetone	513-88-2	C3H4OCL2	5	3	126.97	Pm
1,3-Dichloroacetone	534-07-6	C3H4OCL2	5	3	126.97	P,Pm
Trichloroacetone	918-00-3	C3H3OCL3	6	3	161.42	Pm
1,1,1-Trichloroacetone	918-00-3	C3H3OCL3	6	3	161.42	Pm
1,1,3-Trichloroacetone	921-03-6	C3H3OCL3	6	3	161.42	P,Pm
Tetrachloroacetone	31422-61-4	C3H2OCL4	7	3	195.86	Pm
1,1,1,3-Tetrachloroacetone	16995-36-0	C3H2OCL4	7	3	195.86	Pm
1,1,3,3-Tetrachloroacetone	632-21-3	C3H2OCL4	7	3	195.86	Pm
Pentachloroacetone	1769-31-6	C3H2OCL5	8	3	230.3	Pm
Hexachloroacetone	116-16-5	C3OCL6	9	3	264.75	P,Pm
Trichlorocyclopropanone	95451-72-2	C3HOCL3	6	3	159.4	Pm
Trichlorocyclobutenone	78099-58-8	C4HOCL3	7	4	171.41	Pm
Dichlorocyclopentene-1,2-dione	94650-97-2	C5H4O2CL2	7	5	166.99	Pm
5,5-Dichloro-3-cyclopentene-1,2-dione	110930-98-8					Pm
Trichlorocyclopentene-1,2-dione (2 isomers)	103354-08-1	C5H3O2CL3	8	5	201.44	Pm
3,4,5-Trichloro-3-cyclopentene-1,2-dione	89263-14-7					Pm
3,5,5-Trichloro-3-cyclopentene-1,2-dione	67951-44-4					Pm
4,5,5-Trichloro-3-cyclopentene-1,2-dione	110930-99-9					Pm
Tetrachlorocyclopentene-1,2-dione	4054-42-6	C5H2O2CL4	9	5	235.88	Pm
Dichloro-1,2-benzoquinone	18268-81-0	C6H2O2CL2	8	6	176.99	P,Pm

Table B.1 (cont.)

CHEMICAL NAME	CAS No.	Chemical Formula	(C+Cl) [a] no. N	(C) [b] no. C	M.W.[c]	Data[d] Code
Tetrachloro-1,2-benzoquinone	2435-53-2	C6O2CL4	10	6	245.88	Pm
3-Chloro-4-dichloromethyl-5-hydroxy-2(5H)-furanone	77439-76-0	C5H3O3CL3	8	5	217.44	Pm
Dichloroacetic acid methyl ester	116-54-1	C3H4O2CL2	7	3	142.97	Pm
Chloroacetic acid ethyl ester	105-39-5	C4H7O2CL	5	4	122.55	Pm
Dichloroacetic acid ethyl ester	535-15-9	C4H6O2CL2	6	4	157	Pm
Trichloroacetic acid ethyl ester	515-84-4	C4H5O2CL3	7	4	191.44	Pm
Dichloromethane	75-09-2	CH2CL2	3	1	84.93	H,E,P
Chloroform	67-66-3	CHCL3	4	1	119.38	H,E,P,D
Carbon tetrachloride	56-23-5	CCL4	5	1	153.82	H,P
Bromodichloromethane	75-27-4	CHBrCL2	3	1	163.83	H,P
Dibromochloromethane	124-48-1	CHBr2CL	2	1	208.3	H,P
1,2-Dichloroethane	107-06-2	C2H4CL2	4	2	98.97	H,E,P
1,1,1-Trichloroethane	71-55-6	C2H3CL3	5	2	133.41	H,E,P
1,1,2,2-Tetrachloroethane	79-34-5	C2H2CL4	6	2	167.86	H,E,P
Trichloroethene	79-01-6	C2HCL3	5	2	131.4	H,P
Tetrachloroethene	127-18-4	C2CL4	6	2	165.83	H,P
Tetrachloropropene (3 isomers)	60320-18-5	C3H2CL4	7	3	179.86	Pm
1,2,3,3-Tetrachloropropene	20589-85-9					Pm
1,1,3,3-Tetrachloropropene	18611-43-3					Pm
2,3,3,3-Tetrachloropropene	16500-91-7					Pm
1,3,3,3-Tetrachloropropene	15022-22-7					Pm
1,1,2,3-Tetrachloropropene	10436-39-2					Pm
Pentachloropropene	69102-77-8	C3HCL4	7	3	214.31	Pm
Dichloropropadiene	76720-36-2	C3H2CL2	5	3	108.95	Pm
1,3-Dichloro-1,2-propadiene	53682-32-0					Pm
1,1-Dichloro-1,2-propadiene	108562-60-3					Pm
Trichloropropadiene	20170-70-1	C3HCL3	6	3	143.4	Pm
Tetrachloropropadiene	18606-30-6	C3CL4	7	3	177.84	Pm
Trichlorobutatriene (2 isomers)	95418-84-1	C4HCL3	7	4	155.41	Pm
Tetrachlorobutatriene	19792-18-8	C4CL4	8	4	189.86	Pm
Pentachlorobutadiene (2 isomers)	55680-77-8	C4HCL5	9	4	226.32	Pm
Methyl chlorobutene (5 isomers)		C5H9CL	6	5	104.58	Pm
Tetrachlorocyclopentadiene	695-77-2	C5H2CL4	9	5	203.88	Pm
Hexachlorohexatriene (5 isomers)	95418-94-3	C6H2CL6	12	6	286.8	Pm
1,1,2,5,6,6-Hexachloro-1,3,5-hexatriene	20589-54-6					Pm
1,1,2,3,5,6-Hexachloro-1,3,5-hexatriene	101854-40-4					Pm
1,1,2,4,5,6-Hexachloro-1,3,5-hexatriene	101854-38-0					Pm
1,2,3,4,5,6-Hexachloro-1,3,5-hexatriene	89465-68-3					Pm
Heptachlorohexatriene (2 isomers)	95418-95-4	C6HCL7	13	6	321.24	Pm
Chlorobenzene	108-90-7	C6H5CL1	7	6	112.56	H,E,P,Pm
Dichlorobenzene					147	P
1,2-Dichlorobenzene	95-50-1	C6H4CL2	8	6	147	H,E,P
1,3-Dichlorobenzene	541-73-1	C6H4CL2	8	6	147	E,P
1,4-Dichlorobenzene	106-46-7	C6H4CL2	8	6	147.01	E,P
1,2,3-Trichlorobenzene	87-61-6	C6H3CL3	9	6	181.45	P
1,2,4-Trichlorobenzene	120-82-1	C6H6CL3	9	6	181.5	H,P
Tetrachlorobenzene					215.9	P
1,2,3,4-Tetrachlorobenzene	634-66-2	C6H2CL4	10	6	215.9	P
1,2,3,5-Tetrachlorobenzene	634-90-2	C6H2CL4	10	6	215.9	P
1,2,4,5-Tetrachlorobenzene	95-94-3	C6H2CL4	10	6	215.9	H,P
Pentachlorobenzene	608-93-5	C6HCL5	11	6	250.3	H,E,P
Hexachlorobenzene	118-74-1	C6CL6	12	6	284.8	H,P
Chloro-p-cymene	54411-19-7	C10H13CL	11	10	168.67	P
Chloro-p-cymene (1)	4396-80-6	C10H13CL	11	10		P

Table B.1 (cont.)

CHEMICAL NAME	CAS No.	Chemical Formula	(C+Cl) [a] no. N	(C) [b] no. C	M.W. [c]	Data [d] Code
Chloro-p-cymene (2)	4395-79-3	C10H13CL	11	10		P
Dichloro-p-cymene	65724-12-1	C10H12CL2	12	10	203.12	P
Dichloro-p-cymene (1)	81686-41-1	C10H12CL2	12	10		P
Dichloro-p-cymene (2)	81686-44-4	C10H12CL2	12	10		P
3,5-Dichloro-p-cymene	81686-45-5					Pm
2,6-Dichloro-p-cymene	81686-46-6					Pm
Trichloro-p-cymene	66329-96-2	C10H11CL3	13	10	237.56	Pm
Chlorocalamenene	66365-28-4		14	14	224.77	Pm
Chlorodimethylpropyldihydronaphthalene		C15H20CL	16	15	235.78	Pm
Dichlorodimethylpropyldihydronaphthalene		C15H19CL2	17	15	270.22	Pm
Dichlorodimethylpropylnaphthalene		C15H16CL2	17	15	267.19	Pm
Terpenes						Pm
Chlorotrimethoxybenzene	77241-44-2	C9H11O3CL	10	9	202.64	Pm
2-Chloro-1,3,5-trimethoxybenzene	67827-56-9					Pm
1-Chloro-2,3,4-trimethoxybenzene	54625-53-5					Pm
1-Chloro-2,4,5-trimethoxybenzene	20129-10-6					Pm
5-Chloro-1,2,3-trimethoxybenzene	2675-80-1					Pm
Trichlorotrimethoxybenzene	77223-56-4	C9H9CL3	12	9	271.54	Pm
1,2,4-Trichloro-3,5,6-trimethoxybenzene	99585-78-1					Pm
1,2,3-Trichloro-4,5,6-trimethoxybenzene	2539-13-1					Pm
1,3,5-Trichloro-2,4,6-trimethoxybenzene	1015-96-9					Pm
1,1-Dichlorodimethylsulfone	37557-97-4	C2SH4O2CL2	4	2	163.02	Pm
1,1,3-Trichlorodimethylsulfone	64568-19-0	C2SH3O2CL3	5	2	197.46	Pm
Trichlorothiophene	26761-53-5	C4SHCL3	7	4	187.48	Pm
2,3,4-Trichlorothiophene	17249-78-4					Pm
2,3,5-Trichlorothiophene	17249-77-3					Pm
Tetrachlorothiophe:	6012-97-1	C4SCL4	8	4	221.92	P,Pm
Dichloro-2-formylthiophene	66717-87-1	C5SH2OCL2	7	5	181.04	Pm
3,5-Dichloro-2-formylthiophene	57482-51-3					Pm
3,4-Dichloro-2-formylthiophene	57482-50-2					Pm
4,5-Dichloro-2-formylthiophene	57482-49-9					Pm
Trichloro-2-formylthiophene	66729-79-1	C5SCL3	8	5	214.47	Pm
2-Acetyldichlorothiophene (2 isomers)	66717-88-2	C6SH4OCL2	8	6	195.07	Pm
2-Acetyltrichlorothiophene	65434-11-9	C6SH3OCL3	9	6	229.52	Pm
4,5-Dichloro-2-acetylthiophene	57681-59-1					Pm
3,4-Dichloro-2-acetylthiophene	57681-58-0					Pm
Dichloro-2-propionylthiophene (2 isomers)	66717-89-3	C7SH6OCL2	9	7	209.09	Pm
2,5-Dichloro-3-propionylthiophene	32427-79-5					Pm
Trichloroveratrole	73513-25-4					D,Pm
Tetrachloroveratrole	944-61-6					Pm
Chlorinated Phenols						D,Pm
Chlorinated Resin Acids						D,Pm
Chlorinated Guaiacols						D,Pm
Chlorinated Catechols						D,Pm
Chlorinated Veratroles						Pm
Chlorinated Vanillins						D,Pm
Phenolic Compounds						
Chlorophenol	25167-80-0	C6H5OCL	7	6		E,Pm
2-Chlorophenol	95-57-8	C6H5OCL	7	6	128.56	H,E,P
3-Chlorophenol	108-43-0	C6H5OCL	7	6	128.56	P
4-Chlorophenol		C6H5OCL	7	6	128.56	E,P
2,3-Dichlorophenol	576-24-9	C6H4OCL2	8	6	163	P

Table B.1 (cont.)

CHEMICAL NAME	CAS No.	Chemical Formula	(C+Cl)[a] no. N	(C)[b] no. C	M.W. [c]	Data[d] Code
2,4-Dichlorophenol	120-83-2	C6H4OCL2	8	6	163	H,P,D
2,5-Dichlorophenol	583-78-8	C6H4OCL2	8	6	163	P,Pm
2,6-Dichlorophenol	87-65-0	C6H4OCL2	8	6	163	E,P,D
2,3,6-Trichlorophenol	933-75-5	C6H3OCL3	9	6	197.45	E,P
2,4,5-Trichlorophenol	95-95-4	C6H3OCL3	9	6	197.45	H,E,P,D
2,4,6-Trichlorophenol	88-06-2	C6H3OCL3	9	6	197.45	H,E,P,D
Tetrachlorophenol	25167-83-3	C6H2OCL4	10	6	231.9	Pm
2,3,4,5-Tetrachlorophenol	4901-51-3	C6H2OCL4	10	6	231.9	P
2,3,4,6-Tetrachlorophenol	58-90-2	C6H2OCL4	10	6	231.9	H,E,P,D
2,3,5,6-Tetrachlorophenol	935-95-5	C6H2OCL4	10	6	231.9	E,P,Pm
Pentachlorophenol	87-86-5	C6HOCL5	11	6	266.34	H,E,P,D
Dichlorocatechol (2 isomers)	25167-80-5	C6H4O2CL2	8	6	179	P,Pm
3,4-Dichlorocatechol	3978-67-4	C6H4O2CL2	8	6	179	D,Pm
3,6-Dichlorocatechol	3936-16-7					D,Pm
3,5-Dichlorocatechol	13673-92-2					E,Pm
4,5-Dichlorocatechol	3428-24-8					D,E,Pm
Trichlorocatechol (3 isomers)	25167-84-4	C6H3O2CL3	9	6	213.46	Pm
3,4,5-Trichlorocatechol	56961-20-7	C6H3O3CL3	9	6	213.46	D,Pm
3,4,6-Trichlorocatechol	32139-72-3	C6H3O3CL3	9	6	213.46	P,D,Pm
Tetrachlorocatechol	1198-55-6	C6H2O2CL2	8	6	247.89	E,P,D,Pm
3,4,5,6-Tetrachlorocatechol	1198-55-6	C6H2O2CL2	8	6	247.89	E,P,D,Pm
Chloroguaiacol		C7H7O2CL	8	7	158.6	Pm
5-Chloroguaiacol	3743-23-5					Pm
Dichloroguaiacol	2460-49-3	C7H6O2CL2	9	7	193.04	D,Pm
4,5-Dichloroguaiacol	2460-49-3	C7H6O2CL2	9	7	193.04	P,D,Pm
3,4,6-Trichloroguaiacol	60712-44-9	C7H5O2CL3	10	7	227.49	Pm
3,4,5-Trichloroguaiacol	57057-83-7	C7H5O2CL3	10	7	227.49	E,P,D,Pm
4,5,6-Trichloroguaiacol	2668-24-8	C7H5O2CL3	10	7	227.49	E,P,D,Pm
Tetrachloroguaiacol	2539-17-5	C7H4O2CL4	11	7	261.93	P,D,Pm
3,4,5,6-Tetrachloroguaiacol	2539-17-5	C7H4O2CL4	11	7	261.93	E,D,Pm
6-Chlorovanillin	18268-76-3	C8H7O3CL	9	8	186.59	D,Pm
5,6-Dichlorovanillin	18268-69-4	C8H6O3CL2	10	8	221.04	D,Pm
Dichlorovanillin	18268-69-4	C8H6O3CL2	10	8	221.04	Pm
Trichlorovanillin	119464-51-6	C8H5O3CL3	11	8	255.49	Pm
3,5-Dichloro-4-hydroxybenzaldehyde	2314-36-5	C7H4O2CL2	9	7	191.01	Pm
Chloroprotocatechualdehyde		C7H5O3CL	8	7		Pm
2-Chloroprotocatechualdehyde	32984-11-2					Pm
5-Chloroprotocatechualdehyde	34099-18-5					Pm
6-Chloroprotocatechualdehyde	37585-56-9					Pm
Dichloroprotocatechualdehyde		C7H4O3CL2	9	7		Pm
Chlorosyringaldehyde	76341-69-0	C9H9O4CL	10	9	216.63	Pm
Dichlorosyringaldehyde	76330-06-8	C9H8O4CL2	11	9	251.07	Pm
Chloro-1,2,3-trihydroxybenzene	75562-89-9	C6H5O3CL	7	6	160.56	Pm
Dichloro-1,2,3-trihydroxybenzene	75562-90-2	C6H4O3CL2	8	6	195.01	Pm
Trichloro-1,2,3-trihydroxybenzene	56961-21-8	C6H3O3CL3	9	6	229.45	Pm
Chloro-1,2,4-trihydroxybenzene	75562-91-3	C6H5O3CL3	9	6	160.56	Pm
Dichloro-1,2,4-trihydroxybenzene	75562-92-4	C6H4O3CL2	8	6	195.01	Pm
Trichloro-1,2,4-trihydroxybenzene	56961-22-9	C6H3O3CL3	9	6	229.45	Pm
3,4,5-Trichlorosyringol	2539-26-6	C8H7O3CL3	11	8	257.5	D,Pm
Dichloroacetosyringone	94649-71-5	C10H10O4CL2	12	10	265.09	Pm
Dichloro-1,2-dihydroxy-3-methoxybenzene	75562-93-5	C7H6O3CL2	9	7	209.03	Pm
Chloro-3,4-dihydroxypropiophenone	25069-71-0	C9H9O3CL	10	9	200.62	Pm
Dichloro-3,4-dihydroxypropiophenone (2 isomers)	66021-80-5	C9H8O3CL2	11	9	235.06	Pm
Chloropropioguaiacone (chloropropiovanillone)						Pm

Table B.1 (cont.)

CHEMICAL NAME	CAS No.	Chemical Formula	(C+Cl)[a] no. N	(C)[b] no. C	M.W.[c]	Data[d] Code
Dichlorociconiferyl alcohol		C10H10O3CL2	12	10	249.09	Pm
Trichlorodehydrociconiferyl alcohol		C10H7O3CL3	13	10	281.52	Pm
3,4,5-Trichloro-2,6-dimethoxyphenol	2539-26-6	C8H7O3CL3	11	8	257.5	Pm
3,5-Dichloro-2,6-dimethoxyphenol	78782-46-4	C8H8O3CL2	10	8	223.06	Pm

[a] Number of carbon and chlorine atoms in molecule.
[b] Number of carbon atoms in molecule.
[c] Molecular weight.
[d] Data code:

H - Human health data readily available;
E - Ecotox data readily available;
P - Physical/chemical and fate properties readily available;
P - Minimal p/chem properties readily available; and
D^m - Treated effluent data available.

References: Suntio, et al. (1988); USEPA (1988); NCASI (1977, 1980, 1981, 1982a, 1982b, 1983a, 1983b, 1983c, 1984a, 1984b, 1985a).

TABLE B-2
OTHER CHLORINATED ORGANIC CHEMICALS CANCER RISK

Chemical	CAS No.	Cancer Classification	Oral Slope Factor mg/Kg-day	Drinking Water Unit Risk ug/l	Inhalation Slope Factor mg/Kg-day	Inhalation Unit Risk ug/cu. m.
Carbon Tetrachloride	56-23-5	B2	1.30E-01	3.70E-06	1.30E-01	1.50E-05
Chloroform	67-66-3	B2	6.10E-03	1.70E-07	8.10E-02	2.30E-05
1,1,1-Trichloroethane	71-55-6	D	NA	NA	NA	NA
Bromodichloromethane	75-27-4	NA	NA	NA	NA	NA
Chloral	75-87-6	NA	NA	NA	NA	NA
1,2-Dichloroethane	107-06-2	B2	9.10E-02	2.60E-06	9.10E-02	2.60E-05
Hexachlorobenzene	118-74-1	NA	NA	NA	NA	NA
1,2,4-Trichlorobenzene	120-82-1	D	NA	NA	NA	NA
2,4-Dichlorophenol	120-83-2	NA	NA	NA	NA	NA
Dibromochloromethane	124-48-1	NA	NA	NA	NA	NA
Tetrachloroethylene	127-18-4	NA	NA	NA	NA	NA
Pentachlorobenzene	608-93-5	NA	NA	NA	NA	NA
2,3,4,6-Tetrachlorophenol	58-90-2	NA	NA	NA	NA	NA
Dichloromethane	75-09-2	B2	7.50E-03	2.10E-07	1.40E-02	4.10E-06
Trichloroethylene	79-01-6	NA	NA	NA	NA	NA
1,1,2,2-Tetrachloroethane	79-34-5	C	2.00E-01	5.80E-06	2.00E-01	5.80E-05
Pentachlorophenol	87-86-5	NA	NA	NA	NA	NA
2,4,6-Trichlorophenol	88-06-2	B2	2.00E-02	5.70E-07	2.00E-02	5.70E-06
1,2-Dichlorobenzene	95-50-1	NA	NA	NA	NA	NA
1,3-Dichlorobenzene	541-73-1	NA	NA	NA	NA	NA
1,4-Dichlorobenzene	106-46-7	NA	NA	NA	NA	NA
2-Chlorophenol	95-57-8	NA	NA	NA	NA	NA
1,2,4,5-Tetrachlorobenzene	95-94-3	NA	NA	NA	NA	NA
2,4,5-Trichlorophenol	95-95-4	NA	NA	NA	NA	NA
Chlorobenzene	108-90-7	NA	NA	NA	NA	NA
Chororacetic acid	79-11-8	NA	NA	NA	NA	NA

NA = Not Available

TABLE B-3
OTHER CHLORINATED ORGANIC CHEMICALS DRINKING WATER HEALTH ADVISORIES

Chemical	CAS No.	Maximum Contamination Limit mg/l	Water[a] Quality Criteria ug/l	One-day Child mg/l	Ten-day Child mg/l	Longer-term Child mg/l	Longer-term Adult mg/l	DWEL mg/l	Lifetime mg.l
Carbon Tetrachloride	56-23-5	5.00E-03	4.00E-01	4.00E+00	1.60E-01	7.10E-02	2.50E-01	2.50E-02	None
Chloroform	67-66-3		1.90E-01	NA	NA	NA	NA	NA	NA
1,1,1-Trichloroethane	71-55-6	2.00E-01	1.84E+04	NA	NA	NA	NA	NA	NA
Bromodichloromethane	75-27-4			NA	NA	NA	NA	NA	NA
Chloral	75-87-6			NA	NA	NA	NA	NA	NA
1,2-Dichloroethane	107-06-2	5.00E-03	9.40E-01	7.40E-01	7.40E-01	7.40E-01	2.60E+00	NC	NC
Hexachlorobenzene	118-74-1	0.00E+00 [b]	7.20E-04	NA	NA	NA	NA	NA	NA
1,2,4-Trichlorobenzene	120-82-1	9.00E-03 [b]		NA	NA	NA	NA	NA	NA
2,4-Dichlorophenol	120-83-2		3.09E+03	NA	NA	NA	NA	NA	NA
Dibromochloromethane	124-48-1			NA	NA	NA	NA	NA	NA
Tetrachloroethylene	127-18-4	5.00E-03 [b]	8.00E-01	2.00E+00	2.00E+00	1.40E+00	5.00E+00	5.00E-01	1.00E-02
Pentachlorobenzene	608-93-5		7.40E+01	NA	NA	NA	NA	NA	NA
2,3,4,6-Tetrachlorophenol	58-90-2			NA	NA	NA	NA	NA	NA
Dichloromethane	75-09-2			1.33E+01	1.50E+00	5.00E-01	1.75E+00	1.75E+00	None
Trichloroethylene	79-01-6	5.00E-03	2.70E+00	NA	NA	NA	NA	NA	NA
1,1,2,2-Tetrachloroethane	79-34-5		1.70E-01	NA	NA	NA	NA	NA	NA
Pentachlorophenol	87-86-5	2.00E-01 [b]	1.01E+03	1.00E+00	3.00E-01	3.00E+01	1.05E+00	1.05E+00	2.20E-01
2,4,6-Trichlorophenol	88-06-2		1.20E+00	NA	NA	NA	NA	NA	NA
1,2-Dichlorobenzene	95-50-1	6.00E-01 [b]	4.00E+02	NA	NA	NA	NA	NA	NA
1,3-Dichlorobenzene	541-73-1		4.00E+02	NA	NA	NA	NA	NA	NA
1,4-Dichlorobenzene	106-46-7	7.50E-02	4.00E+02	NA	NA	NA	NA	NA	NA
2-Chlorophenol	95-57-8			NA	NA	NA	NA	NA	NA
1,2,4,5-Tetrachlorobenzene	95-94-3		3.80E+01	NA	NA	NA	NA	NA	NA
2,4,5-Trichlorophenol	95-95-4		2.60E+03	NA	NA	NA	NA	NA	NA
Chlorobenzene	108-90-1	1.00E-01 [b]		NA	NA	NA	NA	NA	NA
Chloroacetic acid	79-11-8			NA	NA	NA	NA	NA	NA

[a] Criteria is for human water and fish consumption.
[b] Proposed MCL.
NA = Not available.

TABLE B-4
OTHER CHLORINATED ORGANIC CHEMICALS REFERENCE DOSES

Chemical	CAS No.	Oral RfD mg/Kg-day	Oral UF	Dermal RfD mg/Kg-day	Dermal UF	Inhalation RfD mg/Kg-day	Inhalation UF	RfD Date
1,1,1-Trichloroethane	71-55-6	9.00E-02	1,000	ND	ND	ND	ND	6/30/88
Carbon Tetrachloride	56-23-5	7.00E-04	1,000	ND	ND	ND	ND	6/30/88
Chloroform	67-66-3	1.00E-02	1,000	ND	ND	ND	ND	6/30/88
Bromodichloromethane	75-27-4	2.00E-02	1,000	ND	ND	ND	ND	12/1/88
Chloral	75-87-6	2.00E-03	10,000	ND	ND	ND	ND	8/22/88
1,2-Dichloroethane	107-06-2	ND	ND	ND	ND	ND	ND	ND
Hexachlorobenzene	118-74-1	8.00E-04	100	ND	ND	ND	ND	2/1/89
1,2,4-Trichlorobenzene	120-82-1	2.00E-02	1,000	ND	ND	ND	ND	6/30/88
2,4-Dichlorophenol	120-83-2	3.00E-02	100	ND	ND	ND	ND	6/30/88
Dibromochloromethane	124-48-1	2.00E-03	1,000	ND	ND	ND	ND	6/30/88
Tetrachloroethylene	127-18-4	1.00E-02	1,000	ND	ND	ND	ND	3/1/88
Pentachlorobenzene	608-93-5	8.00E-04	10,000	ND	ND	ND	ND	3/1/88
2,3,4,6-Tetrachlorophenol	58-90-2	3.00E-02	1,000	ND	ND	ND	ND	3/1/88
Dichloromethane	75-09-2	6.00E-02	100	ND	ND	ND	ND	1/1/89
Trichloroethylene	79-01-6	ND	ND	ND	ND	ND	ND	ND
1,1,2,2-Tetrachloroethane	79-34-5	ND	ND	ND	ND	ND	ND	ND
Pentachlorophenol	87-86-5	3.00E-02	100	ND	ND	ND	ND	6/30/88
2,4,6-Trichlorophenol	88-06-2	ND	ND	ND	ND	ND	ND	ND
1,2-Dichlorobenzene	95-50-1	9.00E-02	1,000	ND	ND	ND	ND	8/1/89
1,3-Dichlorobenzene	541-73-1	ND	ND	ND	ND	ND	ND	ND
1,4-Dichlorobenzene	106-46-7	ND	ND	ND	ND	ND	ND	ND
2-Chlorophenol	95-57-8	5.00E-03	1,000	ND	ND	ND	ND	8/1/89
1,2,4,5-Tetrachlorobenzene	95-94-3	3.00E-04	1,000	ND	ND	ND	ND	2/1/89
2,4,5-Trichlorophenol	95-95-4	1.00E-01	1,000	ND	ND	ND	ND	3/1/88
Chorobenzene	108-90-1	2.00E-02	1,000	ND	ND	ND	ND	8/1/89
Chloroacetic acid	79-11-8	ND	ND	ND	ND	ND	ND	ND

ND = No Data

Table B-5. Species List[a]

Code	Latin name	Common name
AF	Astacus fluviatilis	Crayfish
AM	Ambystoma mexicanum	Salamander
AS	Artemia salina	Brine Shrimp
BBJ	Bufo bufo japonicus	Frog
BG	Biomphalaria glabrata	Snail
BR	Brachydanid rerio	Zebra Fish
CA	Carassius auratus	Goldfish
CC	Cyprinus carpio	Common (Colored) Carp
CF	Colisa fasciata	Giant Gourami
CRV	Crassostrea virginica	American (Virginia) Oyster
CS	Crangon septemspinosa	Sand Shrimp
CV	Cyprinodon variegatus	Sheepshead Minnow
DM	Daphnia magna	Waterflea
GA	Gasterosteus aculeatus	Three Spine Stickleback
HF	Heteropneustes fossilis	Indian Catfish
IP	Ictalurus punctatus	Channel Catfish
LM	Lepomis macrochirus	Bluegill
LP	Lemna perpusilla	Duckweed
MA	Mya arenaria	Soft Shell Clam
ME	Mesidotea entomon	Aquatic Sowbug
MIS	Micropterus salmoides	Largemouth Bass
NN	Notopterus notopterus	Featherback
NS	Nitora spinipes	Copepod
OD	Ophryotrocha diadema	Polychaete
PAP	Palaemonetes pugida	Grass Shrimp, Fresh Water Prawn
PP	Pimephales promelas	Fathead Minnow
PR	Poecilia leticulata	Guppy
RM	Rivulus marmoratus	Rivulus (fish, maybe Marine Minnow)
SC	Salmo gairdneri	Rainbow Trout
ST	Salmo trutta	Brown Trout
TM	Tilapia mossambica	Mozambique Tilapia
XL	Xenopus laevis	Clawed Toad

[a] Corresponds to species codes used in Table B-6.

Table B-6. Toxicity of Other Chlorinated Organics from Bleached Wood Products to Aquatic Species

Other chlorinated organics	Species[a]	Exposure duration (hours)	LC_{50} (mg/l)	Nominal (n) vs measured (m)
2,3,4,6-Tetrachlorophenol	DM	48	0.29	N
	ST	24	0.5	N
	CA	24	0.75	N
	CS	96	11.8	M
	MA	96	11.8	M
	LM	24	0.19	N
	LM	96	0.14	N
	LM	24	0.14	N
	RM	96	1.1	N
2,3,5,6-Tetrachlorophenol	DM	24	2.50	N
	DM	48	0.57	N
	LM	24	0.4	N
	LM	96	0.17	N
2,3,5-Trichlorocatechol	DM	24	3.39	N
2,3,6-Trichlorophenol	AF	8 Days	5.4	M
2,4,5-Trichlorophenol	DM	24	3.8	N
	DM	48	2.7	N
	LM	96	0.45	N
	ST	24	0.9	N
	CA	24	1.7	N
	LM	24	0.61	N
2,4,6-Trichlorophenol	DM	24	5.0	N
	DM	48	6.0	N
	MA	96	3.9	M
	ST	24	1.1	N
	CA	24	10.0	N
	CS	96	2.7	M
	LM	24	0.72	N
	LM	96	0.32	N
	PP	96	9.7	M
	PP	96	8.6	M
	PP	8 Days	5.8	M
	PP	8 Days	6.4	M
	DM	24	13.7	N
2,6-Dichlorophenol	ST	24	4.0	N
	CS	52	19.1	M

Table B-6. (continued)

Other chlorinated organics	Species[a]	Exposure duration (hours)	LC_{50} (mg/l)	Nominal (n) vs measured (m)
Chlorophenol	LM	96	6.6	N
2-Chlorophenol	DM	24	0.06	N
	DM	48	2.6	N
	CA	24	16.0	N
	LM	24	7.2	N
	PP	96	11.0	M
	PP	8 Days	6.3	M
	PP	24	21.96	N
	PP	96	11.63	N
	PP	48	18.0	N
	LM	24	11.31	N
	LM	48	10.59	N
	LM	96	10.0	N
	CA	24	14.48	N
	CA	48	12.37	N
	CA	96	12.37	N
	PR	24	22.17	N
	PR	48	20.78	N
	PR	96	20.17	N
	CS	96	5.3	M
	LM	24	8.2	M
	LM	48	8.2	M
4-Chlorophenol	NS	96	21.0	N
	DM	24	8.8	N
	DM	48	4.1	N
	CA	24	9.0	N
	LM	24	4.0	N
	LM	96	3.8	N
	ME	96	40.3	N
	ME	7 Days	37.5	N
	CS	96	4.6	M
2-Methoxy-4,5,6-trichlorophenol	DM	24	2.8	N
3,4,5,6-Tetrachlorocatechol	ST	24	1.1	N
	ST	24	1.1	N
Tetrachlorocatechol	NS	96	3.3	N
	DM	24	2.23	N
3,4,5,6-Tetrachloroguaiacol	SG	96	0.32	N

Table B-6. (continued)

Other chlorinated organics	Species[a]	Exposure duration (hours)	LC_{50} (mg/l)	Nominal (n) vs measured (m)
Tetrachloroguaiacol	NS	96	3.9	N
	DM	24	4.96	N
3,4,5-Trichloroguaiacol	SG	96	0.75	N
3,5-Dichlorocatechol	ST	24	2.9	N
4,5,6-Trichloroguaiacol	NS	96	5.2	N
	DM	24	2.9	N
	DM	24	2.8	N
4,5-Dichlorocatechol	ST	24	2.3	N
	DM	24	6.64	N
9,10-Dichlorostearic acid	SG	96	2.5	M
Carbon tetrachloride	LM	96	125.0	N
	MB	96	150.0	N
	DM	24	35.0	N
	DM	48	30.0	N
Chlorobenzene	DM	24	140.0	N
	DM	48	86.0	N
	PP	24	25.4	M
Chloroform	SG	28 Days	2.03	N
	SG	28 Days	2.0	N
	SG	28 Days	2.16	M
	SG	32 Days	1.24	M
	SG	32 Days	2.03	M
	DM	48	29.0	N
	MIS	12	56.2	M
	MIS	24	56.2	M
	MIS	12	50.4	M
	MIS	24	50.4	M
	IP	24	235.0	M
	IP	12	126.0	M
	IP	48	191.0	M
	IP	96	75.0	M
	LP	12	17.1	M
	LP	24	17.1	M
	SG	48	21.4	M
	SG	96	18.2	M
	SG	12	37.1	M

Table B-6. (continued)

Other chlorinated organics	Species[a]	Exposure duration (hours)	LC_{50} (mg/l)	Nominal (n) vs measured (m)
Chloroform (Continued)	SG	24	26.1	M
	PR	96	300.0	N
	SG	28 Days	1.240	M
	LM	7 Days	2.03	M
	BR	48	100.0	N
Ethylene dichloride	AS	24	320.0	N
Dichlorodihydroabietic acid	SG	96	0.6	N
	SG	96	0.8	N
Methylene chloride	DM	24	310.0	N
	DM	48	220.0	N
	LM	96	220.0	N
	LM	24	230.0	N
	DM	24	2,270.0	N
	PP	96	193.0	M
	PP	72	232.0	M
	PP	48	265.0	M
	PP	24	268.0	M
	PP	96	310.0	N
	PP	24	112.8	M
	PP	48	99.0	M
	PP	72	99.0	M
Monochlorodihydroabietic acid	SG	96	0.6	M
	SG	96	0.8	N
Pentachlorobenzene	DM	24	17.0	N
	DM	48	5.3	N
Pentachlorophenol	OO	24	1.5	M
	OO	24	2.4	M
	MS	96	0.27	N
	DM	24	1.5	N
	DM	48	0.68	N
	BBJ	24	0.17	N
	BBJ	96	0.12	N
	IP	24	0.12	N
	OO	24	1.5	M
	LM	96	0.26	N
	CA	24	0.27	N
	CS	66	3.3	M

Table B-6. (continued)

Other chlorinated organics	Species[a]	Exposure duration (hours)	LC_{50} (mg/l)	Nominal (n) vs measured (m)
Pentachlorophenol (Continued)	NN	24	0.113	N
	48	0.109	N	
	KN	72	0.09	N
	HF	24	0.41	N
	HF	48	0.38	N
	HF	72	0.33	N
	HF	96	0.29	N
	CF	24	0.57	N
	CF	48	0.52	N
	CF	96	0.45	N
	CD	48	1.1	M
	OD	72	0.9	M
	CA	72	0.057	N
	CA	96	0.023	N
	CA	96	0.056	N
	CA	48	0.08	N
	CA	72	0.084	N
	LM	6	0.057	N
	LM	24	0.038	N
	LM	48	0.031	N
	LM	72	0.025	N
	AF	8 Days	26.0	M
	AF	8 Days	9.0	M
	CA	24	0.052	N
	DM	21 Days	0.48	M
	DM	24	1.7	M
	DM	48	1.0	M
	DM	24	2.8	M
	DM	48	1.5	M
	GA	24	0.37 (LC_{90})	N
	TM	24	0.8	N
	CA	24	1.6 (LC_{90})	N
	BG	24	1.0 (LC_{90})	N
	BG	24	0.25 (LC_{90})	N
	CV	96	0.329	N
	CV	96	0.223	N
	AM	48	0.3	N
	PP	8 Days	0.2	N
	BR	48	0.4	N
	CRV	14 Days	0.07	N
	NN	96	0.083	N
	CF	72	0.48	N

Table B-6. (continued)

Other chlorinated organics	Species[a]	Exposure duration (hours)	LC_{50} (mg/l)	Nominal (n) vs measured (m)
Tetrachloroacetic acid	PP	96	2,000.0	N
	CC	96	2,500.0	N
	PP	96	2,000.0	N
Trichloroacetic acid	PP	96	2,000.0	N
	NS	96	4,800.0	N
	PP	48	2,000.0	N
Trichloroethylene	DM	48	18.0	N
	DM	24	22.0	N
	LM	96	45.0	N
1,1,1-Trichloroethane	DM	48	530	N
	DM	24	1,300	N
	LM	24	40	N
	LM	96	40	N
	PP	72	55.4	M
	PP	96	52.8	N
1,1,2,2-Tetrachloroethane	DM	24	18	N
	DM	48	9.3	N
	LM	24	21	N
	LM	48	21	N
	LM	24	21	N
1,2-Dichlorobenzene	MB	96	7.3	N
	PP	24	25.4	M
	PAP	96	9.4	M
	PAP	48	10.3	M
	PAP	24	14.3	M
	DM	24	2.4	N
	DM	48	2.4	N
	LM	96	5.6	N
	LM	24	6.3	N
1,3-Dichlorobenzene	DM	24	48	N
	DM	48	28	N
1,4-Dichlorobenzene	DM	24	42	N
	DM	48	11	N
1,2-Dichloroethane	DM	24	250	N
	DM	48	250	N

Table B-6. (continued)

Other chlorinated organics	Species[a]	Exposure duration (hours)	LC_{50} (mg/l)	Nominal (n) vs measured (m)
1,2,3,5-Tetrachlorobenzene	CA	3	2.5	N
	CV	3	1.58	N
	DM	24	18	N
	DM	48	9.7	N
1,3-Dichloro-2-proponal	CA	24	680	M

[a] Species codes are listed on Table B-5

Table B-7. Ranking by Acute Toxicity to Aquatic Organisms

Chemical	Lowest LC_{50} (mg/l)	Number of Studies
Pentachlorophenol	0.023	57
2,3,4,6-Tetrachlorophenol	0.14	9
2,3,5,6-Tetrachlorophenol	0.17	4
2,4,6-Trichlorophenol	0.32	13
3,4,5,6-Tetrachloroguaiacol	0.32	3
2,4,5-Trichlorophenol	0.45	6
Dichlorodihydroabietic acid	0.6	1
Monochlorodihydroabietic acid	0.6	1
3,4,5-Trichloroguaiacol	0.75	1
3,4,5,6-Tetrachlorocatechol	1.1	2
Chloroform	1.24	24
4,5-Dichlorocatechol	2.3	2
1,2-Dichlorobenzene	2.4	9
9,10-Dichlorostearic acid	2.5	1
2-Chlorophenol	2.6	21
4,5,6-Trichloroguaiacol	2.8	4
3,5-Dichlorocatechol	2.9	1
2,3,5-Trichlorocatechol	3.39	1
4-Chlorophenol	3.8	9
2,6-Dichlorophenol	4.0	2
Pentachlorobenzene	5.3	2
2,3,6-Trichlorophenol	5.4	1
Chlorophenol	6.6	1
1,1,2,2-Tetrachloroethane	9.3	6
1,2,3,5-Tetrachlorobenzene	9.7	6
1,4-Dichlorobenzene	11.0	6
Trichloroethylene	18.0	3
Dichloroacetic acid	23.0	1
Chlorobenzene	25.4	2
1,3-Dichlorobenzene	28.0	2
Carbon tetrachloride	30.0	5
1,1,1-Trichloroethane	40.0	7
Methylene chloride	99.0	13
1,2-Dichloroethane	220.0	2
Ethylene Dichloride	320	1
1,3-Dichloro-2-propanal	680	1
Tetrachloroacetic acid	2,000	7
Trichloroacetic acid	2,000	5

Table B-8. Acute Toxicity of Selected Chlorinated Organic Chemicals (OCO) to Terrestrial Organisms

Chemical	Lowest LC_{50} mg/kg	Exposure route	Organism
2,3,4,5-Tetrachlorophenol	400	Oral	Mouse
2,3,4,6-Tetrachlorophenol	140	Oral	Rat
	250	Skin	Rabbit
	250	Oral	Guinea pig
2,4,5-Trichlorophenol	1,620	Oral	Rat
Chloroform	1,194	Oral	Rat
	80	Oral	Mouse
2-Chlorophenol	670	Oral	Rat
	670	Oral	Mouse
1,2-Dichlorobenzene	500	Oral	Rabbit
	2,000	Oral	Guinea pig
4-Chlorophenol	261	Oral	Rat
1,4-Dichlorobenzene	500	Oral	Rat
	2,950	Oral	Mouse
	2,800	Oral	Guinea pig
Pentachlorophenol	56	Oral	Rat
	105	Skin	Rat
	168	Oral	Hamster
Pentachlorobenzene	1,080	Oral	Rat
	1,175	Oral	Mouse

TABLE B-9.

SUMMARY OF TREATED-EFFLUENT CHEMICAL CONCENTRATIONS

Chemical	Range of Concentration ug/l [a]		Number of Samples	Corresponding [b] Mill Process		LC50[c] ug/l
	Low	High		Low	High	
2,4-Dichlorophenol	0.4	51	39	CEHDED	CEHH/CEHD	
2,6-Dichlorophenol	1.2		1	CEHDED		4000 (2)[d]
2,4,5-Trichlorophenol	0	12.3	7	BK	BS	450 (6)
2,4,6-Trichlorophenol	1	61	59	CEHDED/BK	CEHH/CEHD	320 (13)
2,3,4,6-Tetrachlorophenol	0.5	5	14	CEHDED	BK	140 (9)
Pentachlorophenol	0	27	27[e]	BK	BS	23 (51)
3,4-Dichlorocatechol	0.8	1.4	2	CEHDED	NG	
3,4,5-Trichlorocatechol	1.7	114	29	BS	BK	
3,4,6-Trichlorocatechol	1.7	19	4	CEHDED	CEHD	
Tetrachlorocatechol	0.1	88	23	CEHDED	NG	
3,4,5,6-Tetrachlorocatechol	2	92	6	CEHDED	BK	1100 (2)
4,5-Dichloroguaiacol	0	24.3	14	BK	NG	
3,4,5-Trichloroguaiacol	1	78	35	CEHD/CEHDED	CEHDED	
4,5,6-Trichloroguaiacol	0.6	42	33	CEHDED	CEHDED	5200 (3)
Tetrachloroguaiacol	0.4	50.1	30	C/DED	C(D)E(O)H/D	
3,4,5,6-Tetrachloroguaiacol	1	36	9	CEHDED	CEHDED	320 (3)
6-Chorovanillin	3.8	21	5	BK	CEH	
5,6-Dichlorovanillin	0.8	12.4	5	BK	C(D)E(O)H/D	
9,10-Dichlorostearic acid	10	115	4	CEHDED	NG	2500 (1)
12-Chlorodehydroabietic acid	2	162	20	BK	BK	
14-Chlorodehydroabietic acid	2	27.3	13	CEHDED	NG	
Dichlorodehydroabietic acid	1	126	27	BK	CEHDED	600 (1)
Neoabietic acid	2	91	16	BK	BK	
Chloroform	3	1688	32	NG	K	1240 (24)
Trichloroveratrole	33.5	59.2	4	NG	NG	
Tetrachloroveratrole	10	47.8	4	NG	NG	

TABLE B-9. (continued)

SUMMARY OF TREATED-EFFLUENT CHEMICAL CONCENTRATIONS

Chemical	Range of Concentration ug/l [a]		Number of Samples	Corresponding [b] Mill Process		LC50 [c] ug/l
	Low	High		Low	High	
Chlorinated Phenols	2.2	143	13	BK	BK	
Chlorinated Resin Acids	0.49	49	5	BK	NG	
Chlorinated Guaiacols	5.4	110	10	BK	C(D)E(O)H/D	
Chlorinated Catechols	11	236	5	BK	NG	
Chlorinated Vanillins	4.6	36.6	5	BK	CEH	

[a] Treated effluent concentration does not account for dilution in stream.

[b] Mill process abbreviations used:

BK - Bleached Kraft
C - Chlorine
D - Chlorine Dioxide
E - Extraction with alkali
K - Kraft
O - Extraction with oxygen used as minor with E.
BS - Bleached Sulfite
D - Chlorine Dioxide
(D) - Chlroine dioxide used as minor method with C
H - Hypochlorite
NG - Not given

[c] Lowest value given here, all values listed in Table B-7.

[d] ( ) = number of LC50 values given in Table B-7.

[e] Compares to DVBL of 1050 ug/L for this compound.

Table B-10. Statistical Analysis of Temporal Changes in Chemical Concentrations in Pulp Mill Effluent[a]

Chemical name	N[b]	Average concentration (µg/L)[c]	Variance	Standard deviation	Minimum concentration (µg/L)	Maximum concentration (µg/L)
3,4,5-Trichlorocatechol	79	107.210	727.769	28.977	53.9	180
Tetrachlorocatechol	79	75.717	321.161	17.921	38.3	119
Dichlorodihydroabietic acid	78	42.299	269.196	16.407	15	85
3,4,5-Trichloroguaiacol	79	29.559	102.192	10.109	12.6	53.4
4,5-Dichlorocatechol	79	28.406	131.607	11.472	4.4	52.3
Tetrachloroguaiacol	79	23.824	33.614	5.798	13	44.2
2,4,6-Trichlorophenol	79	22.453	10.891	3.330	14	31.5
2,4-Dichlorophenol	79	10.924	4.754	2.180	7	17.7
4,5,6-Trichloroguaiacol	81	10.139	10.918	3.304	4.1	18
3,4,6-Trichlorocatechol	79	8.564	6.408	2.531	3.7	13.8
14-Chlorodehydroabietic acid	75	8.365	20.313	4.507	1	25
4,5-Dichloroguaiacol	79	5.686	29.661	5.446	0.3	18
2,3,4,6-Tetrachlorophenol	79	5.501	2.650	1.628	2.3	9
Pentachlorophenol	79	1.749	0.138	0.371	0.8	2.7
3,4-Dichlorocatechol	53	1.700	0.982	0.991	0.4	4.1
3,6-Dichlorocatechol	79	1.517	0.205	0.453	0.5	2.5
2,4,5-Trichlorophenol	79	1.504	0.160	0.400	0.5	2.8
2,6-Dichlorophenol	10	0.789	0.161	0.401	0.5	1.9

[a] Effluent samples were collected every other day during a NCASI study (NCASI technical bulletin #474, November 1985) on cold water stream productivity.

[b] Number of samples collected during the ten-month study.

[c] Average concentration (µg/L) listed highest to lowest.

Table B-11. Regulatory Schedule for OCOs in Some Canadian Provinces and European Countries[a]

Location	TOCl or AOX[b] Discharge (kg/t)	Target date
West Germany	1.0	1989
Finland	2.5	1994
Sweden	1.3-5.5	1988
Sweden	1.5	1992
Sweden	0.1	ca. 2010
Ontario[c]	2.5 (AOX)	1991
British Columbia[c]	2.5 (AOX)	1991
British Columbia[a]	1.5 (AOX)	1994
Quebec[c]	1.5 (AOX)	1993

[a] Source: Clodey (1989).
[b] Kilograms of TOCl or AOX per ton of air dried pulp produced..
[c] Regulations pending.

Figure B-1.

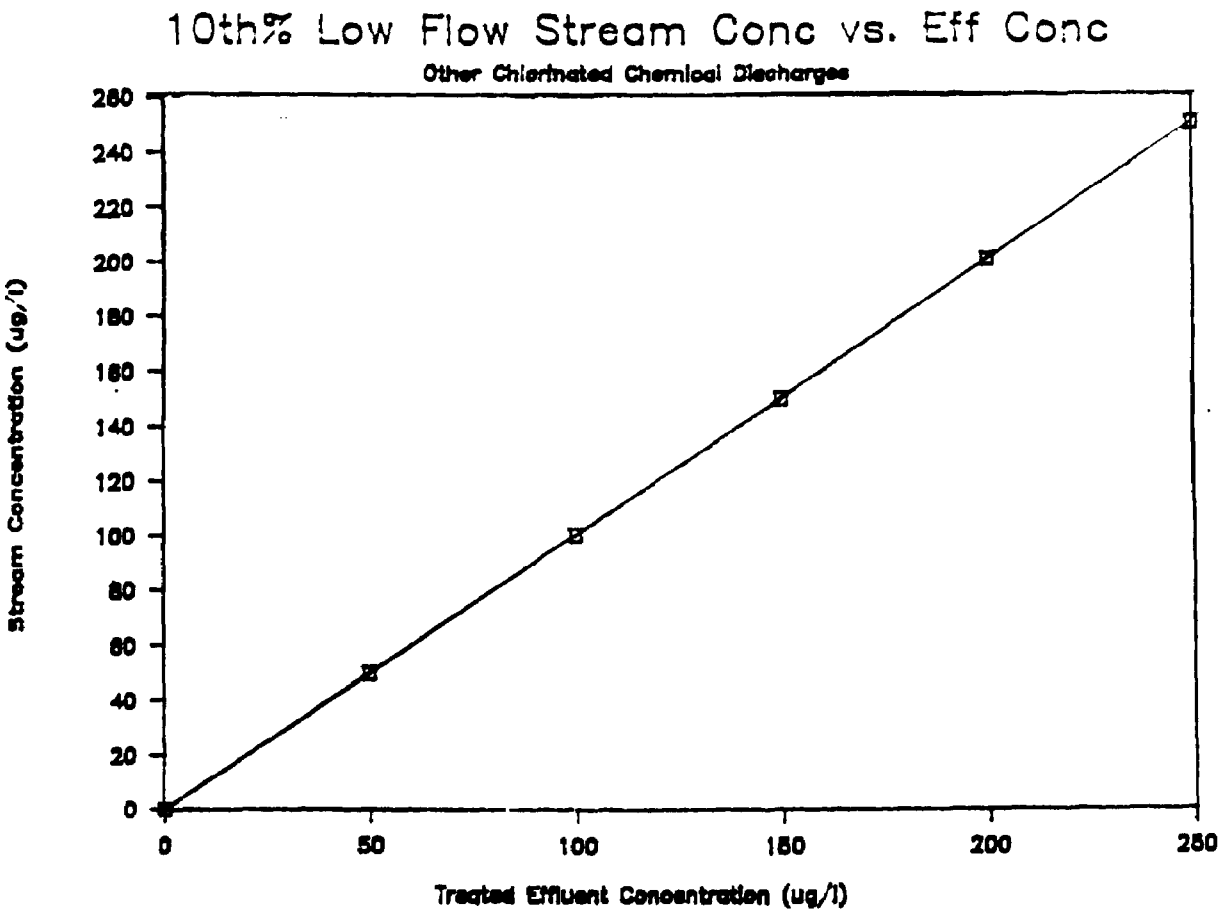
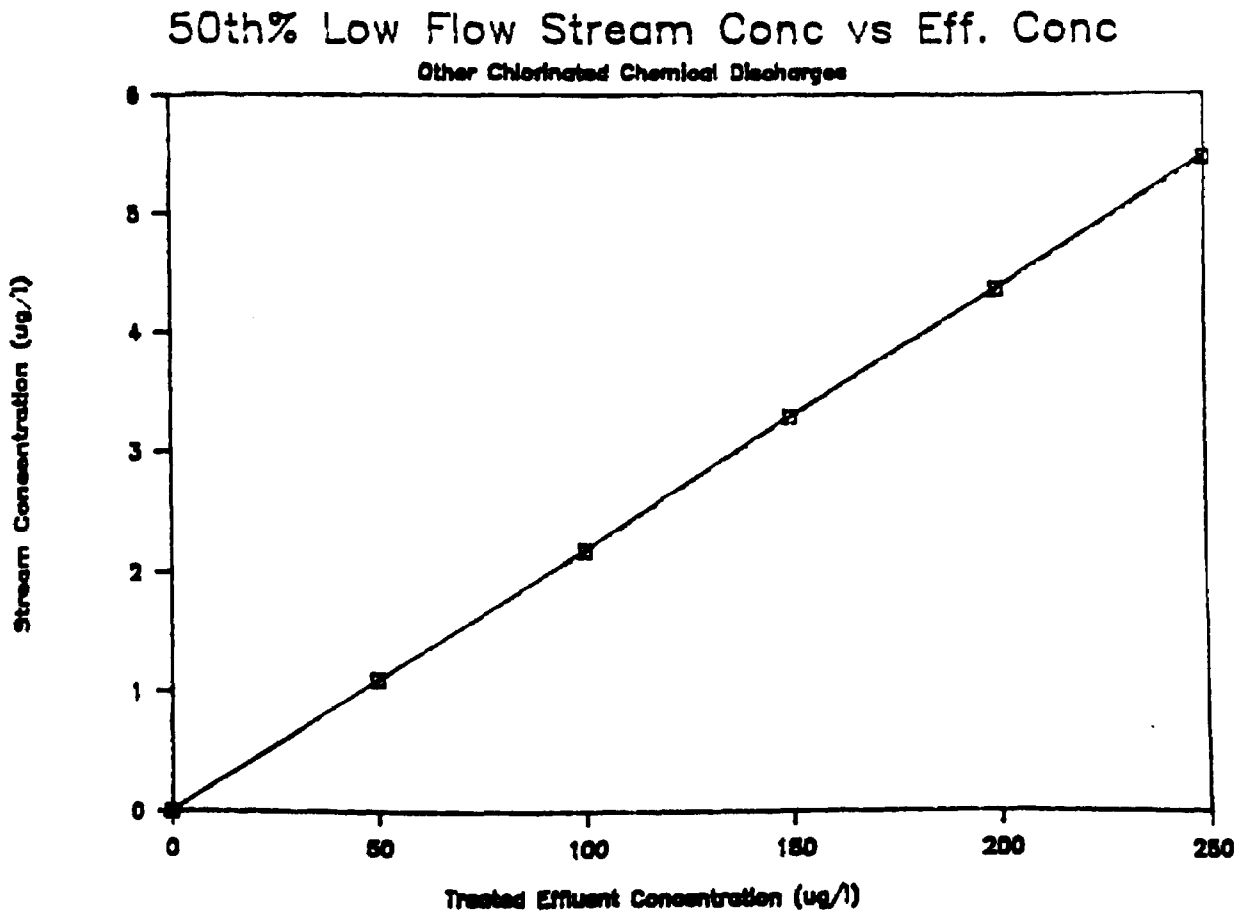


Figure B-2.



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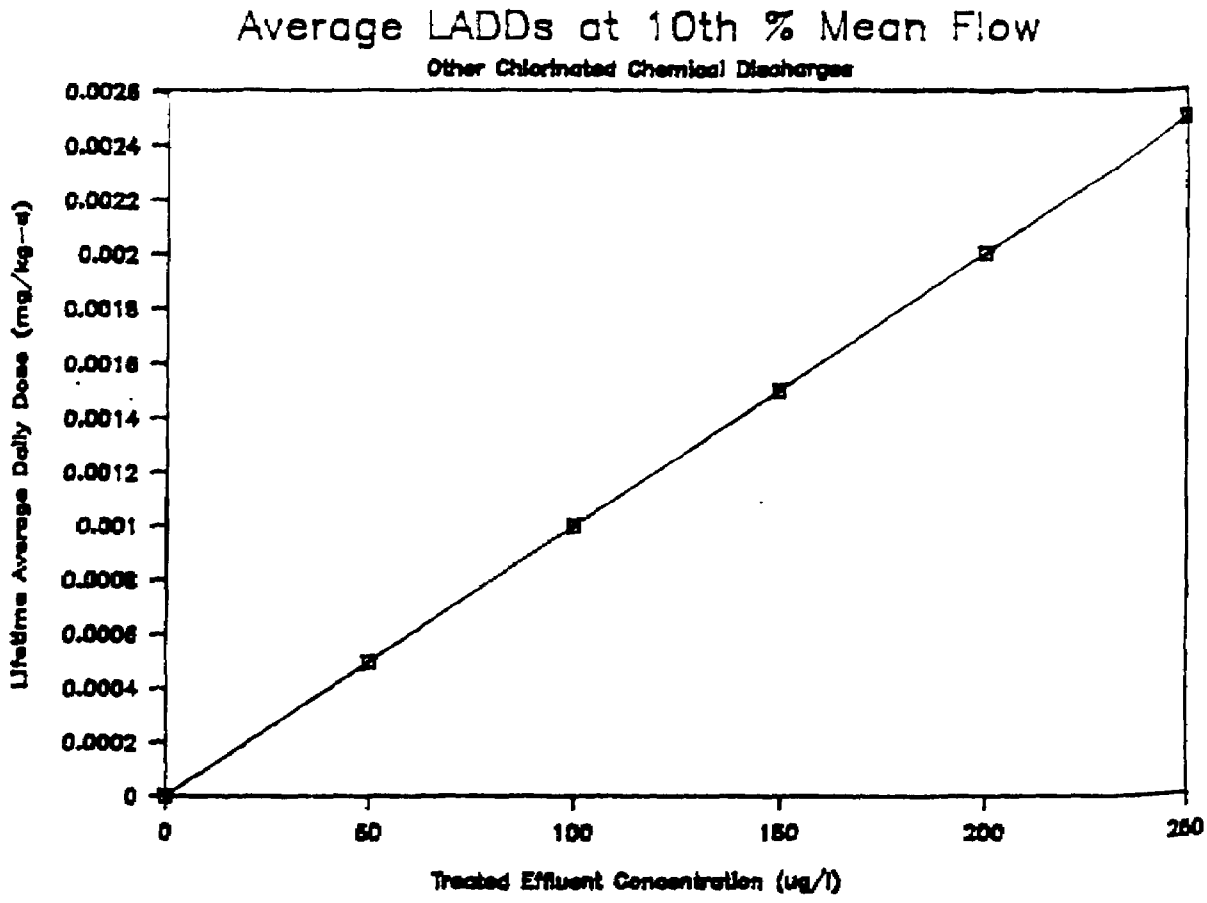


Figure B-4.

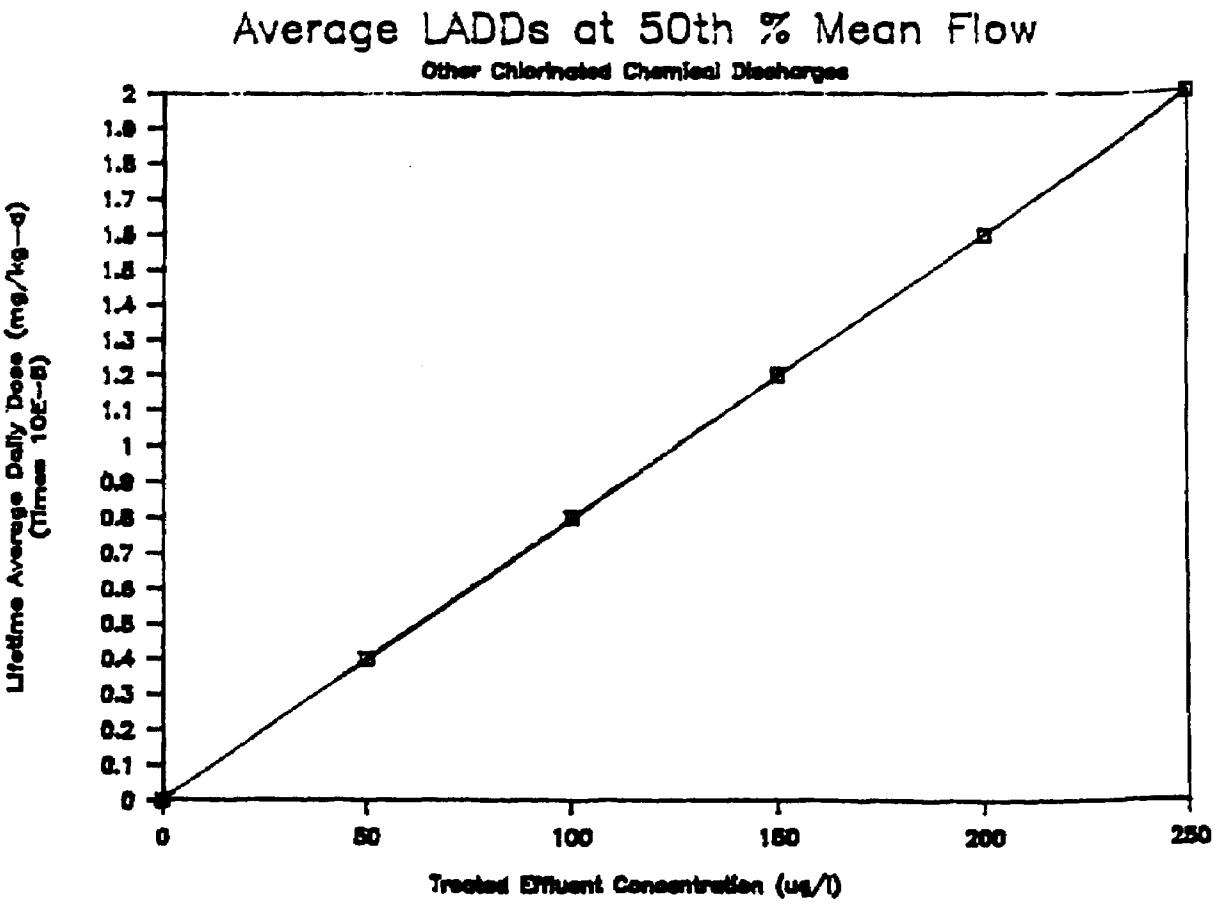


Figure B-5

Temporal Changes in 3,4,5-Trichlorocatechol Conc.

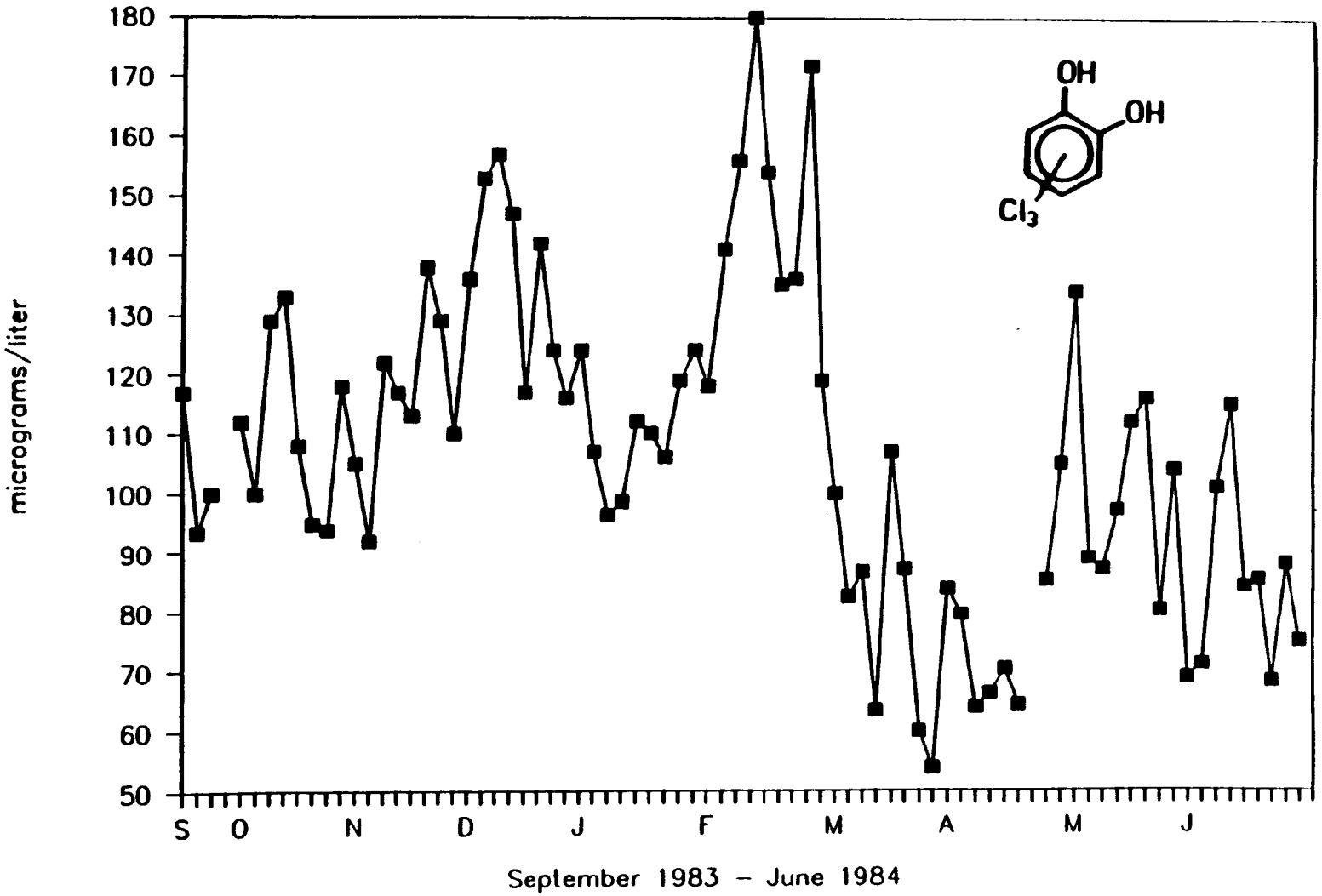
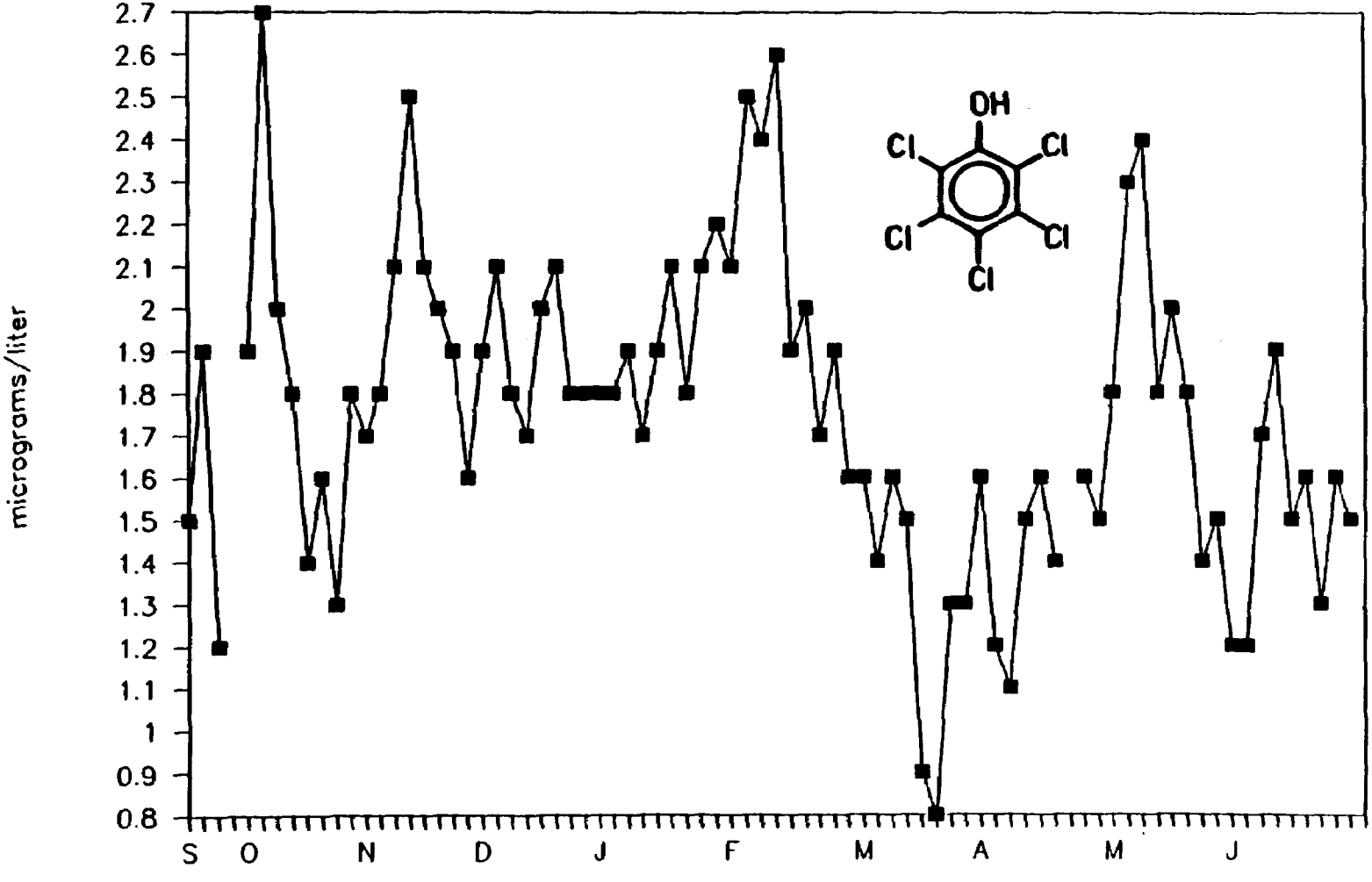


Figure B-6

Temporal Changes in Pentachlorophenol Conc.



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This report presents the detailed summaries of findings, assumptions, and uncertainties of an assessment of risks from exposure of humans, terrestrial and avian wildlife, and aquatic life to dioxins and furans formed during chlorine bleaching at kraft and sulfite pulp and paper mills. This report contains condensed versions of eight major exposure/risk assessments and other support documents prepared by program offices within the U.S. Environmental Protection Agency (EPA), the U.S. Food and Drug Administration (FDA), and the U.S. Consumer Product Safety Commission (CPSC). Risks were evaluated from roughly 120 potential pathways of exposure to pulp and paper products, pulp and paper mill sludges, and pulp and paper mill effluents. The development of this assessment and the individual Agency exposure/risk assessments was coordinated by the Federal Interagency Working Group in Dioxin-in-Paper.

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