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EPA

KANAWHA VALLEY TOXICS SCREENING STUDY FINAL REPORT

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION III
841 Chestnut Building
Philadelphia, Pennsylvania 19107

AUG 13 1987

TO ALL INTERESTED AGENCIES, ORGANIZATIONS AND INDIVIDUALS:

Enclosed is a copy of the Final Kanawha Valley Toxics Screening Study Report which was prepared by the U.S. Environmental Protection Agency in cooperation with West Virginia environmental agencies. The Report presents findings from a unique screening study of several environmental pollutants which are routinely emitted or present within the Kanawha Valley under normal conditions. The purpose of the study was to estimate the relative importance of these chemical pollutants by assessing their potential for causing long-term human health risks.

The procedure used for estimating risks contains many uncertainties and the numbers generated are by no means absolute. Nevertheless, the results are useful for comparing pollutants and sources and are intended to serve as a guide for EPA and West Virginia agencies to set priorities for future assessments.

The Toxics Screening Study provided important preliminary information for environmental agencies, industry, and the public. However , because the scope was very limited and only a few of several hundred chemicals were examined, EPA and other organizations will continue to study potential health threats and reduce unnecessary risks in the Kanawha Valley.

Although not required to take action as a result of this Report, several companies in the Valley which emit or discharge the pollutants studied have recently taken commendable strides to reduce releases of the higher risk chemicals. We hope these preventive actions will continue as we learn more about environmental risks.

I thank all of those who participated or have shown interest in this challenging exercise. If you should have any questions or comments concerning this Report, please feel free to contact Mr. Greene Jones, Director of Environmental Services Division, at the above address.

Sincerely,

James M. Seif
Regional Administrator

Enclosure

- FINAL -

KANAWHA VALLEY,

WEST VIRGINIA

TOXICS SCREENING STUDY REPORT

July, 1987

Prepared by:

U.S. ENVIRONMENTAL PROTECTION AGENCY

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Key individuals who were responsible for study management and content of the report include Greene A. Jones, Division Director, and Thomas Slenkamp of Region III's Environmental Services Division; and Samuel Napolitano and David Lee of the Regulatory Integration Division. Credit should also be given Robert Currie, formerly with the Regulatory Integration Division, as well as current Director Daniel Beardsley, and Stanley L. Laskowski, Deputy Regional Administrator, who collectively initiated study discussions within EPA and with the State of West Virginia and outlined initial study direction.

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Mr. Donald A. Kuntz, P.E., Department of Health
Mr. Lucas M. Neas, Department of Health

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Finally, four private contractors provided significant technical support for the Toxics Screening Study and deserve recognition:

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KANAWHA VALLEY TOXICS SCREENING STUDY REPORT

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NOTICE

This Report has been independently reviewed in public session by the Integrated Environmental Management Subcommittee of the Science Advisory Board (SAB). The SAB is an independent public advisory group providing scientific information and advice to the Administrator and other officials of the Environmental Protection Agency. The Board consists of nationally recognized experts representing a variety of scientific disciplines, and is structured to provide a balanced expert assessment of scientific matters related to issues facing the EPA. The IEM Subcommittee's final comments on the Kanawha Valley Toxics Screening Study, as well as a list of the Subcommittee members are included in Appendix C.

Progress and Compliance with Existing Environmental Laws

EPA and West Virginia agencies administer more than a dozen major environmental laws collectively aimed at controlling environmental pollution to acceptable levels. Considering the large number of industrial facilities operating in Kanawha Valley today (close to 200 of all types), there is reasonably good compliance with these laws (i.e., the large majority of facilities are operating in compliance all or most of the time). This compliance success has resulted in improved environmental conditions throughout the Valley:

- Air quality in the Valley consistently meets applicable primary Ambient Air Quality Standards for the six criteria air pollutants;

- The nineteen (19) major industrial facilities operate under approved National Pollutant Discharge Elimination System (NPDES) permits, and sixteen (16) of these are based on Best Available Technology (BAT) limits;

- All public drinking water supply systems are consistently in compliance with Maximum Contaminant Levels (MCLs) established under the Safe Drinking Water Act;

- Eleven facilities are required to have permits under the Resource Conservation and Recovery Act (RCRA) for handling, treatment, storage, or disposal of hazardous waste. Only one facility is currently not in compliance with RCRA regulations.

While the trend is clearly toward generally improved environmental conditions, there are occasional violations of established criteria resulting in periodic stress to environmental quality. EPA and the State of West Virginia will continue to vigorously enforce existing environmental laws to minimize these violations.

THE KANAWHA VALLEY TOXICS SCREENING STUDY

Although there is generally good compliance with current laws, many hundreds of chemicals routinely released within the Kanawha Valley environment are not regulated by these laws, partly because information is lacking concerning their toxicity, persistence, or fate (i.e., is the population exposed to significant levels of these compounds?). In order to help answer these questions, it was recognized that more information was needed to begin to assess the possible risks posed by some of these hundreds of chemicals.

EXECUTIVE SUMMARY

Introduction

This summary briefly explains the purpose, content and findings of the environmental report on Kanawha Valley, West Virginia which follows. The report was primarily prepared to describe a limited study about toxic chemicals in the Valley undertaken by the U.S. Environmental Protection Agency (EPA), in cooperation with three West Virginia environmental agencies - the Air Pollution Control Commission, Department of Health, and Department of Natural Resources. The report also contains summary information concerning the area's environmental compliance with current environmental laws, and includes, in Appendix A, a brief description of some other ongoing activities concerning Kanawha Valley environmental issues.

Why the Kanawha Valley Was Studied

The Kanawha Valley represents a unique geographical setting of mountainous terrain with winding streams and valleys, and widely variable meteorological conditions. It also includes a large concentration of industrial facilities, with residential population often located in close proximity to these facilities. These features in combination make the protection of public health and the environment a great challenge. During the past 25 years, this challenge has been met by a cooperative effort of government and industry, and has resulted in substantial improvement to the environmental quality of the Valley.

At the same time, in the Kanawha Valley, as in many other locations nationwide, there remain complex environmental challenges which are difficult to fully address, including the question of whether, and to what degree, low levels of chemical pollutants present in the environment may cause unacceptable health and environmental risks.

Because the Kanawha Valley includes many chemical manufacturing and handling facilities which routinely release small quantities of potentially toxic chemicals to the outside environment, Valley residents and officials have long been concerned with the possible health impacts associated with long term exposure to these chemicals. Recent scientific developments have for the first time enabled the Environmental Protection Agency and other organizations to quantify and compare potential health risks from certain chemical contaminants, using an approach termed risk assessment. This capability was applied to the Kanawha Valley to begin to assess some of the chemical exposures and possible health risks which may occur within the Valley.

Objectives of the Study

The Kanawha Valley Toxics Screening Study was designed to provide an initial understanding of selected toxic pollutants which are present in the ambient environment of the Kanawha Valley, to identify possible sources of these pollutants, and to provide a sense of the nature and relative priority of potential health risks associated with public exposure to these chemicals.

Study Scope

Perhaps the most important point to understand about the study's scope is that it was intended to provide only a limited understanding of a few possible environmental risks within certain areas of the Kanawha Valley. Our intent was not to comprehensively analyze all available environmental data in order to determine whether or not there is a significant cancer or other health risk from environmental pollution, nor to identify all possible environmental problems which might be present in the Valley. While we did analyze several issues in moderate depth of detail, the study was not comprehensive in scope. It was thus not a "screening" study in a broad or literal sense. That is, it did not attempt to discover all possible toxic risks from all sources within all potential exposure pathways.

What the study attempted to provide was an initial understanding of potential risks from current ambient exposures for a few selected chemicals, so that residents and officials could begin to understand the nature of some risks present in the Valley and could set priorities for further analysis of risks, and, where appropriate, control actions.

Omitted from this study were several types of environmental risks, including those from cigarette smoking and diet, from indoor air exposures, from occupational exposures, and from accidental releases of chemicals. Although these are all potentially important exposure pathways, with limited funding and the need to rely on existing data, we decided that the screening study could best meet objectives by focusing on long term health risks posed by average ambient exposure to selected chemicals.

The Toxics Screening Study examined potential community exposure to several toxic pollutants through four exposure pathways by which contact could conceivably occur: through (1) inhalation of ambient (outdoor) air, (2) consumption of public drinking water, (3) consumption of fish from the lower Kanawha River, and (4) consumption of groundwater which could be contaminated by hazardous waste sites.

In the Air Analysis, twenty (20) known or suspected cancer-causing (carcinogenic) chemicals (14 from point sources) were evaluated out of more than 450 chemicals listed on an inventory of Kanawha Valley air emissions prepared by the West Virginia Air Pollution Control Commission. Potential community exposure to these 20 chemicals was estimated through mathematical modeling within four distinct geographic zones: Belle, Charleston/South Charleston, Institute, and Nitro.

For Drinking Water, nineteen (19) chemicals for which monitoring is required were evaluated from the study area's fourteen public water supply systems. Both potential cancer risks and non-cancer effects were evaluated. Private wells and non-community supplies were not included.

The Surface Water analysis focused on potential health effects, both cancer and non-cancer, from the possible consumption of contaminated fish taken from the Kanawha River near Nitro. In addition, concentrations of chemicals monitored within the River were compared with water quality criteria established for the protection of human health and aquatic life.

The Hazardous Waste analysis examined data from known hazardous waste sites in the Kanawha Valley in order to devise hypothetical hazardous waste settings (i.e., combinations of various facility types, hydrogeology, and waste constituents) which could present a risk to human health through public consumption of contaminated ground water. It did not include any site-specific health risk estimates.

Chemicals were selected for study within each media analysis primarily on the basis of the following criteria: (1) their known or estimated quantities or concentrations within the Kanawha Valley; and (2) sufficient information about their health effects to enable a reasonable estimate to be made of potential risk. Pollutants studied do not necessarily represent those posing the highest risks within the Valley.

A large number of man-made chemicals are present or routinely released to the environment of Kanawha Valley. However, because information concerning the above criteria was often lacking or limited, we were only able to roughly evaluate potential risks for a few of those chemicals.

Because the study originated out of concern over air toxics, the air portion of the study received by far the majority of attention and allocation of available resources. We estimate that approximately 80% of study effort was devoted to the air analysis, 10% to the hazardous waste analysis, and 5% each for the drinking water and surface water analyses. This distribution of effort should not be construed as correlating with the actual distribution of environmental problems by media in the Valley.

Study Methodology and Limitations

The Kanawha Valley Toxics Screening Study used a risk assessment screening methodology to evaluate and compare, in a limited fashion, potential human health risks from current ambient exposure to a limited set of pollutants. The purpose of this methodology was to permit a comparison of one risk with another and to provide a general sense of the risk a given chemical substance may present. This methodology does not permit a definitive statement concerning the absolute risk posed by a particular pollutant, source, or exposure pathway.

The two key elements in estimating risk are a chemical's estimated potency, or toxicity, and human exposure to that chemical. EPA estimates a chemical's toxicological potency on the basis of available scientific evidence. Exposure to a chemical is estimated by measuring or estimating the concentration at which a chemical is present in the air or water, for example, and then making assumptions about how much air a person breathes or water he or she drinks. Potency and exposure estimates are combined to estimate individual and population risks.

The Toxics Screening Study examined potential cancer risks, and, in a more limited way, non-cancer risks where possible. For cancer risks, available scientific data allowed for a quantitative estimate of potential risks. For non-cancer effects, a qualitative analysis was performed, simply indicating whether or not estimated exposure appeared to be high enough to place an individual at increased risk of an adverse health effect.

For this analysis, risk to an individual is defined as the increased probability that an individual exposed to one or more chemicals will experience a particular adverse health effect (e.g., will contract cancer) during the course of his or her lifetime. The risk estimated for a particular type of exposure is the incremental risk beyond that which a person faces from exposure to other environmental or hereditary causes of disease, sometimes referred to as the background rate of disease. Calculated risks are considered plausible, upper-bound estimates of potential risk rather than predictions of most likely risk.

For cancer effects, two types of individual risks were estimated: (1) average individual risk, for a typically exposed individual, and (2) for the air analysis only, risks to an individual who may be particularly close to a source or who is likely to receive higher than average exposure. Risk to the population as a whole is expressed as the plausible, upper-bound increased incidence (number of cases), above the background rate, within an exposed population.

Despite the application of similar risk assessment techniques, each media analysis differed in terms of geographic coverage, chemicals studied, whether ambient concentrations were measured or estimated, the potential health risks considered (cancer or non-cancer), and in the means of expressing the risks studied (e.g., risk assuming uniform ingestion of drinking water versus risk per pound of fish consumed). Table 1 illustrates some of these differences.

TABLE 1

DIFFERENCES IN RISK ASSESSMENT APPROACHES[1]
BY MEDIA ANALYSIS

MEDIA STUDIED	PRIMARY ROUTE OF EXPOSURE ANALYZED	RISK ASSESSMENT APROACH			
		Exposure Data		Health Effects Considered	
		Measured	Modeled	Cancer	Non-Cancer
Air Analysis	Inhalation of Ambient Air		X	X	
Drinking Water Analysis	Ingestion of Public Drinking Water	X		X	X
Surface Water Analysis	Consumption of Contaminated Fish	X		X	X
Hazardous Waste Analysis	Ingestion of Contaminated Ground Water		X	X	X

[1]In addition to these differences, the area of geographic coverage and types of chemicals studied for each media analysis were different, as was the means of expressing the risks studied - see text.

The application of risk assessment techniques in a complex geographic area such as the Kanawha Valley is difficult because there are many scientific and technical limitations. For example, there are uncertainties in estimating the amount of pollutants released from a given source, in estimating the extent of exposure of the general public to a particular pollutant(s), and in extrapolating health effects evidence derived from animal studies to estimate corresponding human responses. In general, the uncertainties introduced by the exposure data are usually much smaller than those associated with the dose response information used to estimate human health risks.

Because of these uncertainties, EPA prefers to err on the side of caution in making risk assessments. Therefore, following standard EPA protocol, we use conservative estimates of risks that probably overestimate the actual risk from any one chemical. While the risks outlined in the study should not be interpreted as a definitive prediction of actual cancer risk to the general public in the Valley, these estimates are useful in comparing the relative risks among various chemicals.

SUMMARY OF STUDY FINDINGS

Considering the approach and limitations noted above, following is a summary of the study's major findings:

Air Analysis

A total of 20 chemical carcinogens were evaluated within the four air zones (Belle, Charleston/South Charleston, Institute, and Nitro), although only 14 of these are emitted from specific point sources (e.g., industrial facilties). Not all chemicals are emitted in all four zones. For the study area in general, point sources accounted for the great majority of risks studied, while areawide, or non-point sources (e.g., automobiles, dry cleaners, gasoline marketing) contributed relatively little.

Within the Belle zone, the increased lifetime cancer risk for an average individual from exposure to the chemicals studied was estimated to be 2.5×10^{-4}, or two and one half chances in ten thousand of contracting cancer, aside from other causes. Within certain neighborhoods nearby major industrial facilities, the increased individual risk could be as high as 3×10^{-3}, or three in one thousand. That is, at most, 3 out of every 1000 people living within these Belle neighborhoods could contract cancer as a result of lifetime inhalation of the chemicals studied. Table 2 portrays these risk results by zone.

Within the Charleston zone, the increased lifetime cancer risk to an average individual from exposure to selected chemicals was estimated to be 2.9 in 10,000. Within certain neighborhoods within the Charleston zone, this risk could be as high as 5 in 1000. Within Institute, average individual risk was estimated to be 1.1 in 1000, while in certain locations individual risk could approach 1 in 100. For those chemicals studied in the Nitro zone, estimated air risks were considerably less than in the other three zones.

By assuming that all 20 of the air pollutants studied were evenly dispersed throughout all four Valley zones, a box modeling approach conservatively estimated that an additional 126 cancer cases could result over a 70 year period of exposure.

* *
* Note: The numerical health risk estimates presented herein are based *
* on conservative assumptions which tend to produce an upperbound, or *
* maximum, value. Actual risks are not likely to be any higher; however,*
* they could be significantly lower, and may even approach zero. Because*
* of the many uncertainties and limitations in the methods used to *
* calculate risks, the risk estimates should not be considered defini- *
* tive predictions of actual health risk. The proper function of the *
* estimates is to help responsible officials select issues and set *
* priorities for future study. *
* *

TABLE 2

AIR ANALYSIS RESULTS BY ZONE

ZONE	POPULATION AFFECTED Total in Zone	Nearby Facilities	POLLUTANTS CONTRIBUTING MOST TO RISK	ESTIMATED UPPER BOUND INDIVIDUAL LIFETIME CANCER RISK[1] Average	Nearby Facilities
Belle	15,530	600	Chloroform	$<2.5\times10^{-4}$	$<3\times10^{-3}$
Charleston/ South Chas.	51,750	2700	Ethylene Oxide Acrylonitrile	$<2.9\times10^{-4}$	$<5\times10^{-3}$
Institute	22,390	1300	Ethylene Oxide 1,3-Butadiene	$<1.1\times10^{-3}$	$<8\times10^{-3}$
Nitro	9,990	1450	Trichloro-ethylene	$<2.6\times10^{-6}$	$<6\times10^{-6}$
	99,660	6050			

[1] Risks presented are a sum of the risks from individual chemicals studied within a given zone

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

Drinking Water Analysis

Estimated individual cancer risks from certain chemicals studied which are present in public drinking water supplies were approximately 1 in 10,000. For the total population served by these systems, this risk would mean an additional 46 cancer cases over a 70 year period of exposure. The primary chemical of concern is chloroform, a byproduct formed during the disinfection process, and one of a class of compounds called trihalomethanes (THMs). THMs are formed as the result of chlorination of drinking water, which is used to prevent outbreaks of waterborne bacterial diseases. All Kanawha Valley public water supplies studied currently meet the EPA standard for THMs. If THMs do not exceed the EPA standard, they are generally considered to be an acceptable risk.

For non-cancer risks, public drinking water data available for nineteen chemicals was compared with the corresponding threshold levels (Reference Doses, or RfDs), below which adverse health effects are unlikely to occur. None of the pollutants studied exceeded the RfDs. In addition, an analysis of finished water for 129 priority pollutants done for the West Virginia Water Supply Company in July, 1985 revealed no appreciable contamination. We did not have similar data available for the smaller systems (<10,000 people served).

Among several limitations in this analysis was our inability, due to a lack of data, to characterize risks posed by private wells, which by one estimate number 22,000 in Kanawha County alone.

Surface Water Analysis

Fish tissue samples taken from the Kanawha River near Nitro in September, 1985 showed elevated levels of several chemical compounds, including PCBs, pesticides and dioxin. As a result, the State of West Virginia issued a fishing advisory for a stretch of the Kanawha River extending from the Coal River downstream to the Ohio River. Although actual fish consumption rates are unknown, our study revealed that risks could be significant if fish are regularly consumed from this area of the Kanawha River. For example, consumption of from one tenth to one pound per week of certain species over a lifetime could result in an increased risk of contracting cancer of 1 in 1000.

Analysis of non-cancer risks was difficult, since very few pollutants studied in the Surface Water Analysis have verified Reference Doses with which to compare. However, based on limited data, we found that consumption of between 3/10 and 2 pounds of fish per week, depending on the species, could exceed the Reference Dose for chlordane, resulting in an increased probability of chronic, non-cancer health risks. Also, consumption of as little as 1 pound per week of certain species could result in exceedance of the RfD for methyl mercury at the highest observed concentrations. For some species, these pollutants were only found at significantly lower levels.

When we compared concentrations of metals measured in the Kanawha River to water quality criteria for protection of aquatic life, we found that lead and cadmium exceeded the criteria for 44 and 30 percent of the observations, respectively. In general, however, the chemicals for which monitoring data was available only infrequently exceeded any water quality criteria.

Hazardous Waste Analysis

Although there are many potential hazardous waste sites in the Kanawha Valley, preliminary risk assessments have been conducted at only some of these sites. Active programs to clean up and manage hazardous waste at specific sites are underway at EPA and the State.

For the Toxics Screening Study, representative hazardous waste site conditions were formulated to gain a sense of some risks which could be associated with different conditions. Based on limited data, the study identified potential risks which could occur, under hypothetical conditions, by consumption of ground water contaminated by hazardous waste. Under the worst case analyzed, potential individual cancer risks could approach one in a thousand. Risks were not estimated for any actual, specific sites in the study area. The established EPA and State programs for controlling hazardous waste are currently evaluating whether ground water might be contaminated near any specific sites.

Overall Comparison of Risk Estimates

As indicated earlier, a precise estimate of risk from any one pollutant is difficult to determine due to limitations concerning emission levels, exposure, health effects, and other factors. However, the study was valuable in quantifying potential cancer risks from selected pollutants which allows for comparing these risks to get a sense of the relative importance of the chemicals studied. Table 3 presents the chemicals studied which resulted in the highest cancer risk estimates. Other chemicals not studied and those associated with other than cancer effects would not be shown here, but are nonetheless also of possible concern.

Comparison of Risks with Other Areas of the Country

It is difficult to compare risks between different geographic areas. Studies done for different regions with different objectives often have exposure and risk measures that are not easily comparable. However, EPA has estimated some ambient concentrations and risks associated with certain chemicals in different areas of the country. Where those chemicals and exposure assumptions were similar to those studied in Kanawha Valley, we have attempted to present a comparison of estimated levels and risks in Chapter Eight.

In general, the ambient concentrations and estimated risks from many of the toxic chemicals which we evaluated in the Kanawha Valley appear to be within the range we see in other areas where EPA has data for similar individual pollutants. However, we see concentrations in the Kanawha Valley at both the high and low end of this range.

The risk associated with drinking public water in the Kanawha Valley appears to be about the same as in other cities in the United States which also use surface water as a source. However, within the air analysis, many risks estimated in Kanawha Valley appear to be appreciably higher than comparable point source pollutant risks evaluated by EPA in national air toxics studies.

Future Direction

The principal goal of this study was to assess some of the environmental risks in the Kanawha Valley, in order to help set priorities for future study and for managing those risks. A review of the analytical findings and discussion within each media report suggests two major priorities for future action: (1) a need to take steps to reduce the significant risks identified, and (2) a need for continued analysis of other chemicals, health effects and risks in the Valley.

Although there were many uncertainties and limitations in this study, in certain instances the potential risks calculated are sufficiently high to suggest that action to reduce the risks should be initiated by EPA, the West Virginia regulatory agencies, and/or industry. Where necessary, these risk management actions can be accomplished through government regulation or through voluntarily negotiated reductions with the responsible industries.

Risk management is a process which involves residents, industry, and government, and which includes many considerations beyond simply the risk assessment results, such as available control methods, costs, benefits, public opinion, and legal authority. All of these factors must be evaluated before deciding what represents an acceptable level of risk and what are the best means and tradeoffs for reducing an existing risk.

TABLE 3

KANAWHA VALLEY CHEMICALS STUDIED
CONTRIBUTING MOST TO ESTIMATED CANCER RISKS

CHEMICAL	EXPOSURE PATHWAY	GEOGRAPHIC AREA	PROBABLE SOURCE
Potential Risks*on the Order of 1 in 1000 (within specific locations):			
1,3 - Butadiene	Air	Institute	Union Carbide/ Rhone-Poulenc
Ethylene Oxide	Air	Charleston/ South Chas.	Union Carbide-South Charleston
Chloroform	Air	Belle	Occidental[2]
Ethylene Oxide	Air	Institute	Union Carbide/ Rhone-Poulenc
PCBs and Dioxin[3]	Surface Water (Fish Tissue)	Nitro	Unknown
Potential Risks*on the Order of 1 in 10,000 (within specific locations):			
Acrylonitrile	Air	Charleston/ South Chas.	Union Carbide-South Charleston
Methylene Chloride	Air	Belle	Occidental[2]
Chloroform (THMs)	Drinking Water	Water Supply Service Areas	Public Water Suppliers
Acrylonitrile	Air	Institute	Union Carbide/ Rhone-Poulenc
Carbon Tetrachloride	Air	Belle	Occidental[2]
Chloroform	Air	Institute	Rhone-Poulenc[1]

[1] Formerly Union Carbide
[2] Formerly Diamond Shamrock
[3] Assumes consumption of 1/10 lb/week of contaminated fish over a lifetime; Higher consumption rates would result in higher risks

*NOTE: BECAUSE OF SIGNIFICANT UNCERTAINTIES IN THE UNDERLYING DATA AND ASSUMPTIONS, THESE ESTIMATES OF INDIVIDUAL RISK ARE ONLY ROUGH APPROXIMATIONS OF ACTUAL RISK. THEY ARE BASED ON CONSERVATIVE ESTIMATES OF EXPOSURE AND POTENCY, AND ARE MORE LIKELY TO OVER-ESTIMATE RISKS THAN TO UNDERESTIMATE THEM. See Text.

Source: Regulatory Integration Division, EPA, 1987

EPA, in cooperation with West Virginia agencies, will continue to reduce risks, where possible, by maintaining compliance with existing environmental laws. Provisions for control of certain toxic chemicals are available, for example, in the Clean Air Act for designated Hazardous Air Pollutants (HAPs), through the Toxic Substances Control Act if an "unreasonable" health risk is identified, by limiting toxic discharges in point source surface water (NPDES) permits, and by enforcing the Maximum Contaminant Levels (MCLs) set for certain drinking water pollutants.

Where identified risks are not controllable through existing Federal laws and programs, the State of West Virginia could independently pursue a regulatory approach to control toxic pollutants in a variety of ways (e.g., risk-based, control technology based, specified ambient levels), or can continue to negotiate, with EPA assistance, with specific industries on a voluntary basis to achieve further emission reductions. In the past two years, major industries in the Valley have voluntarily committed to reducing emissions of several toxic air pollutants, and have recently moved to further decrease emissions based on results of this Study.

The Kanawha Valley Toxics Screening Study can be viewed as an initial step in a continuing process to assess and, where necessary, reduce risks. Many chemicals, health effects, and types of risks not considered in the Screening Study (e.g., acute health effects, accidental releases) may be individually or collectively important and may merit further attention. The Study identified many scientific uncertainties and data needs that may be appropriate research and management priorities for followup by EPA and West Virginia agencies and other groups. Future direction should include, in addition to reducing risks from chemicals identified in the Study, further analysis of these additional topics.

EPA is now working with West Virginia's environmental agencies to develop specific plans for responding to the risks and needs identified in this Study. Some of the possible activities include the following:

Air - in addition to the initial priority of reducing emissions for those chemicals posing significant risks, the following activities would contribute to a better understanding of air toxics risks in the Kanawha Valley:

1. Improve the emissions inventory. Although the Kanawha Valley inventory represents state-of-the-art estimation techniques, some of the uncertainties can be further reduced.

   (a) Ensure emission estimates reflect documented voluntary reductions committed to by industry from 1984 production levels.

(b) Apply recently developed and EPA approved plant-specific methods for more accurately estimating contributions from fugitive (dispersed) sources.

(c) Include estimates of short-term variable emissions to enable future analysis of possible acute and sub-chronic health effects.

(d) Prioritize chemicals listed on the inventory into levels of concern for future study, based on emission quantities, toxicity, and potential for exposure.

2. Improve methods for measuring air toxics (especially for those posing significant risks) and conduct further ambient monitoring within selected areas to obtain more precise estimates of exposure and health risks.

3. Develop an approach for estimating non-cancer health risks to complement the quantitative analysis of cancer risks.

4. Perform additional risk assessments to include additional chemicals and short-term effects, and using the most recent health effects information.

5. Evaluate potential risks from accidental releases.

Drinking Water - the most pressing need appears to be for a survey of the location and water quality of private drinking water wells in the area, some of which could be impacted by hazardous waste sites. Also, testing of smaller (<10,000 people served) public systems for trihalomethanes and additional toxic contaminants would be useful.

Surface Water - additional ambient monitoring of Kanawha River water quality and further fish tissue analysis would be helpful to establish whether the elevated concentrations of PCBs, dioxin, chlordane, methyl mercury, and other chemicals found near Nitro are also present in other River reaches. Knowledge of fish consumption rates and patterns would allow for a better estimate of potential risks. West Virginia's Department of Natural Resources' creel survey should provide this latter type of information.

Hazardous Waste - the efforts to assess and cleanup specific sites should continue under the established Federal and State hazardous waste programs. From the Toxics Screening Study, sites in the shale/sandstone hydrogeologic regime tend to present the greatest potential health risks due to the increased probability of private well contamination.

In addition to the Toxics Screening Study, there are many other programs and activities underway in the Valley and in the Nation collectively aimed at better understanding and reducing risks to public health and the environment. Some of these activities are described in Appendix A.

Implementation of these efforts and the risk assessment analyses presented in this report provide a resevoir of valuable information for government, industry, and citizens to work toward continued environmental improvement in the Kanawha Valley.

Chapter One

Introduction

CHAPTER ONE

INTRODUCTION

I. BACKGROUND AND ENVIRONMENTAL ISSUES IN KANAWHA VALLEY

The Kanawha River Valley in central West Virginia, from Gauley Bridge to Winfield Dam (see Figure 1-1), has often been an area of environmental concern, principally due to its heavy concentration of chemical and other industrial facilities situated in close proximity to populated areas, and also because of unique topographical and meteorological conditions.

Beginning with the development of the chemical industry in the Valley in approximately 1915, and continuing through several decades of industrial expansion and accompanying population growth, the area often experienced severe environmental stress.

Large volumes of municipal and industrial waste and other pollutants were discharged to the Kanawha River resulting in gross pollution. At times, the River was unable to support even pollution tolerant fish populations. Taste and odor problems were severe in public water supplies obtained from the River. Beneficial uses of the River were severely impaired.

Degradation of air quality was also a major problem. Emissions of air pollutants from chemical plants, powerplants, and other manufacturing facilities, and from automobiles and localized sources in heavily populated areas, were often trapped in the narrow Valley by adverse meteorological conditions. This often resulted in severe air pollution episodes in the populated areas.

Large volumes of hazardous waste and other waste residuals were disposed of in landfills, dumps, and surface impoundments that were not properly designed, constructed, or maintained to adequately contain the toxic substances present in the waste. As a result, toxic pollutants were released in the air, to surface water, and to ground water.

Beginning in the 1960's, major new Federal, State, and local environmental laws and programs were initiated, and municipalities and industries invested major capital expenditures in plant improvements and pollution controls. During the last 25 years, these combined efforts have improved environmental quality in the Kanawha Valley and elsewhere throughout the country. Many of the most obvious and pressing problems were greatly reduced, or even eliminated, by removing large amounts of conventional pollutants from the air and water. As a result, the risks faced by the public and the environment from these highly visible forms of pollution have been greatly reduced.

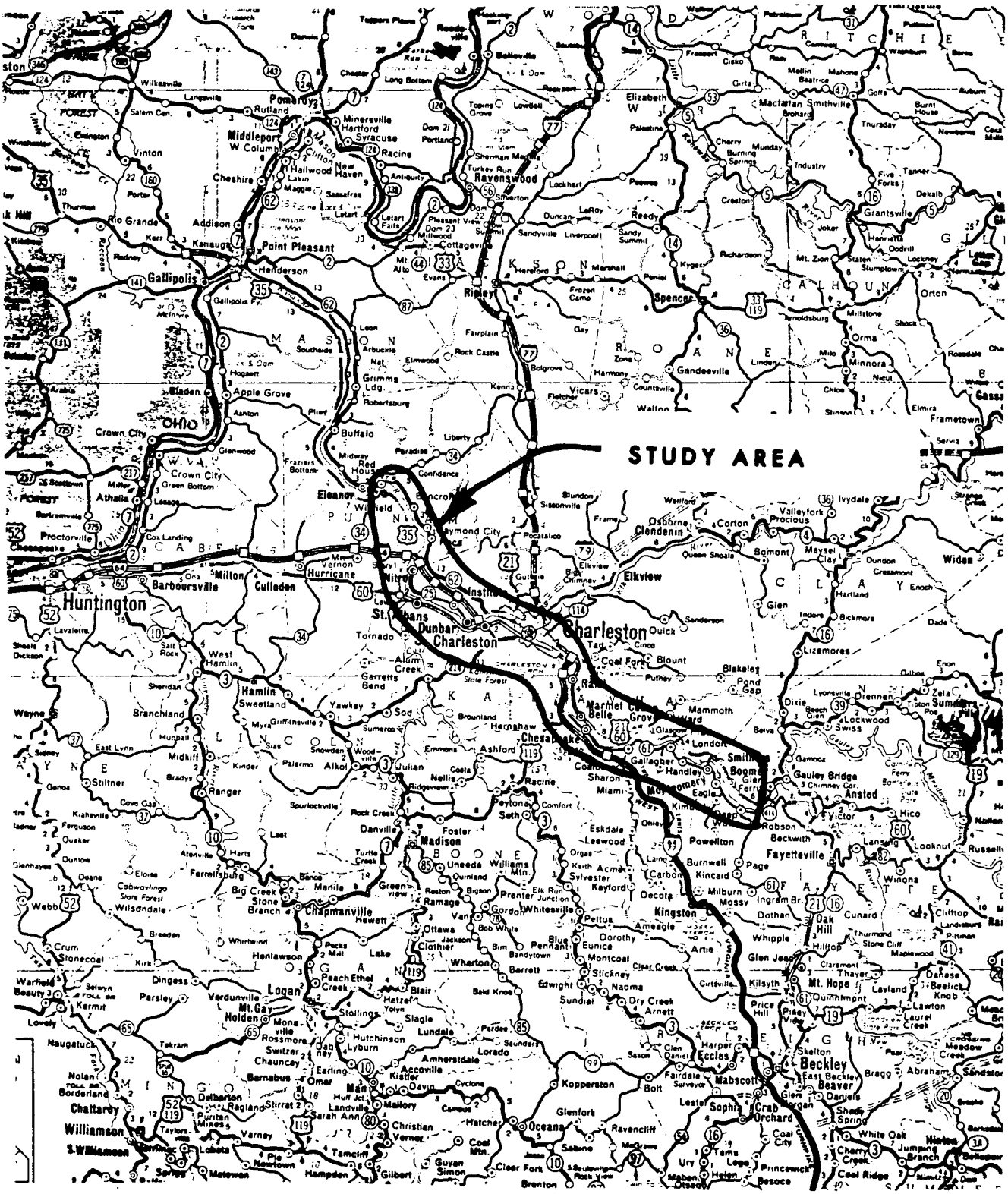

Figure 1. Location Map - Kanawha Valley Study Area

Most of the pollution controls implemented to date have concentrated on removing or reducing releases of "conventional" pollutants such as oxygen demanding substances in water and suspended particulates in air. The Kanawha River now meets most applicable water quality criteria most of the time. Sport fish are again present in the River. Similar improvements in air quality have also been achieved, with significant observed reductions of major ("criteria") air pollutants (ie., particulate matter, carbon monoxide, sulfur dioxide, nitrogen dioxide, lead, and ozone) within the Kanawha Valley Air Quality Control Region.

Although much environmental progress has occurred and conditions have generally improved, there remain several issues of concern to environmental agencies and the public which have not been fully addressed. There remain today in the Valley nearly 200 industrial and manufacturing facilities, about 20 of which are considered to be major sources of toxic substances. Although most existing regulatory limits for conventional pollutants are usually met, occasional violations do occur, indicating the need for continued oversight and enforcement, where warranted.

II. PROGRESS IN CONTROLLING POLLUTION

In July of 1986, EPA Region III prepared a draft report covering existing environmental conditions and compliance with current environmental laws within the Kanawha Valley, West Virginia. This compliance status report was a follow-up to an August, 1984 report prepared by EPA's National Enforcement Investigations Center (NEIC) entitled "Overview of Environmental Pollution in the Kanawha Valley". Together, these two reports evaluated the prevailing environmental conditions in the Valley, identified pollutants of particular concern, and located the major sources from which these pollutants were being emitted.

While these reports concluded that major improvements in environmental quality were being made when "conventional" pollutants were assessed, they also identified potentially significant concerns when "non-conventional" pollutants (toxic substances and carcinogens) were considered. It is largely as a result of these findings that EPA decided to undertake the study of toxic pollution described later in this Report. This Section will provide a synopsis of the findings discussed in the July, 1986 compliance status report.

Existing Environmental Programs and Compliance

As noted earlier, industrial development and population growth in the Kanawha Valley sometimes resulted in environmental degradation. Such degradation was common to large industrial centers throughout the United States and was a major reason for the passage of new and expanded environmental legislation in the 1960's and 1970's. As a result of these control programs, major improvements in air and water quality have been realized over the past 25 years.

For example, due to the development of National Ambient Air Quality Standards (NAAQS), and State Implementation Plans, which outline control strategies necessary to assure compliance with NAAQSs, present air quality in the Valley is in compliance with all applicable primary ambient air quality standards.

Similarly, as a result of permitting programs such as the National Pollutant Discharge Elimination System (NPDES) under Section 402 of the Clean Water Act, water quality in the Kanawha River has made a dramatic improvement from the gross pollution of the past. All of the nineteen major facilities evaluated in EPA's 1986 compliance report operate under approved NPDES permits, and sixteen are based on Best Available Technology (BAT) limits.

Even though 100% compliance rates have not been achieved (ten facilities were out of NPDES compliance at the time of the report), water quality parameters necessary to support aquatic life have improved. In addition, concentrations of arsenic, copper, cadmium, cyanide, lead, silver, and zinc all declined significantly between 1970 and 1984.

However, certain water quality criteria are still exceeded (e.g., silver, arsenic, chloroform, carbon tetrachloride, dichloroethene, etc), which indicates a continuing environmental problem. Also, even while general conditions are improving, there are still major concerns.

One of the most significant recent findings has been high concentrations of dioxin, PCBs, and chlordane found in fish tissue near Nitro, which prompted the State of West Virginia in March, 1986 to issue a fish consumption advisory for a stretch of the lower Kanawha River. These problems notwithstanding, when compared to the quality of the River prior to the issuance of environmental regulations, there has been significant improvement.

Several environmental programs have been established which will help to continue these improvements. The permit program established under the Resource Conservation and Recovery Act (RCRA) helps to protect both groundwater and surface water from hazardous waste facilities including landfills. Of the eleven facilities in the Valley required to have a RCRA permit, only one facility is not in compliance with RCRA interim status. The Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), amended in 1986, will play a major part in enhancing environmental conditions in the Valley, where 100 potential hazardous dumpsites have been discovered.

Monitoring programs have also been established to assess compliance with the various laws. For example, in addition to regular air and water quality monitoring programs, five industries in the Valley are also monitored for compliance with Toxic Substance Control Act (TSCA) requirements concerning storage, use, or disposal of PCBs. Likewise, in accordance with the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), eleven facilities in the Valley that produce or hold pesticides for distribution are monitored for compliance with EPA requirements. All facilities inspected at the time of the compliance report were in compliance with TSCA and FIFRA regulations.

Table 1-1, taken from the 1986 compliance report, indicates the compliance status of the major Kanawha Valley facilities with various environmental programs (at the time of the report).

Current Issues

Recent environmental attention has focused on potentially toxic chemical substances present in the environment in low concentrations, for which few regulatory limits have been established. Various volatile organic chemicals, some with known or suspected human health effects at relatively low concentrations, are known to be emitted to the atmosphere from area industries and have been detected in the ambient air of the Valley. Collection of air monitoring data has recently begun, but there are few Federal or State regulatory limits for these chemicals from which a comparison can be made.

The two Kanawha Valley compliance reports noted above identified fish tissue contamination by dioxin, PCBs, and chlordane. Up to 100 potential hazardous waste dumps were identified. Surface and groundwater contamination by toxic substances was also suspected from the data that had been reviewed, and releases of toxic airborne pollutants were indicated. Although an estimated 10 percent of the population in the Kanawha Valley depends on groundwater as a drinking water source, the qualtiy of groundwater and possible threat of contamination is not well known. All of these findings indicate potential health hazards to residents in the Valley.

TABLE 1-1

MULTI-MEDIA COMPLIANCE PROGRAM SUMMARY

FACILITY	AIR (TSP & SO_2 Only)		WATER 4th Qtr 1986		RCRA		TSCA		FIFRA	
	IN COMP.	OUT OF COMP.	IN COMP.	OUT OF COMP.	IN COMP.	OUT OF COMP.	IN COMP.	OUT OF COMP.	IN COMP.	OUT OF COMP.
Allied Chemical, Nitro	X		X		N/A		N/A		N/A	
Appalachian Power, St. Albans	X		X		N/A		N/A		N/A	
Appalachian Power, Glasgow	X		X		N/A		N/A		N/A	
Coastal Tank Lines, Nitro	N/A		X		N/A		N/A		N/A	
Chemical Leaman, Institute	N/A		X		N/A		N/A		N/A	
CST/Fike Chemical (Artel) Nitro (2 RCRA Permits)	N/A			X		X,X	N/A		X	
Diamond Shamrock (Occidental), Belle	N/A		X		N/A		N/A		N/A	
DuPont, Belle	X		X		X		N/A		X	
Elkem Metals, Alloy		X		X	N/A		N/A		N/A	
FMC, Nitro	X		X		X		N/A		N/A	
FMC, S. Charleston	X		X		X		N/A		X	
Hatfield-Henson, Dunbar	N/A		X		N/A		N/A		N/A	
Kinkaid, Nitro	N/A		X		N/A		N/A		X	
Mason & Dixon, St. Albans	N/A			X	N/A		N/A		N/A	
Monsanto, Nitro	X		X		X		N/A		X	
S. Charleston, Sewage Treatment Plant	N/A			X	N/A		N/A		N/A	
Union Carbide(Rhone-Polenc) Institute (2 RCRA Permits)	X			X	X,X		X		X	
Union Carbide, (2 RCRA S. Charleston Permits)	X		X		X,X		X		X	
Union Carbide, Tech. Ctr. S. Charleston	X		N/A		X		N/A		N/A	

III. NEED TO EXAMINE TOXIC POLLUTANTS

Everyone, in all sections of the country, faces risks to their health and welfare in everyday life. These risks come in many forms and include risks from diet, smoking, driving, accidents, environmental pollution, etc. When the Environmental Protection Agency was formed in 1970, it initially set out to eliminate what Americans considered to be the greatest environmental risks to their health and welfare. These risks appeared in the form of "conventional" pollution such as smog and sewage, which were choking the nation's air and waterways. Traditionally, the largest and most visible national environmental programs have addressed mainly risks from elevated pollutant levels in the ambient environment[1] (primarily outdoor air and surface waters).

Control of particulates and other relatively well understood "criteria" air pollutants (sulfur dioxide, carbon monoxide, etc.) and water parameters such as total suspended solids, biological oxygen demand, and organic wastes, have enabled significant reductions of risks posed by these gross and largely visible forms of pollution.

With the growth of environmental sciences and the refinement of analytical techniques, however, environmental scientists and policymakers have discovered gaps in the programs that protect us against environmental risks. Toxic and hazardous chemicals[2] are present thoughout the country, in our homes (paints, cleaners, etc.), schools and buildings, outdoor environment, and work places. Unlike conventional pollutants, which are more easily detectable and relatively simple to clean up, these potentially toxic chemicals frequently pose health threats in even small quantities. They also sometimes change form or migrate, and can be very persistent in the environment.

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[1] The typical method of addressing these risks to the ambient environment from specific point sources of pollution has been essentially two fold: the first is to establish acceptable ambient concentrations for particular pollutants and to calculate the pollution controls needed to attain emissions/discharge levels that would meet the required ambient levels; the second method is to prescribe specific control technology (many times the "best available technology") regardless of acceptable ambient concentrations. Often the most stringent of the two approaches are used to control air and water sources. Where risk from pollution is not caused by a specific point source, other methods are used by EPA to control these risks. Examples of this would be EPA requirements for applicator training for use of pesticides, and requirements under the Toxic Substances Control Act to test new chemicals before production and distribution.

Because of the pervasiveness and complexity of pollution from toxic substances, and the limited resources of government and industry, policymakers must try to determine which risks from which toxics may be significant, and then reduce the worst and most controllable risks first. However, the science of quantifying public risk from "environmental" factors is very much in its infancy. Although the public and scientists now widely accept a few risks as being well-defined (e.g., the risk of lung cancer from smoking), the impact of a large majority of risks cannot be quantified with much certainty because of many unknowns (e.g., the extent of an individual's actual exposure and sensitivity to a given chemical).

Even defining which risks to include when considering "environmental" risks in a specific geographic area raises many legitimate questions. The long-term health effects of ambient air and water pollutants have historically been logical candidates for inclusion in risk studies. However, to be exhaustive in defining total environmental risks, a study would have to address accidental releases of chemicals from industrial plants, indoor air pollution, and non-cancer health risks. And for a more comprehensive outlook on these risks, the results of the study should be compared with the risks that workers and residents in the study area are exposed to from smoking, poor diet, hazardous occupations, individual accidents, and the like.

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[2] As used in this report, "toxic substances" are defined as those chemical substances which pose a substantial potential or actual hazard to human health or the environment when present even in relatively small quantities or low concentrations. The term "toxic substances" is often used indiscriminately to describe a wide variety of chemical substances, many of which may not pose a hazard but are perceived to pose a hazard. The toxicity of a given chemical compound is dependent, in addition to its chemical properties, on the dosage received. For example, ordinary table salt can become "toxic" in large enough dosages. Various references discussed in this report use the term "toxic substances" in a broad context; in such discussions, the term is used in the context explicity expressed in the reference. Whenever possible, however, the report uses specific teminology keyed to environmental laws to identify the substances present in the environment. For example, when discussing wastewater discharges or surface water or groundwater, "priority pollutants" refers to the group of 129 organic and inorganic chemical substances and heavy metals classified as toxic pollutants, as defined by Section 307(a) of the Clean Water Act. "Hazardous wastes", another term frequently misused, in this report refers to specific types of industrial, municipal, and commercial solid or liquid wastes that have been classified as hazardous wastes by EPA.

The national environmental programs administered by the Environmental Protection Agency in the past ten years have begun to also address non-conventional pollutant risks, but only to a limited extent (e.g., priority pollutants in drinking water, asbestos-in-schools, radon). Some other risks that are faced by the public are regulated by other government agencies such as the Occupational Safety and Health Administration (workplace risks), Food and Drug Administration (food intake risks), Department of Transportation (transportation related risks) Consumer Product Safety Commission (product risks) and Federal Emergency Management Agency (emergency actions).

Private industry also addresses these risks both because of the need to comply with government regulations, and for other reasons (e.g., protection of property from fire/explosions, potential liability from injuries/illness caused, protection of their work force and the public, and the need to be socially responsible "good neighbors").

Only in very recent years has EPA attempted to look beyond the respective programs that Congress has mandated to consider more broadly the wide variety of risks that we face. This so-called integrated approach to managing environmental risks has taken a number of forms. One approach has been to look at all major uses of a chemical (e.g., a specific solvent) before regulating it, so that the major risks of the chemical (and its potential substitutes) can be considered.

Another approach has been to identify environmental risks in a particular geographic area so that the relative risks from various sources can be compared and, if needed, control strategies for cost-effective reductions adopted. These geographic studies to date have largely focused on ambient air and water problems although other risks (e.g., indoor air) are beginning to be included in some studies. These studies typically have not included the consideration of risks from accidental (sometimes called catastrophic) releases of toxic pollutants nor have they included non-cancer health risks.

The Concepts of Risk Assessment and Risk Management

The concepts of risk assessment and risk management have been endorsed by EPA during the past several years, and have played a large role in developing the concept of integrated environmental management. Since 1983, EPA has increasingly used risk assessment to identify and measure toxic problems, and to compare them to one another to establish priorities ("Risk Assessment and Management: Framework for Decision Making," EPA, December, 1984).

In essence, risk assessment allows one to evaluate the health effects from exposures to toxic contaminants by estimating the risk from that exposure. Quantitative estimates allow us to compare risks from different chemicals, exposure pathways, and sources.

While risk assessment is rooted in scientific principles and knowledge of toxicology, epidemiology, and environmental exposure, it is inherently imprecise - necessary data are often limited, if available at all. Consequently, the results of a risk assessment depend heavily on assumptions about physical relationships and biochemical transformations, among other factors.

Despite its uncertainties, risk assessment provides a useful framework for comparing different environmental risks. As long as one under problems and establishing priorities for risk management. In a 1983 speech to the National Academy of Sciences, former EPA Administrator William Ruckelshaus distinguished between risk assessment and risk management:

> Scientists assess a risk to find out what the problems are. The process of deciding what to do about the problems is risk management. The second procedure involves a much broader array of disciplines, and is aimed toward a decision about control. Risk management assumes we have assessed the health risks of a suspect chemical. We must then factor in its benefits, the costs of the various methods available for its control, and the statutory framework for decision.

Risk Assessment and Risk Management Help Set Priorities

There are thousands of chemicals present in commerce today, and an unknown number of contaminants and unintended by-products. Scientific evidence suggests that some of these chemicals can cause cancer and other diseases. We are exposed to a complex mixture of these chemicals through air, water, and food. These chemicals are proper targets for Federal, state, or local regulation if they pose significant risks to health or the environment.

Regulatory priorities are often set in response to public pressures to address particular environmental issues of the day, rather than on the basis of systematic analyses of risks, costs and benefits. This would not be a problem if the issues of greatest public concern also posed the greatest risk. At times, however, public perception and actual risk diverge widely. We cannot know which problems are the most serious in terms of public health without some kind of rational, systematic evaluation of the issues. Risk assessments help identify and set priorities among those pollutants posing significant risks.

Some priorities will always be set by Congress or state legislatures, or through the press of emergencies. However, a consistent analytic approach is helpful in setting an agenda for effective risk management. Analyzing alternative strategies for reducing risk provides a useful basis for such an approach. Time and resources are best spent controlling those toxic chemicals to which people are most exposed and for which practical controls are possible. It is less productive to spend limited resources regulating chemicals, even highly toxic ones, to which no one will ever be exposed, or for which there is no capability to impose additional controls.

Given the level of public anxiety, extreme points of view often emerge when regulatory actions may involve substantial health or economic impacts in the face of scientific uncertainty. This situation may polarize debate and bring public policy to an impasse. Consistent use of risk assessment and risk management can assist by explaining the scientific basis for the risk estimates, including the confidence we have in such numbers; by placing the risk reduction expected from control measures in context with other risks and opportunities for their reduction; and by explaining the broader social values which affect risk management.

Moreover, communication of policies based on well articulated scientific principles is perhaps the most important element in creating a strong base of public understanding of environmental control programs. Enhancing efficiency through risk management means examining the regulatory options and selecting those that reduce risk the most for any given level of resources. The risk assessment analysis presented in this report is intended to provide a better understanding of some of the issues associated with toxic pollution in the Kanawha Valley and give decision makers a sense for the relative priority of these issues.

Risks in the Kanawha Valley

The types of potential environmental risks that exist in the Kanawha Valley are not considered to be significantly different from risks in other industrialized areas. They involve potential air and water pollution problems, indoor air pollution, short-term air or water releases, etc. Although the types of potential environmental risks are not considered different from other industrialized areas, the potential degree of risk has been questioned because of the high concentration of major chemical facilities, the geography and meteorology of the Valley, and the proximity of the residents.

Though the total number of industrial facilities in the Valley has declined somewhat in recent years, there remain nearly 200 of these facilities today, many of which process, handle, or dispose of a wide variety of chemical compounds. This geographic cluster of chemical/ industrial complexes is thought to represent one of the five most concentrated industrial areas in the United States. Given that many of these sources are located in close proximity to residential areas, Valley residents and officials have long been concerned with the possible health implications associated with long-term exposure to these chemicals. These environmental consequences are often more subtle in their effect and require more sophisticated approaches to analysis and control than the pollution problems of the past.

Previous environmental studies of the Kanawha Valley have pointed to a need for further evaluation of potential health effects from community exposures to these potentially toxic substances.

A limited 1981 pilot geographic study of Kanawha Valley performed under contract by EPA suggested that several environmental toxicants might be present at ambient levels which would warrant further investigation. In 1982, the West Virginia Department of Health conducted a brief comparison of cancer mortality rates and found that the incidence of malignant neoplasms (tumors) in a North Charleston area exceeded that which would be expected from national cancer mortality data. This brief analysis did not attempt to examine possible causitive factors, but did suggest the need for further study.

A 1984 report by EPA's National Enforcement Investigations Center identified several possible sources of toxic contaminants within Kanawha Valley, and concluded that while many sources, and sometimes specific chemicals and quantities released, were known, information was lacking on the relative threats these substances posed based on population exposure and inherent toxicity. The report also pointed to difficulties in assessing the impact of toxics in the absence of established standards or guidelines for many of the substances.

Though numerous studies of the Valley have been completed indicating the presence of a variety of chemical compounds, none has attempted to link ambient concentrations with human exposure and health effects.

EPA recognizes that the expense of quantifying <u>all</u> risks from all pollutants in the Kanawha Valley would be prohibitive, and that our ability to quantify these risks using even state-of-the-art techniques is very limited. However, a screening of the relative severity of some of these risks could be provided as an initial step in characterizing some environmental risks in the Valley.

IV. EPA's GOALS AND OBJECTIVES FOR KANAWHA VALLEY

The mission of the Environmental Protection Agency is to improve and protect the condition of the environment - both to reduce human health risks to acceptable levels and to protect and enhance the quality of natural ecosystems. Consistent with that mission, and as a result of the above Kanawha Valley background, EPA's Region III Office in Philadelphia outlined three broad goals for its involvement in the Kanawha Valley. These goals are:

1. To ensure compliance with existing Federal environmental laws, and to enforce requirements of those laws as necessary;

2. To determine the extent and sources of toxic pollutants in the Valley, and to estimate the risks to public health and the environment from exposure to these pollutants.

3. To reduce to acceptable levels the risks to public health and the environment from toxic pollutants, in the most effective way possible.

Many public and private organizations, in addition to EPA, are active in attempting to meet the above goals. And there are many activities underway and planned to assist in this endeavor. Many of these efforts involve analytic approaches and techniques which are on the forefront of environmental health protection.

The major purpose of this Report is to describe EPA's and West Virginia's initial efforts to date in meeting the above goals, primarily Goal No.2, and to provide insights into activities of other organizations who are also addressing in some fashion environmental health risks in the Kanawha Valley.

V. REPORT ORGANIZATION

Most of this Report (Chapters Two through Seven) is devoted to a detailed discussion of a limited screening study of selected toxic compounds recently completed by EPA and West Virginia environmental agencies. The Kanawha Valley Toxics Screening Study, though limited, is a first step in attempting to roughly assess and compare potential health risks from chemical exposures in the Kanawha Valley.

Since this Screening Study represents only a very limited perspective on a few selected toxics issues, we have attempted to describe in summary form some other environmental issues and activities of EPA and other organizations in Appendix A of the Report. Other Report Appendices provide information indirectly related or supporting the body of the main Report.

Chapter Two, "Kanawha Valley Toxics Screening Study - Framework", describes general information about the Study, including reasons for the Study, analytical study scope, specific objectives, study area, and management organization and communications.

Chapter Three, "General Methodology", describes the risk assessment screening methodology used to analyze all issues studied on a common basis. More importantly, it presents limitations and uncertainties in scope, exposure data, and toxicological data which have a profound bearing on the conclusions which can be drawn.

Chapters Four through Seven present detailed technical reports for air, drinking water, surface water, and hazardous waste. Each of these independent reports provides an analysis of several chemicals and possible human health risks associated with routine community exposures. A list of technical support documents is provided at the end of each of these Chapters. Copies of the support documents are available upon request from EPA's Region III Office.

Chapter Eight, "Putting Risk in Context", presents a brief discussion and some comparative tables which attempt to relate the Kanawha Valley risk estimates with similar estimates which have been calculated for other geographic areas or types of risks.

Chapter Two

Kanawha Valley Toxics Screening Study — Framework

CHAPTER TWO

KANAWHA VALLEY TOXICS SCREENING STUDY - FRAMEWORK

I. BACKGROUND

As noted in Chapter One, the Kanawha Valley has been the subject of environmental concern because of a combination of industrial facilities' location, nearby population, topography, and meteorologic conditions. Several previous studies of the Valley have indicated a need to more fully evaluate the health effects from chemicals released or present in the Valley. A 1984 study by EPA's National Enforcement Investigations Center stated:

> Air and water quality defined by traditional pollutant parameters (such as dissolved oxygen in water and suspended particulates in air) are generally better than applicable regulatory limits. However, some regulatory limits are occasionally not met indicating the need for further controls on traditional pollutants. Of more concern, however, is the presence of various potentially toxic chemical substances in the environment for which no regulatory limits have been established.

The Bhopal, India tradegy in December, 1984 further heightened public, industry, and government concern, both nationally and in the Kanawha Valley, over the issue of chemical plant safety and toxic air emissions. Subsequent Congressional inquiries into Bhopal and the U.S. chemical industry focused attention on Kanawha Valley due to its concentration of chemical facilities, and the presence of a production unit (MIC) similar to that connected with the Bhopal accident.

The increased public concern and Congressional attention over toxic air emissions also accelerated EPA's development of a National Air Toxics Strategy which was announced on June 4, 1985. The strategy included two major components:

1. A plan to address sudden, accidental air releases of chemicals which could cause immediate (acute), often life-threatening health effects; and

2. A plan to address routine releases of chemicals, normally over a prolonged period of time, where long-term health effects were of primary concern.

The first component included a variety of recommended actions designed to prevent, or respond, if necessary, to an accidental release of chemicals. Some of these actions are described in Appendix A. The second component included several recommended actions to help deal with the more subtle health risks which can occur from continual low level chemical exposures. The second component became the focus of the Kanawha Valley Toxics Screening Study described herein.

Concurrent with development of the second component of the Air Toxics Strategy, EPA had been evaluating options for how to address the above mentioned issues of long-standing concern in Kanawha Valley, primarily the long term threat of public exposure to air toxics. In addition, the West Virginia Air Pollution Control Commission had requested assistance from EPA in identifying and prioritizing volatile organic chemicals emitted in the Valley that could pose a significant health risk.

After weighing several options, the EPA decided that because of uncertainty as to the potential magnitude of the perceived problems, that, it would be prudent to first try to better define some air toxic issues in the Valley by performing a brief, limited scoping, or "screening level" exercise, in order to gain a rough understanding of the nature and potential seriousness of some toxics issues. This screening level assessment would use available health effects data, exposure models, and risk assessment techniques developed within EPA. It would provide an initial appraisal of whether some problems may exist, and what, if anything, should be done next. Explicit in this decision was an understanding that we would not be able to do a comprehensive study, or address, even on a screening level, all areas of environmental risk in the Kanawha Valley.

It was agreed that this screening study should rely primarily on existing data, and that the study would attempt to analyze multi-media exposure pathways, recognizing that the air pathway was of greatest interest, and should be the focus of study.

Based on these assumptions, State environmental officials were briefed on the study concept and agreed to participate. A workplan was developed, and the screening study began in the summer of 1985.

II. STUDY OBJECTIVES

Study objectives were shaped by previous Kanawha Valley studies' recommendations, State advice, available analytical methods, budget and resource constraints, and compatability with other ongoing environmental activities in the Kanawha Valley. The screening study is designed to begin to address one of EPA's three broad goals for Kanawha Valley to identify the extent and causes of toxic pollution in the Valley.

The overall objective for the screening study is to provide a general sense of the nature and relative priority of some ambient toxics pollution risks in the Valley, for the purpose of helping EPA and other agencies to make decisions about the need for further study and control of toxics in the Valley.

Specific objectives of the Toxics Study included the following:

1. Using available data bases, identify many of the toxic chemicals routinely released or present within various human exposure pathways within the Kanawha Valley.

2. Develop a sense of potential public health concerns which these toxic pollutants may pose in various pathways, based on health effects and exposure information.

3. For a select number of pollutants, provide an initial, conservative assessment of the potential cancer risks, and other potential non-cancer health risks where possible, from predicted or observed concentrations within exposure pathways.

4. Identify data and information gaps, and outline needs and options for future study direction to enable a more detailed investigation of health issues where warranted.

III. ANALYTICAL STUDY SCOPE

Multi-media screening study should not be confused with "comprehensive" problem analysis - the Kanawha Valley Toxics Screening Study is not a study of all potential toxic pollution in the Kanawha Valley.

Although the Kanawha Valley Study considers a variety of issues and exposure pathways, it does not examine risks from all pollutants. We decided to focus on "toxic" pollutants (i.e. those capable of causing adverse health effects at relatively low level of exposure) because such pollutants are relatively less studied and less controlled then more 'conventional' pollutants, such as ozone precursors in air and oxygen-depleting substances in water. Conventional pollutants may also affect health, although generally not at the low levels at which more toxic pollutants may be harmful.

We did not evaluate all toxic pollutants in the Kanawha Valley. We decided which pollutants to study largely on the basis of their known presence in the environment and available health effects data (see the discussion on pollutant selection in Chapters Four through Seven). The study did not attempt to identify the "most dangerous" or "highest risk" chemicals in the Valley. We attempted to identify and study those toxic pollutants which we believed may represent some substantive health risks in the Kanawha Valley. Nevertheless, it is likely that some of the pollutants we are not studying because of a lack of data concerning their presence or toxicity may also represent some potentially significant health risks.

It was recognized from the outset that all types of environmental risks could not be addressed. With limited funding and reliance on existing data, it was decided that the screening study could best meet objectives by focusing on potential long-term health risks from current ambient exposures. This necessarily excluded other types of risk, such as those from accidental releases of chemicals, from voluntary intakes (such as cigarette smoking and diet), and from exposures to indoor residential air, occupational chemicals, or toxic residues in food.

A major environmental concern among Valley residents is the risk posed by a sudden, accidental release of chemicals. There is extensive work continuing in this area by organizations like the Kanawha Valley Emergency Planning Council, the National Institute for Chemical Studies, and the chemical industry itself. We did not feel that we could measureably improve on these efforts or contribute to a better understanding of the issue within our limited study focus. Nevertheless, accident prevention and emergency response capabilities are recognized as very important issues which demand continuing attention and vigilance.

The Toxics Screening Study examined potential public exposure to several toxic pollutants through four routes by which contact could conceivably occur: through ambient (outdoor) air, consumption of public drinking water, fish consumption from the lower Kanawha River, and consumption of groundwater which could be contaminated by hazardous waste sites.

In the Air Analysis, twenty (20) known or suspected cancer-causing (carcinogenic) chemicals (14 from point sources) were evaluated out of more than 450 chemicals listed on an inventory of Kanawha Valley air emissions prepared by the West Virginia Air Pollution Control Commission. Potential community exposure to these 20 chemicals was estimated through mathematical modeling within four distinct geographic zones: Belle, Charleston/South Charleston, Institute, and Nitro.

For Drinking Water, nineteen (19) chemicals for which monitoring is required were evaluated from the study area's fourteen public water supply systems. Both potential cancer risks and non-cancer effects were evaluated. Private wells and non-community supplies were not included.

The Surface Water analysis focused on potential health effects, both cancer and non-cancer, from the possible consumption of contaminated fish taken from the Kanawha River near Nitro. In addition, concentrations of chemicals monitored within the River were compared with water quality criteria established for the protection of human health and aquatic life.

The Hazardous Waste analysis examined data from known hazardous waste sites in the Kanawha Valley in order to devise hypothetical hazardous waste settings (i.e., combinations of various facility types, hydrogeology, and waste constituents) which could present a risk to human health through public consumption of contaminated ground water. It did not include any site-specific health risk estimates.

IV. DESCRIPTION OF THE STUDY AREA

The Kanawha Valley is the narrow, winding Valley of the Kanawha River in west-central West Virginia surrounding the capitol city, Charleston. The Kanawha River traverses the western foothills of the Appalachian Mountains for 97 miles from its origin at Gauley Bridge at the confluence of the New and Gauley Rivers to its confluence with the Ohio River at Point Pleasant, West Virginia, northwest of Charleston.

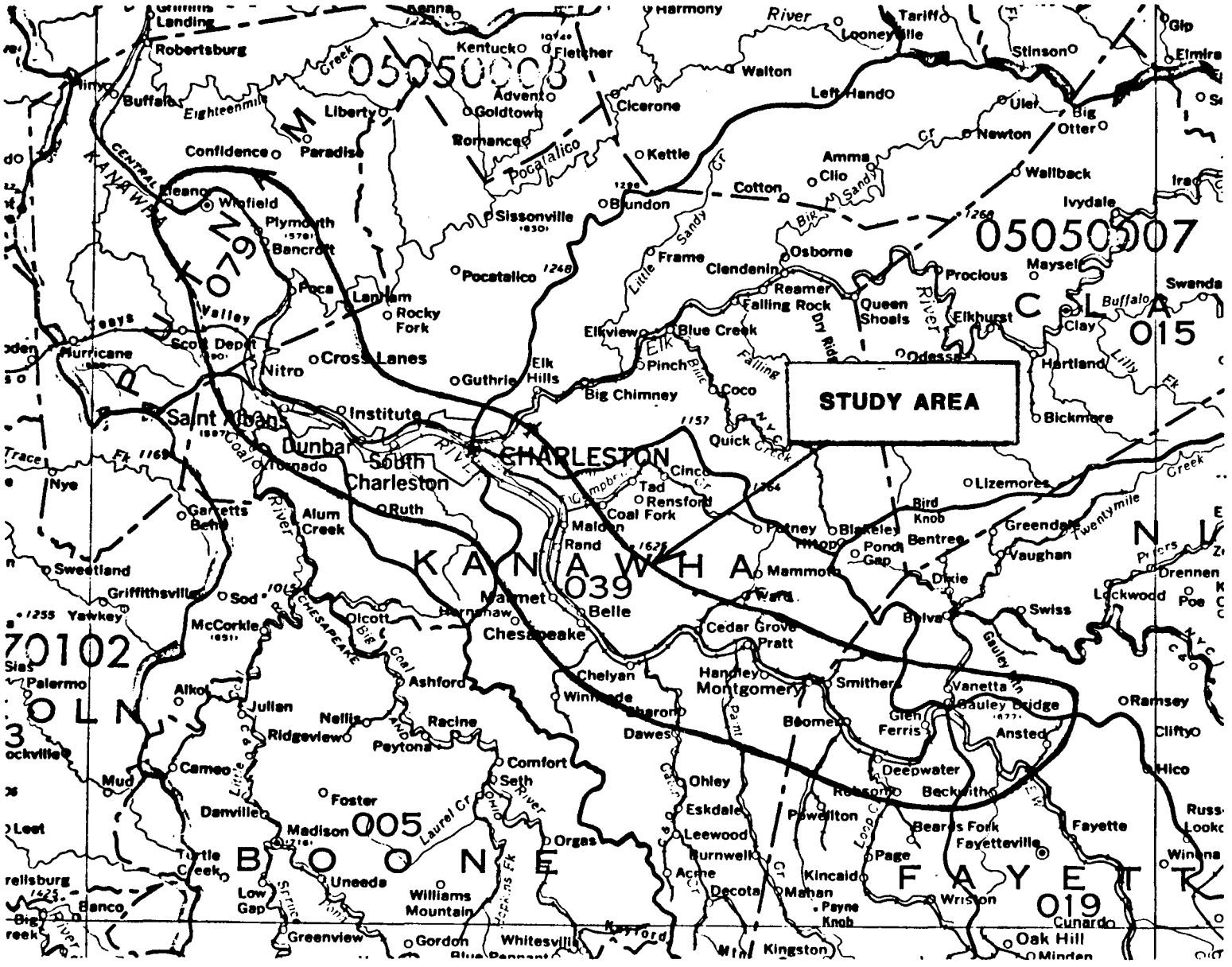
The general study area for the Toxics Screening Study encompassed a roughly 60-mile section of the Valley between Alloy and Windfield Dam (see Figure 2-1). The study area boundaries were selected to encompass major possible sources of toxics pollution and the population most likely impacted by this pollution.

Each media analysis - that is, those for air, drinking water, surface water, and hazardous waste - included a somewhat different geographic focus, although all were within the general Kanawha Valley study area. For example, the air analysis focused on four major industrial clusters at Belle, Charleston/South Charleston, Institute, and Nitro. Figures 2-2 and 2-3 indicate the geographic coverage of the first three media analyses. For the hazardous waste analysis, only hypothetical scenarios were constructed, and so no particular geographic area is shown.

The total population of the general study area is about 200,000 people. The four zones covered by the air analysis include approximately 100,000 people, while the drinking water anaylsis covered approximately 180,000 residents. Population densities are relatively low in the upper and lower thirds of the Valley with most of the population concentrated in Charleston and adjacent communities in the central portion of the Valley.

Because the Kanawha River traverses mountainous terrain, the Valley is relatively narrow, reaching a maximum width of only about 1 mile in the study area. The area of the valley floor in the study area is less than 50 sq. mi., much of which is developed for urban, industrial, or residential uses. The elevations of flanking mountains range from 300 to 1,300 feet above the valley floor. This particular topography sometimes act to trap air pollutants from industrial and municipal sources in the valley in close proximity to populated areas.

FIGURE 2-1 LOCATION MAP - KANAWHA VALLEY STUDY AREA


STUDY AREA
KANAWHA
039
CHARLESTON
South Charleston
Dunbar
Saint Albans
Institute
Nitro
Cross Lanes
Poca
Cedar Grove
Montgomery
Smithers
Belle
Chesapeake
Ashford
Racine
Peytona
Madison
Danville
Foster
Comfort
Seth
Orgas
Uneeda
Williams Mountain
Gordon
Whitesville
Kingston
Mammoth
Dixie
Bickmore
Hartland
Elkhurst
Clay
Swanda
Ivydale
Wallback
Newton
Stinson
Tariff
Looneyville
Walton
Harmony
Fletcher
Kentucky
Adventa
Goldtown
Romance
Cicerone
Kettle
Sissonville
Blundon
Pocatalico
Frame
Clendenin
Osborne
Reamer
Falling Rock
Queen Shoals
Procious
Maysel
Elkview
Blue Creek
Pinch
Big Chimney
Coco
Quick
Guthrie
Elk Hills
Lanham
Rocky Fork
Paradise
Liberty
Buffalo
Robertsburg
Winfield
Plymouth
Bancroft
Eleanor
Confidence
Eighteenmile
Scott Depot
Hurricane
Nye
Sweetland
Griffithsville
Yawkey
McCorkle
Sod
Alum Creek
Ruth
Garretts Bend
Tad
Rensford
Coal Fork
Maidon
Rand
Marmet
Hernshaw
Olcott
Alkol
Julian
Nellis
Ridgeview
Cameo
Mud
Turtle Creek
Low Gap
Greenview
Banco
Palermo
Chelyan
Winifrede
Sharon
Dawes
Handley
Ohley
Eskdale
Leewood
Burnwell
Acme
Decota
Mahan
Payne Knob
Kincaid
Powellton
Boomer
Glen Ferris
Deepwater
Beckwith
Page
Beards Fork
Fayetteville
Wriston
Cunard
Oak Hill
Minden
Fayette
Ansted
Gauley Bridge
Vanetta
Swiss
Bolivar
Lizemores
Bird Knob
Bentree
Pond Gap
Blakeley
Putney
Greendale
Vaughan
Drennen
Lockwood
Ramsey
Clifty
Hico
Russ
Winona
Cotton
Amma
Clio
Sandy
Big Otter
Left Hand
Cicerone
Pocatalico
Elk
Little Sandy
Falling
Dry Ridge
Big Sandy
Buffalo
Lilly Fk
Twenty Mile
Gauley Mtn
Pam
Loop Cr
Laurel Cr
Ohio River
Coal River
Little Coal
Chesapeake
Trace Fk
Spring Fk
Hughes Fk
Kanawha River
Elk River
Gauley River
Twenty Mile Creek
Lilly Fk
Big Coal River
Big Creek
Mill Cr
Kanawha
Fayette
Boone
Clay
Lincoln
Putnam
Raleigh
Nicholas
Greenbrier
Mingo
Wyoming
Logan
Mercer
Summers
Monroe
Wayne
Cabell
Mason
Jackson
Roane
Calhoun
Gilmer
Braxton
Clay
Nicholas
Webster
Pocahontas
Randolph
Upshur
Lewis
Gilmer
Wirt
Wood
Pleasants
Ritchie
Doddridge
Tyler
Wetzel
Marshall
Ohio
Brooke
Hancock
Marion
Harrison
Taylor
Barbour
Tucker
Preston
Monongalia
Marion
Harrison
Doddridge
Tyler
Pleasants
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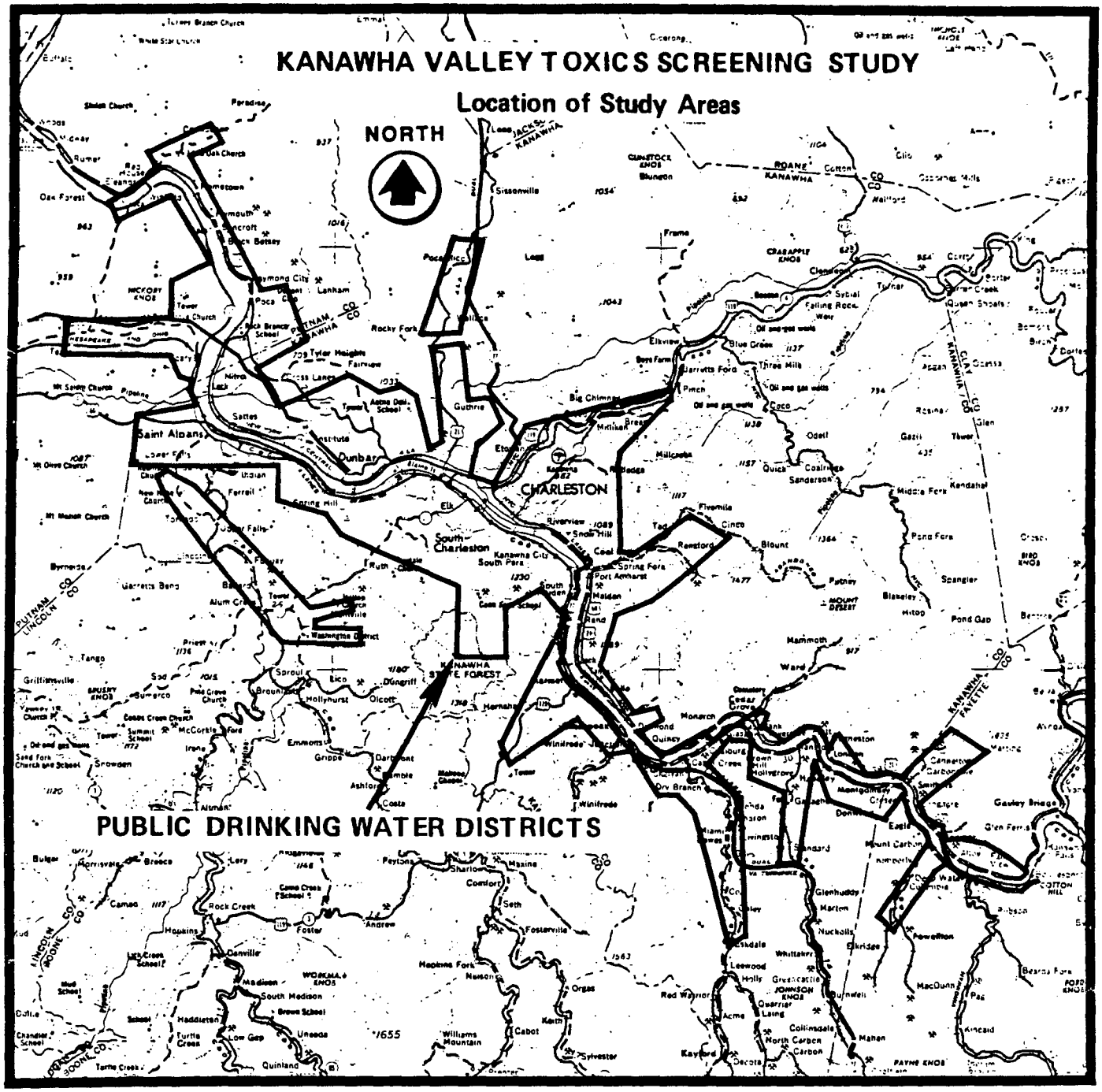


FIGURE 2-2. STUDY AREA - DRINKING WATER ANALYSIS

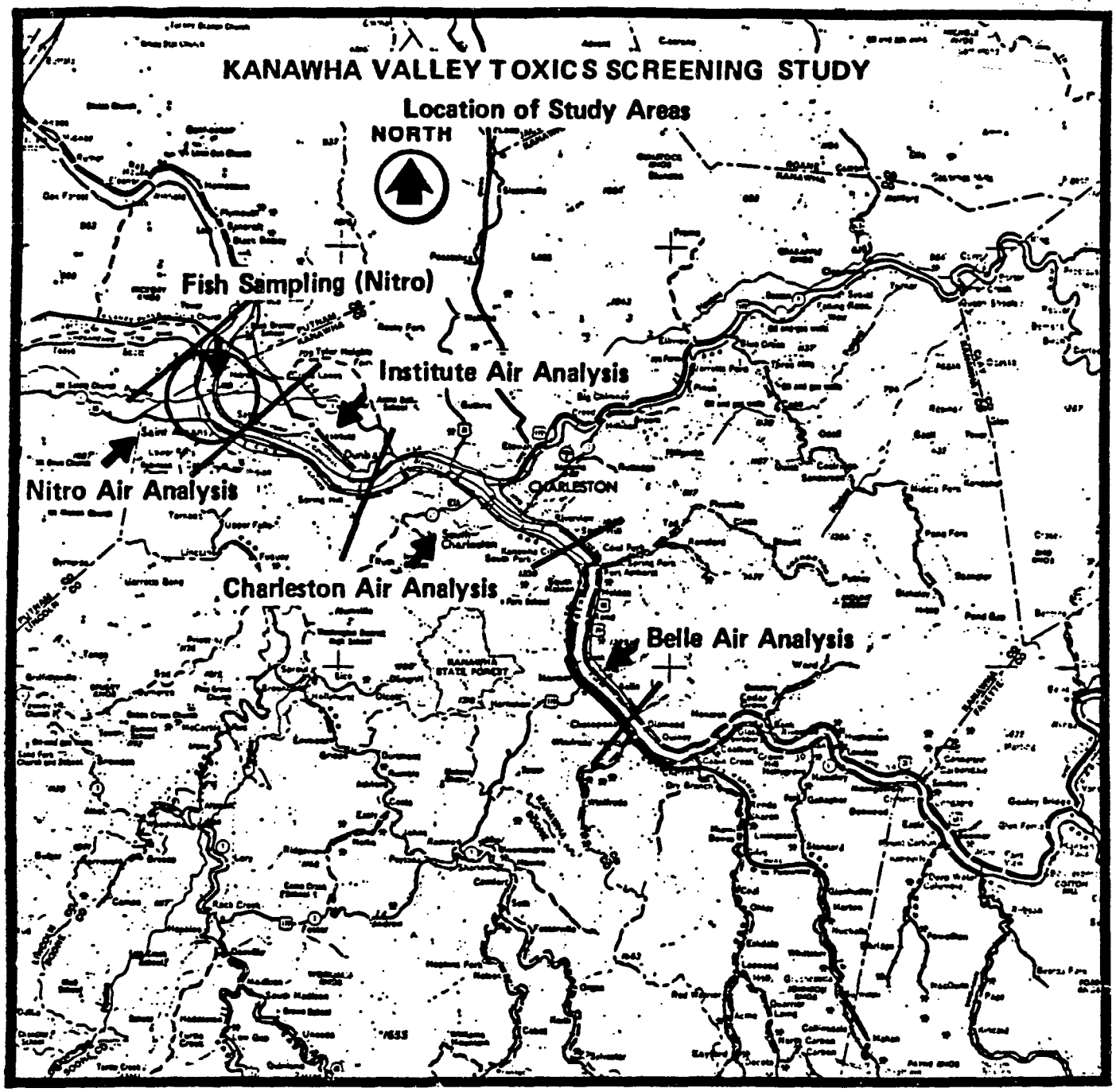


FIGURE 2-3 STUDY AREA - AIR AND SURFACE WATER ANALYSES

Streamflow in the Kanawha River can be highly variable ranging from flooding conditions in late winter and spring to low flows in late summer and fall. Extreme low flows range from about 600 cfs near Belle to about 1,500 cfs at Windfield Dam. This contrasts with average flows of about 11,000 cfs near Belle and 15,000 cfs at Windfield Dam. These flows are influenced by releases from upstream storage resevoirs, power generation, and the effects of the navigation dams in the study area. The Kanawha River is navigable throughout the study area with slack water provided by London, Marmet, and Windfield Dams. Major tributaries that join the Kanawha River within the study area are the Elk, Coal, and Pocatalico Rivers.

Public water supplies in the study area are obtained entirely from surface water. A major portion of the study area (about 180,000 population) is in the Kanawha Valley District of the West Virginia Water Company that obtains water from the Elk River. This system serves most of the Valley between Belle and Nitro. There are eight small water supply systems serving a population of about 20,000 in the upper valley upstream of Belle. These systems obtain water from the Kanawha River upstream of all major chemical manufacturing facilities. St. Albans Water Department serves about 20,000 persons in St. Albans with water obtained from the Coal River. St. Albans has an auxiliary intake near the mouth of the Coal River in backwater from the Kanawha River for use in times of extremely low streamflow in the Coal River.

There are no longer any public water supplies using Kanawha River water downstream from Belle. The Nitro area was formerly served by a West Virginia Water Company facility using Kanawha River water. Severe taste and odor problems necessitated a high level of water treatment including activated carbon columns. This plant has been closed and service is now provided from Elk River sources.

The Kanawha River is used by the various large industrial facilities for cooling water and some process water. Some industrial use of ground water may also be occuring. Groundwater is also used for private water supplies for scattered residences and small commercial facilities, mostly in areas away from the main Valley floor.

V. INSTITUTIONAL RELATIONSHIPS

At the outset of the study, it was clear that numerous offices within EPA and the State of West Virginia, as well as many other organzations, would have an interest in the issues surrounding the study. While a full public participation program was not established, EPA's Region III Office in Philadelphia took responsibility for communicating with a variety of groups about the nature and scope of the project as it progressed. These groups included local mayors

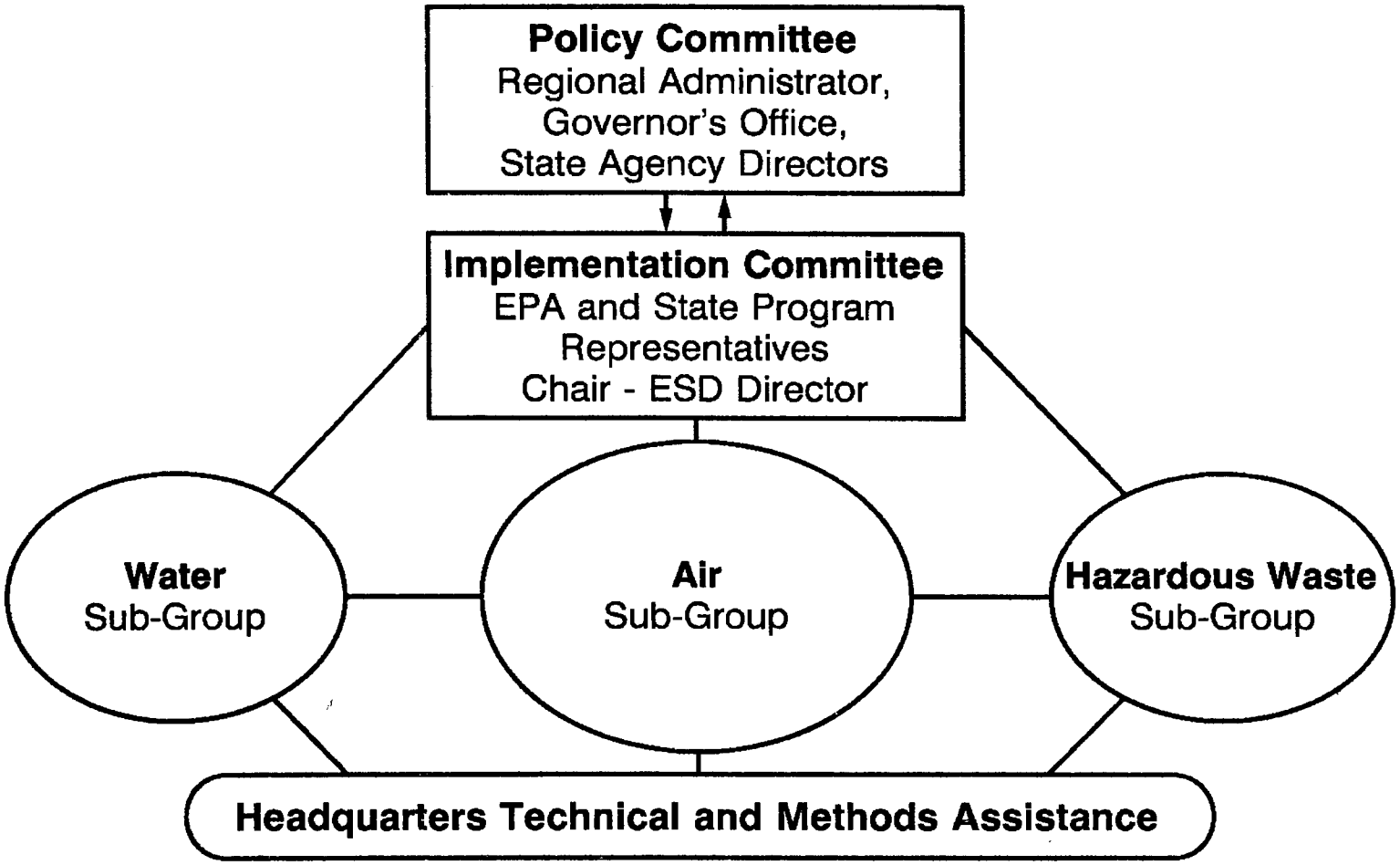
and officials, the National Institute for Chemical Studies, the Kanawha Valley Emergency Planning Council, environmental and industrial representatives. Each of these groups provided valuable perspectives, advice, and feedback that greatly helped to improve the study.

To manage the study itself, two intergovernmental committees were established. The first was a Policy Committee comprised of the EPA Region III Administrator, West Virginia Governor's Office, and the Directors of three West Virginia State Agencies: Air Pollution Control Commission (APCC), Department of Natural Resources (DNR) and Department of Health (DOH). The Policy Committee provided overall guidance for study direction, approved topics for study, and reviewed and evaluated major issues and findings of the study. They also retained ultimate authority for all policy issues, and made all decisions regarding allocation of their respective agencies' staff time and resources.

A second group, the Implementation Committee, was composed of EPA's Region III Environmental Services Division Director and West Virginia State Program representatives. The Implementation Committee was primarily responsible for defining the technical scope of the study, for guiding specific technical components, overseeing the analyses performed, arranging adequate staff time and data necessary as input to the analyses, assuring the quality of data used, and evaluating key issues and questions as they arose. The Implementation Committee kept the Policy Committee informed of project status and all areas of deliberation, making periodic recommendations to the Policy Committee on courses of action for unresolved issues.

Major technical support and analyses for the study was provided by EPA's Office of Policy Analysis (OPA) in Washington, D.C. In addition, specific workgroups for each media area of analysis (i.e., air, water, and hazardous waste) were established composed of senior staff from EPA and West Virginia programs. These groups monitored progress in each media area and provided information concerning ongoing program activities that could affect issues of the Toxics Study. Also, several offices within EPA contributed heavily to study direction and content. These included the Office of Air Quality Planning and Standards (OAQPS), in North Carolina, and the Office of Research and Development (ORD).

KANAWHA VALLEY INTEGRATED TOXICS STUDY MANAGEMENT ORGANIZATION



Chapter Three

General Methodology

CHAPTER THREE

GENERAL METHODOLOGY

The purpose of the Kanawha Valley Toxics Screening Study was to compare potential health risks attributable to different sources (e.g., chemical plants, runoff, hazardous waste sites), pollutants (organics, metals, etc.), and exposure pathways (air, drinking water, surface water, and ground water) in order to set research and management priorities. To make those comparisons, we needed to first estimate the risks quantitatively, where possible. This section discusses our methods of estimating and comparing risks. These methods are generally consistent with the approach to quantitative risk assessment for toxic air and drinking water contaminants that EPA has used in other recent studies.

I. LIMITATIONS AND UNCERTAINTIES

An understanding of the uncertainties and limitations that underlie the Kanawha Valley Toxics Screening Study is critical to a proper interpretation of its results. Limitations in the scope of what was studied, as previously discussed, as well as scientific and technical uncertainties in both the exposure and toxicological data (see below), caution against using the results as absolute health risk estimates. Nevertheless, decisionmakers must often act to protect against health threats from toxic chemicals without always having absolute scientific certainty. The Kanawha Valley analyses used the best information and techniques available today to estimate potential health risks from toxics so that decisionmakers will be as informed as much as possible.

Several general points concerning the limitations and uncertainties in our risk assessment deserve consideration. First, the risk estimates are based primarily on existing knowledge about pollutant releases, ambient conditions, and chemical toxicity; these data vary widely in quality and are almost always incomplete. Second, the exposure estimates incorporate a series of simplifying assumptions; although these assumptions are necessary, they remain open to question and may be controversial. Third, the potency estimates are necessarily based on current knowledge of the toxicological effects of various substances. Considerable controversy exists about the degree of hazard posed by different pollutants, and about whether some are hazardous at all. Finally, resource and time constraints and the breadth of our focus prevent us from analyzing individual issues in as much depth as might be possible. We have attempted to strike a balance between the desire for exhaustive and definitive analysis and the need for timely results at a reasonable cost.

To assure that our data, assumptions and analytic methods are as good as they can be, we have sought and will continue to solicit review from a range of knowledgeable sources: EPA regional and headquarters staff, Science Advisory Board reviews, state and local government officials, industry representatives, university faculty, and members of public interest groups. Our risk assessment procedures must be seen in light of their basic objective: to allow us to compare one risk to another, rather than to make definitive statements about the absolute risk posed by a particular substance, pollution source, or exposure pathway.

Limitations in Scope

It is important to realize that our analysis does not directly examine disease incidence in the local population and attempt to link it with environmental exposure. For many of the exposures and health effects with which we are concerned, such epidemiologic study is difficult because background incidence is high, exposures to particular individuals are difficult to quantify, and a variety of factors--genetics, occupational exposures, food, exposures from other places, and many others--might be the cause of an observed effect. Also, many effects may take years to manifest themselves, and would not be evident in the current population.

Because it is not an epidemiologic study, the risk assessment methodology is not intended to and does not answer questions such as what caused a statistically higher rate of cancer or birth defects in a particular neighborhood. Instead, it attempts to evaluate what health effects might result from current and future environmental exposures.

While different in approach and interpretation, risk assessments and epidemiologic work are complementary. Risk assessment can help to identify populations and geographic areas that appear to be at risk and that therefore might be appropriate subjects for an in-depth epidemiologic study. Epidemiologic analysis increases scientific understanding of the relationship between exposure and health effects, thereby strengthening the basis of risk assessment. Epidemiologic analysis may also, in some cases, be useful for confirming specific hypotheses suggested by risk assessment.

This analysis does not attempt to estimate the health risks from all chemicals or types of risks that individuals may be exposed to in their daily lives. We chose not to analyze exposure to and risks from conventional pollutants in air and water (such as ozone and oxides of nitrogen and sulfur in air, and oxygen-depleting substances and oil and grease in water) because we believed we could make a more significant contribution by concentrating on less well understood and less regulated toxic chemicals (largely organic chemicals thought to be potentially hazardous at low levels).

Finally, the study did not estimate risks from possible infrequent, accidental releases of toxic chemicals, such as a major release of a toxic gas. The probability and magnitude of such an accident is very difficult to estimate, and the likely risk from such an event is therefore difficult to quantify. (Appendix A does discuss some current activities related to prevention and response to such accidents). The omission of such events from this analysis does not imply that possible accidents are not an important environmental and public health concern.

The uncertainty in the risk assessment assumptions is great enough that results should be considered rough indicators of the probable magnitude of effects, not as precise, site-specific predictions of effects.

Limitations in Exposure Data

Beyond these intentional limitations in scope, the study's exposure and toxicological estimates are uncertain in a number of potentially important ways. For exposure assessment, one limitation of the analysis is that it did not exhaustively examine all sources and pollutants. While the study has tried to identify and assess risks from many significant sources and pollutants, it was unable to estimate exposure to some chemicals because of a lack of data. However, we recognize that chemicals not included in the study may also pose some health risk.

Even where exposure data were available, those data varied significantly in their quality. Thus, the resulting exposure estimates vary in their reliability. Those based on extensive field monitoring, such as for trihalomethanes and inorganic substances in drinking water, are the best types of exposure estimates we have. Those based on less extensive monitoring, such as for fish tissue analysis, are somewhat more uncertain.

Exposure estimates derived from modeling also vary in their reliability. Estimates of exposure to toxic organic chemicals in air, calculated using a dispersion model, are primarily dependent on the quality of the emissions estimates and other factors such as meteorological data. In general, the uncertainties introduced by the exposure data are usually much smaller than those associated with the dose-response information used to estimate human health risks. The analysis of future risks from groundwater contamination, which relies heavily on engineering assumptions and modeling of future events, is more uncertain. Where there are significant uncertainties, such as in the groundwater exposure analysis, we have attempted to make assumptions that are likely to err on the side of overestimating possible health impacts.

Limitations in Toxicology Data

Estimates of the potential health effects of particular chemicals are designed to be conservative (i.e., more likely to overestimate toxic health risks than to underestimate them) in several ways. When evaluating potential health hazards from a chemical, EPA scientists assume that health effects observed in laboratory animals are a reasonable indicator of potential effects in humans. In converting the animal data to predicted human responses, and in extrapolating from high doses to low doses, EPA uses models that yield a plausible upper-bound estimate of potency rather than a "best guess" estimate.

Many substances of potential concern have never been evaluated scientifically, or have not been evaluated in sufficient detail to allow estimation of specific effects on humans. For example, the element lead (present in air, water, and dust) is thought to pose a health risk to children at low ambient levels; currently, however, EPA has no accepted Agency method for quantifying individual risks or numbers of possible cases from non-carcinogens, although much work on this issue is in progress.

EPA is likely to be aware of the dangers from many of the most potent chemicals, since the evidence for their toxicity will typically be the most obvious; however, it is possible that some chemicals about which we currently know little may someday be demonstrated to be toxic. In addition, EPA currently has very little information on possible synergistic or antagonistic effects among pollutants.

Because of the many uncertainties and potential omissions, it is difficult to say whether the risk estimates presented here are over- or underestimates of actual health risks from pollutants studied. For those chemicals for which the study was able to make quantitative estimates of exposures and risks, it is more likely that risks are overestimated than underestimated. To the extent that toxic chemicals about which we currently know little have been left out, risks may be underestimated.

The value of the study methodology is that it allows an evaluation and comparison of the potential health risk from chemicals and pollution sources about which we have reasonably good information. Management of these risks, based on the best current information, can proceed, while research continues on the effects of chemicals about which little is currently known.

II. OVERVIEW OF METHODOLOGY

The Kanawha Valley study uses a risk assessment screening methodology to evaluate and compare, in a very limited fashion, the potential health risks from exposure to a limited set of pollutants. The purpose of this methodology is to permit a comparison of one risk with another and to provide a general sense of the risk a substance may present. This methodology does not make a definitive statement concerning the absolute risk posed by a particular pollutant, source or exposure pathway.

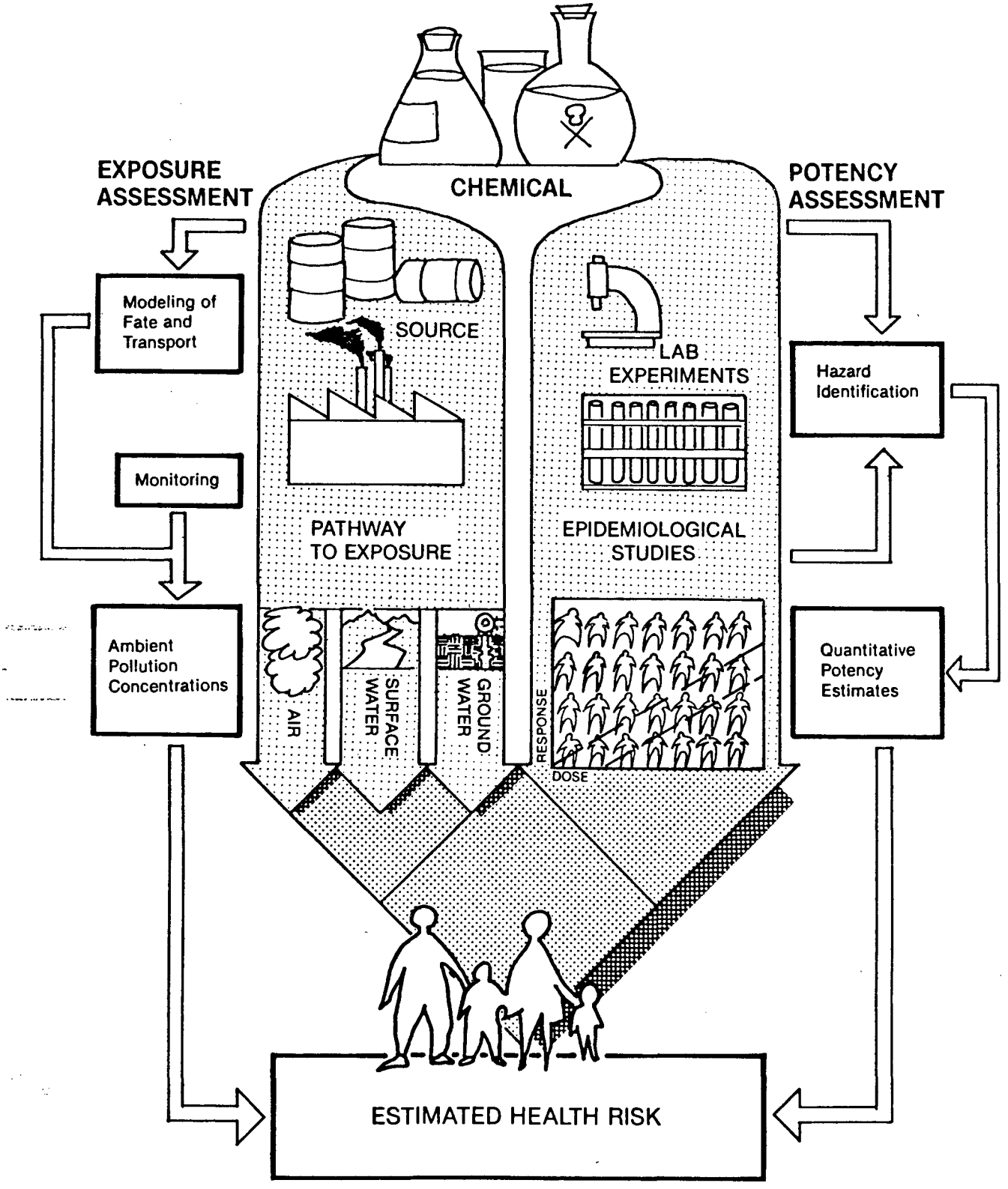
The following overview of the methodology for quantifying health risks applies, in theory, to both cancer and non-cancer health effects. However, the Kanawha Valley study contains quantitative risk estimates for cancer effects only. A brief discussion of our method for cancer risk assessment begins below. [See Appendix D for a more detailed discussion].

The risk assessment methodology combines estimates of toxicological potency, derived from laboratory and occupational studies, with estimates or measurements of local contamination (or exposure) levels to estimate risk to the local population. This approach involves a relatively high degree of uncertainty, and, in most cases, cannot easily be verified or disproven by observation. It is useful, however, because it is analytically straightforward and is the only method available for estimating the future health effects of current and future exposures.

The two key elements in estimating risk are a chemical's estimated toxicological potency and an individual's exposure to that chemical. This risk assessment methodology is illustrated in Figure 3-1.

The toxicological potency assessment involves (1) a qualitative 'hazard identification' to determine if evidence exists that a chemical causes an adverse health effect; and if so, (2) a quantitative estimate of the 'doseresponse relationship' or potency of a chemical, linking specific quantities of a chemical to particular health effect levels. Quantitative potency estimates typically relate given dose (equivalent to exposure) levels to the percent disease incidence expected in a population, or to a probability that an individual will be affected during the course of his or her lifetime. (See Figure 3-2 for an example of a dose-response curve.)

FIGURE 3-1 RISK ASSESSMENT METHODOLOGY



Exposure is estimated by measuring or estimating the ambient and making assumptions about the relationship between ambient conditions and actual human exposures. These standard assumptions, called exposure constants, account for the amount of air or water a typical person takes in in a day, and the weight of an average person.[1]

We assume that an average person weighs 70 kilograms (or roughly 154 pounds), breathes 20 cubic meters of air each day (700 cubic feet), and drinks 2 liters (2.1 quarts) of water each day.

Finally, exposure and potency estimates are combined to estimate individual and population risks.

We define risk to an individual as the increased probability that an individual exposed to one or more chemicals will experience a particular adverse health effect during the course of his or her lifetime. (An average lifetime is assumed to be 70 years). Risk to the population is the expected increased incidence (number of cases), above the background rate, of an adverse health effect in an exposed population. For cancer risks, we present both types of quantitative risk estimates in the Kanawha Valley study.

If we make the simplifying assumption that the dose-response curve is linear, potency (risk per unit of exposure) is constant at any level of exposure in which we are interested (unless the chemical has a threshold, below which we believe there is no effect). Under these assumptions, lifetime risk to the exposed individual is simply the product of exposure and potency:

R	=	E	x	P
(individual risk)		(exposure)		(potency)

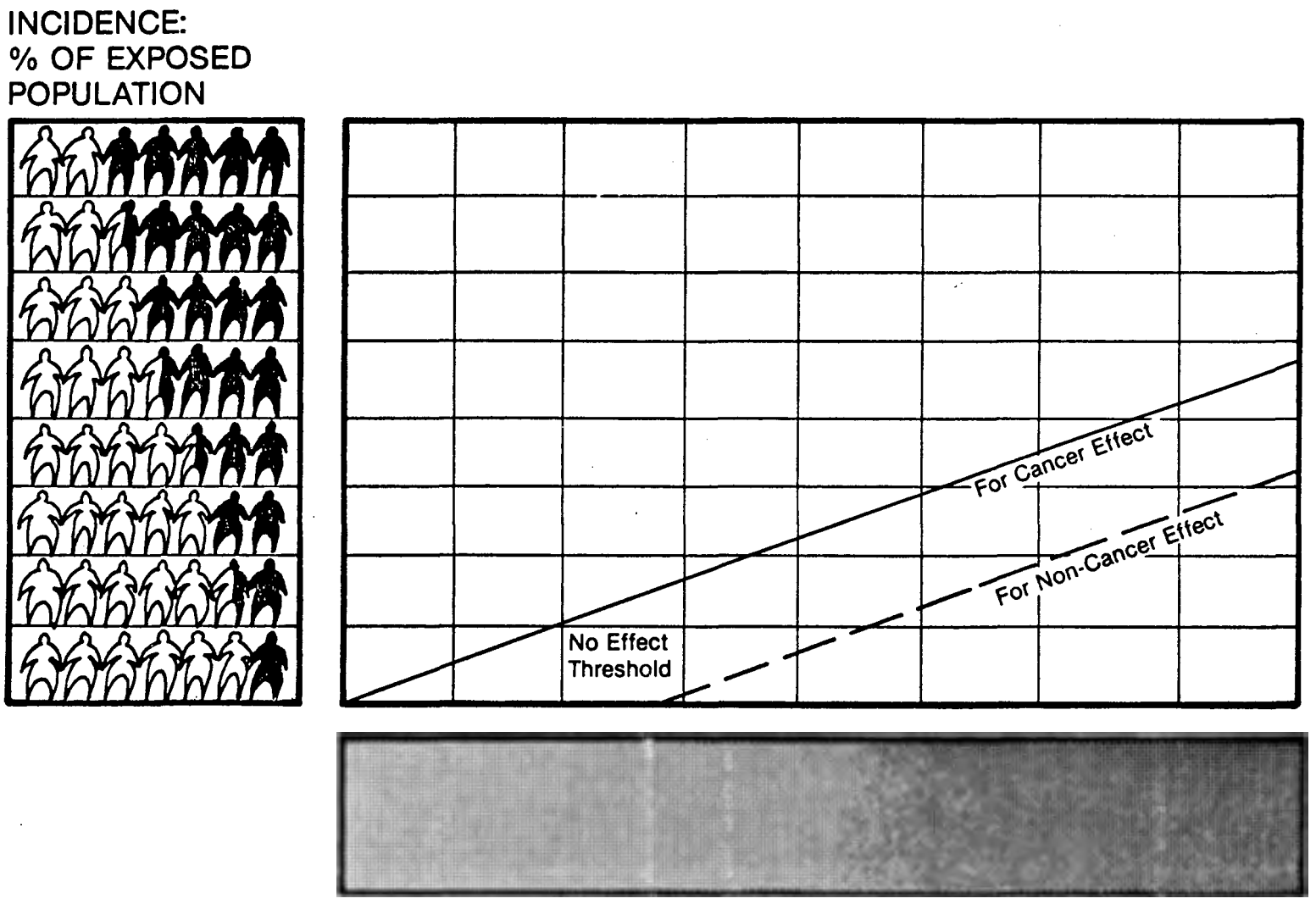
As discussed above, exposure is the product of ambient levels of the pollutant in the medium of concern (air, drinking water, fish tissue) and exposure constants (i.e., the standard assumptions about body weight, intake and absorption):

E	=	Y	x	Z
(exposure)		(ambient concentrations in medium of concern)		(exposure constants)

---

1 A further calculation of the actual dose likely to be absorbed into the body involves an estimate of the body's propensity to absorb a chemical to which it is exposed. This conversion of exposure to dose is estimated for each chemical and exposure route, and is contained within the potency estimate.

FIGURE 3-2 DOSE-RESPONSE CURVE



Therefore, individual lifetime risk can be calculated by multiplying the ambient concentration (Y) and exposure constants (Z) by potency (P):

R	=	Y	x	Z	x	P
(individual risk)		(ambient concentration in medium of concern)		(exposure constants)		(potency)

For example, we might wish to calculate the lifetime risk of cancer for an individual exposed to ten micrograms per cubic meter of benzene in the air. Filling in equation (3) presented above, the ambient concentration (Y) is ten micrograms per cubic meter (which is the same as 0.01 milligrams per cubic meter or 0.01 mg/m^3). Our exposure constants (Z), as discussed above, assume a typical 70 kilogram person breathing 20 cubic meters of air each day ($20m^3/day$).

Our potency value (P) is the cancer potency score for benzene inhalation developed by EPA's Carcinogen Assessment Group (CAG) of 0.029 $(mg/kg/day)^{-1}$. This value indicates that an individual taking in one milligram of benzene per kilogram of body weight per day (this would be 70 milligrams per day for a typical 70 kilogram person) for a lifetime has an estimated chance of about three in a hundred of contracting cancer. (If the units are confusing, think of the score as indicating cancer risk for a given dose of benzene.)

Using this information to fill in the equation presented above, we can estimate risk as follows [THIS IS ONLY AN EXAMPLE]:

$$R = 0.01 \frac{mg}{m^3} \times \left[20 \frac{m^3}{day} \times \frac{1}{70\ kg} \right] \times \frac{0.029}{(mg/kg\ day)}$$

(individual risk) (ambient concentration) (exposure constants) (potency)

Working through the arithmetic, individual risk (R) in this case is 8×10^{-5}, or eight chances in 100,000 of contracting cancer over a lifetime.

To calculate the aggregate expected increased incidence of disease, we multiply the average individual risk by the number of individuals exposed:

I	=	R	x	P
(aggregate expected increased incidence of disease)		(individual risk)		(exposed population)

Following this example, suppose that we estimate that 100,000 people are exposed to 10 micrograms per cubic meter (or 0.01 mg/m^3) of benzene. We would estimate incidence as follows [THIS IS ONLY AN EXAMPLE]:

$$I = 8 \times 10^{-5} \times 100{,}000 \text{ people}$$

(aggregate incidence of cancer) (individual risk) (exposed population)

The product of this equation is 8 cases of cancer over a 70 year period (the assumed average lifetime in the individual risk calculations). By convention, incidence (unlike individual risk) is reported in annual cases, so we divide our product, 8 cases, by 70 to arrive at our estimate of 0.1 cases per year, or about one case every ten years.

Despite the application of similar risk assessment techniques, each media analysis differed in terms of geographic coverage, chemicals studied, whether ambient concentrations were measured or estimated, the potential health risks considered (cancer or non-cancer), and in the means of expressing the risks studied (e.g., risk assuming uniform ingestion of drinking water versus risk per pound of fish consumed). Table 3-1 illustrates some of these differences.

TABLE 3-1

DIFFERENCES IN RISK ASSESSMENT APPROACHES[1] BY MEDIA ANALYSIS

		RISK ASSESSMENT APROACH			
		Exposure Data		Health Effects Considered	
MEDIA STUDIED	PRIMARY ROUTE OF EXPOSURE ANALYZED	Measured	Modeled	Cancer	Non-Cancer
Air Analysis	Inhalation of Ambient Air		X	X	
Drinking Water Analysis	Ingestion of Public Drinking Water	X		X	X
Surface Water Analysis	Consumption of Contaminated Fish	X		X	X
Hazardous Waste Analysis	Ingestion of Contaminated Ground Water		X	X	X

[1]In addition to these differences, the area of geographic coverage and types of chemicals studied for each media analysis were different, as was the means of expressing the risks studied - see text.

The application of risk assessment techniques in a complex geographic area such as the Kanawha Valley is difficult because there are many scientific and technical limitations. For example, there are uncertainties in estimating the amount of pollutants released from a given source, in estimating the extent of exposure of the general public to a particular pollutant(s), and in extrapolating health effects evidence derived from animal studies to estimate corresponding human responses. In general, the uncertainties introduced by the exposure data are usually much smaller than those associated with the dose response information used to estimate human health risks.

Because of these uncertainties, EPA prefers to err on the side of caution in making risk assessments. Therefore, following standard EPA protocol, we use conservative estimates of risks that probably overestimate the actual risk from any one chemical. While the risks outlined in the study should not be interpreted as a definitive prediction of actual cancer risk to the general public in the Valley, these estimates are useful in comparing the relative risks among various chemicals.

Chapter Four

Air Quality Analysis

KANAWHA VALLEY AIR QUALITY REPORT

I. INTRODUCTION AND PURPOSE

The Kanawha Valley Toxics Screening Study has four stated objectives that have provided overall direction to this technical report. The four objectives are:

(1) Using available data bases, identify many of the chemicals routinely released or present within various exposure pathways;

(2) Develop a sense of the potential public health concerns these toxic pollutants may pose in various pathways, based on health effects and exposure information;

(3) For a select number of chemicals, provide an initial conservative assessment of potential cancer risk and potential noncancer health risk for predicted or observed concentrations within exposure pathways; and

(4) Identify data and information gaps and outline needs and options for future study directions to enable a more detailed investigation of health issues where warranted.

These four objectives have guided the development of the methodology for this air quality report. We reviewed available data bases of air emissions within the study area to identify many of the chemicals routinely emitted by point and county-wide area sources within the Kanawha Valley. Although we cannot at this time assess the potential health concerns all toxic pollutants within the inventories may pose in the ambient air, we do provide a conservative assessment of potential cancer risk from predicted exposures from a small set of pollutants. Because of limitations in our exposure assessment and health effects information, we do not assess potential noncancer health effects of these pollutants.

The chapter is divided into seven sections. Section I is the introduction. Section II explains our methodology for selecting a limited set of pollutants emitted by facilities within a set of defined air sheds. Section III describes the general approach to the analysis including discussions of technical issues involved in determining predicted ambient concentrations of pollutants within the Kanawha Valley. Section IV presents the exposure assessment methodology and exposure results. We examined two types of exposures: ambient exposures of pollutants within pre-selected neighborhoods surrounding the major facilities in this study area, and ambient exposures to the average individual within the study area. Section V discusses the risk assessment methodology and limitations, and Section VI presents the results of this risk assessment methodology. The risk assessment results

are based on modeled concentrations developed within the exposure assessment section, standard EPA exposure assumptions, and carcinogenic unit risk factors. The results, by pollutants and sources, are presented in terms of lifetime incremental risk to the individual and incidence, which is the predicted number of cancer cases per year during a lifetime exposure from these pollutants. Finally, Section VII compares the results across valley zones to identify the most important pollutants, facilities, and release types, in terms of risk and incidence, within the Kanawha Valley.

II. STUDY AREA DEFINITION AND POLLUTANT AND SOURCE SELECTION

Study Area Focus

The Kanawha River Valley is a 60-mile river stretch in a region of rolling terrain with numerous ridges and valleys. The major industrial areas that are the focus of this study are not distributed throughout this region, but rather are concentrated along the valley floor of the Kanawha River between Belle and Nitro, an area of roughly 45 km^2. The study area for the air analysis was therefore defined to include only this key central area. Within this area we further divided the valley into four valley zones: Belle, Charleston/South Charleston, Institute, and Nitro. The selection of this study area was based on four justifications:

(1) The population of the Kanawha region is heavily clustered within this 45 km^2 area around the industrial facilities.

(2) Based on the 1984 WVAPCC emission inventory, an inventory of organic pollutants released into the ambient air by industry, there are no major releases from Kanawha, Putnam, or Fayette Counties of the potential pollutants of concern from industrial facilities outside of the 45 km^2 area between Belle and Nitro.

(3) Current air dispersion models are unable to effectively assess the entire 60-mile river stretch with its complex meteorology and terrain.

(4) Current air dispersion models <u>can</u> be applied within the valley, provided the analysis is limited to evaluating concentrations at receptors on the valley floor within limited distances of the facilities (see Section III below).

The valley varies from a minimum of about 1 km in width at Belle to as wide as 2 km in the South Charleston region; valley walls vary in height from approximately 200 m to 300 m. Wind flow is complicated not only by the changing orientation of the valley as the flow is channeled

within it, but also by daily wind flow patterns. By dividing the valley into four separate zones (see Figure 1) it was possible to estimate ambient concentrations in neighborhoods close to the industrial facilities contained in each zone and to determine exposure to the average individual within each zone by applying existing air pollution dispersion models without modification.

Pollutant Selection

The West Virginia Air Pollution Control Commission (WVAPCC) has compiled an extensive emissions inventory for the Kanawha Valley. According to that inventory, at least 570 pollutants are emitted by the principal 17 point source facilities in the area. Since one objective of this study is to estimate potential carcinogenic risk from a limited set of pollutants and facilities, the modeling analysis focused primarily on pollutants that have received a unit risk factor for carcinogenicity from EPA's Carcinogen Assessment Group (CAG). In addition to these pollutants, the study modeled ambient concentrations of two other chemicals, formaldehyde and propylene oxide, which the West Virginia Department of Health and EPA have identified as potentially carcinogenic and which are emitted by major industrial facilities in the region.

Table 1 lists the 20 pollutants selected for evaluation in this study. The list includes 12 CAG-scored organic compounds known to be emitted by industrial sources in the valley, 11 CAG-scored compounds that are commonly emitted on an area-wide basis from dispersed nonindustrial sources (county-wide area source emissions), and the 2 compounds identified by the West Virginia Department of Health.

Sources

Point Sources: The selected study area of the Kanawha Valley includes distinct clusters of industrial sources. These clusters include some of the largest chemical manufacturing facilities in the United States. Table 2 lists seven industrial facilities that were part of the 1984 WVAPCC inventory and that emit one or more of the studied pollutants addressed in this study; it also lists five area source categories. These industrial sources emit 14 of the 20 study pollutants; area sources emit the additional 6 pollutants. Table 3 presents the available emissions data from each industrial point source and area source category evaluated in this study.[1]

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1 The EPA report "Overview of Environmental Pollution in the Kanawha Valley, West Virginia" (Vincent 1984) provides a summary of the products produced by the major industrial facilities in the area. Products produced in the Valley by these industries include insecticides, pharmaceuticals, plastics, and fibers.

FIGURE 1

KANAWHA VALLEY TOXICS SCREENING STUDY
KANAWHA VALLEY AIR QUALITY REPORT

THE LOCATION OF THE FOUR CHEMICAL FACILITIES IDENTIFIED FOR THE KANAWHA VALLEY STUDY

CIRCLES REPRESENT LOCATIONS OF FACILITY WITHIN VALLEY ZONES

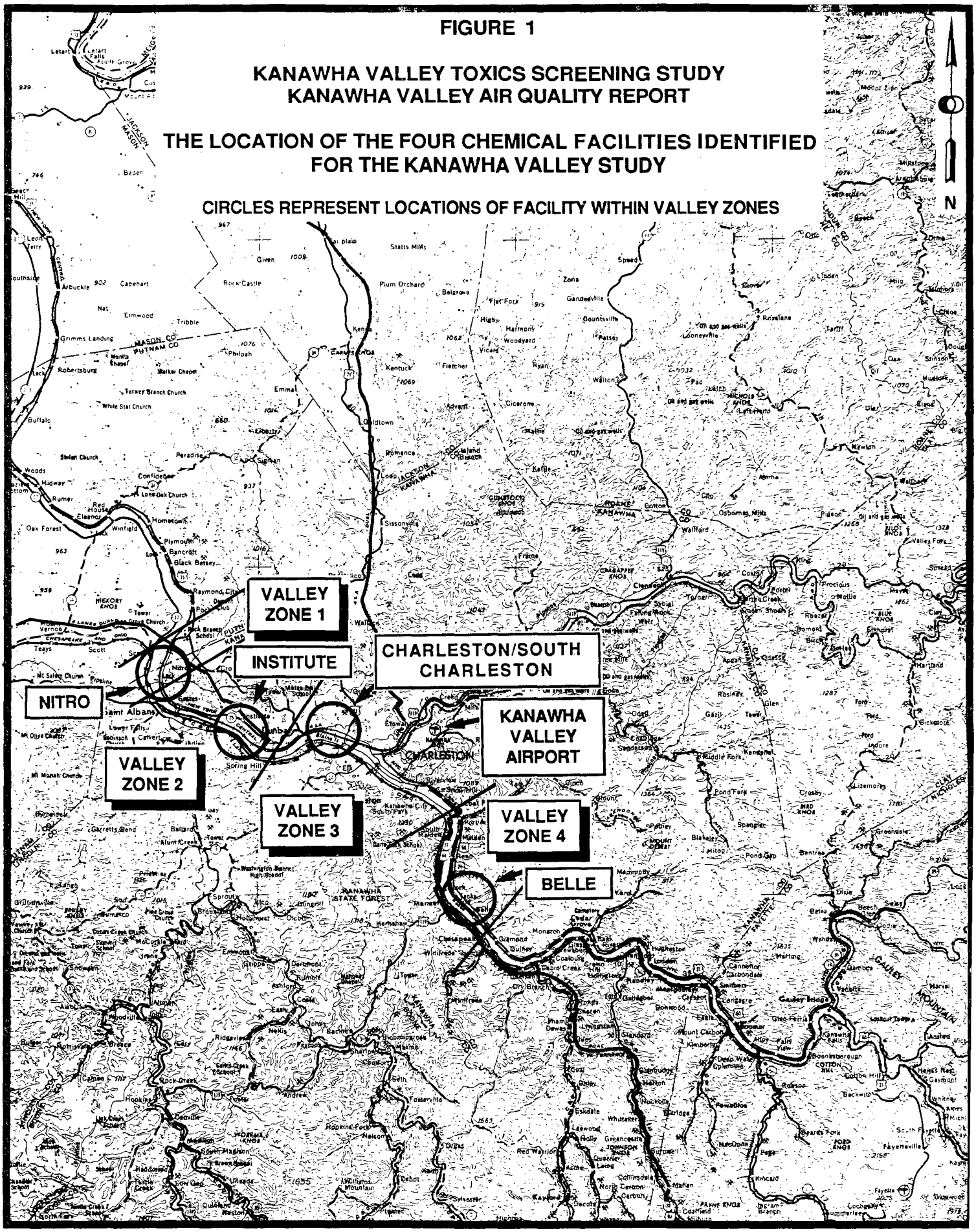


TABLE 1

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

Pollutants Evaluated in This Study

Pollutant	Emitted by Industrial Sources	Emitted by County-wide Sources
Benzene	X	X
Perchloroethylene		X
Methylene chloride	X	X
Formaldehyde	X	X
Trichloroethylene	X	X
1,3-Butadiene	X	X
Ethylene chloride	X	X
Ethylene bromide		X
Arsenic		X
Benzo(a)pyrene		X
Cadmium		X
Beryllium		X
Carbon tetrachloride	X	
Chloroform	X	
Ethylene oxide	X	
Acrylonitrile	X	
Allyl chloride	X	
Vinylidene chloride	X	
Vinyl chloride	X	
Propylene oxide	X	

TABLE 2

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

SOURCES OF THE 20 POLLUTANTS EVALUATED IN THIS STUDY

Industrial Facilities	Location
DuPont	Belle
Union Carbide	S. Charleston/Charleston
Union Carbide	Tech. Center
Rhone Poulenc	Institute
Occidental	Belle
Monsanto	Nitro
Artel	Nitro
County-wide Area Source Categories	
Solvent Use	-
Road Vehicles	-
Heating	-
Gasoline Marketing	-
Waste Oil Burning	-

TABLE 3

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

SUMMARY OF EMISSIONS DATA FOR SELECTED POLLUTANTS
(METRIC TONS/YEAR)
ZONE: BELLE

	Point Sources[2]			Area Sources[3]				
Pollutant	Occidental	Dupont-Belle	Solvent Uses	Road Vehicles	Heating	Gas Marketing	Waste Oil Burning	Totals
Methylene chloride	83.0	2.9	7.0	0.0	0.0	0.0	0.0	92.9
Chloroform	70.8	1.3	0.0	0.0	0.0	0.0	0.0	72.1
Carbon tetrachloride	20.1	2.8	0.0	0.0	0.0	0.0	0.0	22.9
Formaldehyde	0.0	8.1	0.0	4.0	2.5	0.0	0.0	14.6
Benzene	0.0	0.0	0.0	13.9	6.1×10^{-2}	0.4	4.0×10^{-5}	14.4
Perchloroethylene	0.0	0.0	12.6	0.0	0.0	0.0	1.0×10^{-4}	12.6
Trichloroethylene	0.0	0.0	1.7	0.0	0.0	0.0	1.0×10^{-4}	1.7
1,3-Butadiene	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.1
Ethylene chloride	0.0	0.0	0.0	0.0	0.0	3.7×10^{-2}	0.0	3.7×10^{-2}
Ethylene bromide	0.0	0.0	0.0	1.4×10^{-2}	0.0	3.7×10^{-3}	0.0	1.8×10^{-2}
Arsenic	0.0	0.0	0.0	0.0	1.2×10^{-2}	0.0	8.0×10^{-4}	1.3×10^{-2}
Benzo(a) pyrene	0.0	0.0	0.0	2.3×10^{-3}	3.1×10^{-3}	0.0	2×10^{-5}	5.4×10^{-3}
Cadmium	0.0	0.0	0.0	8.0×10^{-4}	3.4×10^{-3}	0.0	2×10^{-4}	4.4×10^{-3}
Beryllium	0.0	0.0	0.0	0.0	4.0×10^{-4}	0.0	8.0×10^{-5}	4.8×10^{-4}

[2] Source: West Virginia Air Pollution Control Commission 1984 Emission Inventory, 1987.

[3] Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987.

TABLE 3 (CONTINUED)

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

SUMMARY OF EMISSIONS DATA FOR SELECTED POLLUTANTS
(METRIC TONS/YEAR)
ZONE: CHARLESTON/SOUTH CHARLESTON

	Point Sources[2]		Area Sources[3]					
Pollutant	Carbide-S. Char.	Carbide-Tech. Ctr.	Solvent Uses	Road Vehicles	Heating	Gas Marketing	Waste Oil Burning	Total
Propylene oxide	44.6	5.0×10^{-4}	0.0	0.0	0.0	0.0	0.0	44.6
Benzene	0.0	0.0	0.0	40.3	0.2	1.3	1.0×10^{-4}	41.8
Perchloroethylene	0.0	0.0	36.5	0.0	0.0	0.0	3.0×10^{-4}	36.5
Ethylene oxide	35.7	0.4	0.0	0.0	0.0	0.0	0.0	36.1
Methylene chloride	5.0×10^{-4}	4.2	20.4	0.0	0.0	0.0	0.0	24.6
Formaldehyde	0.0	0.0	0.0	11.7	7.3	0.0	0.0	19.0
Acrylontrile	17.3	0.2	0.0	0.0	0.0	0.0	0.0	17.5
Trichloroethylene	0.0	0.0	4.9	0.0	0.0	0.0	4.0×10^{-4}	4.9
Vinylidene chloride	0.1	0.3	0.0	0.0	0.0	0.0	0.0	0.4
1,3-Butadiene	0.0	0.0	0.0	0.3	0.0	0.0	0.0	0.3
Ethylene chloride	0.0	0.0	0.0	0.0	0.0	0.1	0.0	0.1
Ethylene bromide	0.0	0.0	0.0	4.0×10^{-2}	0.0	1.1×10^{-2}	0.0	5.1×10^{-2}
Arsenic	0.0	0.0	0.0	0.0	3.5×10^{-2}	0.0	2.2×10^{-3}	3.7×10^{-2}
Benzo(a) Pyrene	0.0	0.0	0.0	6.6×10^{-3}	9.0×10^{-3}	0.0	4.0×10^{-5}	1.6×10^{-2}
Cadmium	0.0	0.0	0.0	2.3×10^{-3}	9.9×10^{-3}	0.0	5.0×10^{-4}	1.3×10^{-2}
Beryllium	0.0	0.0	0.0	0.0	1.0×10^{-3}	0.0	2.0×10^{-4}	1.2×10^{-3}

[2] Source: West Virginia Air Pollution Control Commission 1984 Emission Inventory, 1987.

[3] Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987.

TABLE 3 (CONTINUED)

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

SUMMARY OF EMISSIONS DATA FOR SELECTED POLLUTANTS
(METRIC TONS/YEAR)
ZONE: INSTITUTE

	Point Sources[2]	Area Sources[3]					
Pollutant	Rhone Poulenc	Solvent Uses	Road Vehicles	Heating	Gas Marketing	Waste Oil Burning	Total
Ethylene oxide	97.5	0.0	0.0	0.0	0.0	0.0	97.5
Propylene oxide	71.2	0.0	0.0	0.0	0.0	0.0	71.2
Chloroform	62.1	0.0	0.0	0.0	0.0	0.0	62.1
Acrylonitrile	28.1	0.0	0.0	0.0	0.0	0.0	28.1
1,3-Butadiene	25.2	0.0	0.1	0.0	0.0	0.0	25.3
Methylene chloride	15.3	9.1	0.0	0.0	0.0	0.0	24.4
Benzene	0.3	0.0	18.0	8.0×10^{-2}	0.6	5.0×10^{-5}	19.0
Perchloroethylene	0.0	16.3	0.0	0.0	0.0	2.0×10^{-4}	16.3
Formaldehyde	0.1	0.0	5.2	3.3	0.0	0.0	8.6
Trichloroethylene	0.0	2.2	0.0	0.0	0.0	2.0×10^{-4}	2.2
Ethylene chloride	0.0	0.0	0.0	0.0	4.8×10^{-2}	0.0	4.8×10^{-2}
Ethylene bromide	0.0	0.0	1.8×10^{-2}	0.0	4.8×10^{-3}	0.0	2.3×10^{-2}
Arsenic	0.0	0.0	0.0	1.6×10^{-2}	0.0	10.0×10^{-4}	1.7×10^{-2}
Benzo(a) Pyrene	0.0	0.0	3.0×10^{-3}	4.0×10^{-3}	0.0	2.0×10^{-5}	7.0×10^{-3}
Cadmium	0.0	0.0	1.0×10^{-3}	4.4×10^{-3}	0.0	2.0×10^{-4}	5.6×10^{-3}
Beryllium	0.0	0.0	0.0	5.0×10^{-4}	0.0	10.0×10^{-5}	6.0×10^{-4}

[2] Source: West Virginia Air Pollution Control Commission 1984 Emission Inventory, 1987.

[3] Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987.

TABLE 3 (CONTINUED)

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

SUMMARY OF EMISSIONS DATA FOR SELECTED POLLUTANTS
(METRIC TONS/YEAR)
ZONE: NITRO

	Point Sources[2]		Area Sources[3]					
Pollutant	Monsanto-Polymer	Artel	Solvent Uses	Road Vehicles	Heating	Gas Marketing	Waste Oil Burning	Total
Trichloroethylene	22.5	0.0	0.8	0.0	0.0	0.0	7.0×10^{-5}	23.3
Formaldehyde	4.7	0.6	0.0	2.0	1.3	0.0	0.0	8.6
Benzene	0.0	9.0×10^{-4}	0.0	7.1	3.1×10^{-2}	0.2	2.0×10^{-5}	7.3
Perchloroethylene	0.0	0.0	6.4	0.0	0.0	0.0	6.0×10^{-5}	6.4
Methylene chloride	0.0	5.0×10^{-4}	3.6	0.0	0.0	0.0	0.0	3.6
Allyl chloride	0.0	2.0	0.0	0.0	0.0	0.0	0.0	2.0
Ethylene chloride	0.0	5.2×10^{-2}	0.0	0.0	0.0	1.9×10^{-2}	0.0	7.1×10^{-2}
1,3-Butadiene	0.0	0.0	0.0	5.4×10^{-2}	0.0	0.0	0.0	5.4×10^{-2}
Ethylene bromide	0.0	0.0	0.0	6.9×10^{-3}	0.0	1.9×10^{-3}	0.0	8.8×10^{-3}
Arsenic	0.0	0.0	0.0	0.0	6.1×10^{-3}	0.0	4.0×10^{-4}	6.5×10^{-3}
Acrylonitrile	0.0	5.9×10^{-3}	0.0	0.0	0.0	0.0	0.0	5.9×10^{-3}
Benzo(a) Pyrene	0.0	0.0	0.0	1.2×10^{-3}	1.6×10^{-3}	0.0	8.0×10^{-6}	2.8×10^{-3}
Cadmium	0.0	0.0	0.0	4.0×10^{-4}	1.7×10^{-3}	0.0	9.0×10^{-5}	2.2×10^{-3}
Carbon tetrachloride	0.0	9.0×10^{-4}	0.0	0.0	0.0	0.0	0.0	9.0×10^{-4}
Chloroform	0.0	9.0×10^{-4}	0.0	0.0	0.0	0.0	0.0	9.0×10^{-4}
Vinyl chloride	0.0	5.0×10^{-4}	0.0	0.0	0.0	0.0	0.0	5.0×10^{-4}
Beryllium	0.0	0.0	0.0	0.0	2.0×10^{-4}	0.0	4.0×10^{-5}	2.4×10^{-4}

2 Source: West Virginia Air Pollution Control Commission 1984 Emission Inventory, 1987.

3 Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987.

Although the study excluded no known sources of the 20 pollutants addressed within the study area, it did not model any sources outside of the study area, such as the Amos power plant in Poca and the Kanawha River station power plant at Cedar Grove. Although these facilities are a potential source of metals, they were excluded because emissions data were unavailable and because the expected incremental impacts in the residential areas closest to it within the study area were assumed to be minimal.

Area Sources: Estimates were made of the emissions for pollutants from five county-wide area source categories: (1) solvent use, (2) road vehicles, (3) heating, (4) gasoline marketing, and (5) waste oil burning. All available emissions data for pollutants addressed in this study were compiled for these categories and then apportioned into a modeling grid.

Emissions were first estimated at the county level, based on factors such as vehicle miles traveled, population, and fuel consumption. Emissions factors were used to estimate the mass of specific pollutants emitted as a function of the above factors. The county-wide estimates were then distributed among the 2.5 km grid squares that were within the study area. Refer to Technical Appendix D for a detailed description of county-wide area sources.

III. GENERAL METHODOLOGY

The two principal elements of this study's methodology are exposure assessment and risk assessment. This section dicusses general study design issues pertaining to these key elements. Details on implementation of the general design for exposure assessment are presented in Section IV, and risk assessment is discussed in Section V.

The general methodology is summarized as follows:

- The approach for exposure assessment is presented by identifying five major complications specific to this study area, and then indicating how these technical problems were overcome.
- The risk assessment subsection then summarizes how the exposure data were used to estimate risk on two scales: neighborhood scale and average individual exposures for each zone.

Exposure Assessment

The exposure assessment used in this study had two objectives:

(1) To characterize concentrations of toxic air pollutants in neighborhoods adjacent to the major industrial complexes, and

(2) To provide conservative assessments of average concentrations of toxic pollutants in each of the four valley zones studied.

To achieve these objectives, the study relied principally on the use of dispersion modeling analysis. A limited amount of monitored data was gathered as part of the effort, but these data were used only as a reference for evaluating the general magnitude of the results developed through dispersion modeling. The estimated pollutant concentrations used in the risk analysis were not modified in any way to reflect the results of the monitoring program.

The study relied to the extent possible on standard methods and procedures. A detailed modeling protocol for conducting Gaussian diffusion modeling within the valley was written and submitted to EPA for review and approval by various offices within EPA; it is reproduced in Technical Appendix C.

The study's general exposure assessment methodology can best be discussed in relation to five basic problems specific to evaluating air quality within the industrialized sectors of the Kanawha River Valley:

(1) Estimating releases from the hundreds or thousands of potential sources within each industrial complex;

(2) Accounting for the variability of emissions from the facilities under study;

(3) Developing appropriate modeling approaches to deal with the highly complex terrain of the valley;

(4) Obtaining acceptable meteorological data representative of conditions in and around each major facility; and

(5) Correctly identifying population locations, especially for those nearest the major facilities.

Each of the five points is discussed below.

Multiple Release Points: Many of the chemical complexes under study release air pollutants from literally hundreds of separate stacks, valves, pipes, flanges, vents, and other sources; release characteristics from these different types of sources vary significantly. In addition,

each of the industrial complexes covers many acres. Simplified modeling techniques that characterize releases as originating at only one, or even several, different release points would inevitably yield inaccurate and misleading estimates of pollutant concentrations in the local area, especially those concentrations for the residential neighborhoods closest to the plants. It was therefore imperative to estimate release points in as much detail as possible, both in terms of where pollutants are released and in terms of the characteristics of each release point.

To respond to this problem, the study relied on the WVAPCC 1984 Toxic Air Pollutant Inventory, which separately describes up to thousands of individual release points within each chemical complex in the valley in terms of source characteristics and substances released. Staff of the WVAPCC aggregated these release points within each of the seven facilities, typically identifying 25 to 50 separate source groups within each facility to be used as input to the computer modeling effort. These aggregated sources represented three basic types of releases: stacks, building vents, and industrial area sources (such as tank farms). This level of detail increases the resolution of estimated pollutant concentrations in the neighborhoods adjacent to the chemical plants.

Source Variability: Another major difficulty in conducting air exposure assessments in this area is accounting for the variability of source releases over time. Chemical manufacturing processes often operate in batches. Releases may therefore vary substantially from hour to hour and from day to day. Since air flow in the valley also tends to change significantly over each 24-hour period (with nighttime conditions often being considerably more stable and less likely to vigorously disperse air pollution than more turbulent daytime conditions), characterizing source release variations is critical to understanding exposures in neighborhoods surrounding the facilities and to the average exposed individual.

To respond to this problem, the study relied on the data gathered by the WVAPCC, which considers hours of operation of the various processes in the plants. This permitted the development of considerably more representative day/night air dispersion model treatments.

Topographic Effects: In their current state of development, air dispersion models have only limited success in characterizing dispersion over terrain defined as "complex," that is, where the release height of a pollutant is at a lower elevation than a receptor point (such as a nearby hill).

To accurately estimate concentrations of pollutants at all locations through the valley, one approach would be to develop a site-specific air dispersion model to predict concentrations under the unique conditions

imposed by the local terrain. This would have been too expensive and time consuming for use in this study, and the accuracy of modeling transport in complex terrain on the order of 40 km to 50 km would still have involved considerable uncertainty. Instead, the study relied on available Gaussian dispersion models, but limited the area of concern to the valley floor, where their predictions of dispersion over flat terrain are likely to be reasonably reliable. This approach is especially applicable to the estimation of concentration in neighborhoods in proximity to industrial facilities, provided that available meteorological data accurately represent each of the valley zones (see below). The approach will satisfy the exposure assessment's first objective, assessing concentrations in neighborhoods nearest the chemical plants.

To meet the study's second objective over the full area of each zone, ISCLT, a gaussian model, was used for industrial sources, and box model estimates were used for county-wide area sources to determine exposures to the average individual. This provided one estimate of exposures to the average individual. Since this approach does not allow for interzone transport of pollutants, another approach was developed to provide likely conservative estimates of exposure and to allow interzone transport of pollutants. For this second approach, we applied the box model to all pollutant sources. Although the results of this box model analysis of total emissions in the study area are not as detailed as those of Gaussian dispersion models for each valley zone, the box model can estimate conservative upper bounds of annual average concentrations to which the population as a whole may be exposed. The box model is a simple treatment, but true concentrations of pollutants in the area are unlikely to be any higher than the concentrations it predicts when conservatively applied. Section IV, "Sensitivity Analysis," presents these conservative estimates of average concentrations to which the population within the study area could be exposed.

Meteorological Conditions: Air flow within the Kanawha River Valley is strongly channeled by the valley walls. The models used for evaluating pollutant concentrations require estimates of wind direction, wind speed, and atmospheric stability on an annual basis. The study therefore established meteorological monitoring stations within each of the four valley zones and operated the network continuously for a period of six months in cooperation with the WVAPCC. In addition to recording wind speed and direction, the system also continuously recorded turbulent intensity at the Belle and Institute zones, thereby providing detailed data to help document valley dispersion characteristics, including dispersion under nighttime conditions, when stable air can lead to higher concentrations of pollution within the areas downvalley of the chemical facilities.

Population Distribution: Since one objective of the study was to characterize exposures in the neighborhoods adjacent to the industrial facilities, it was necessary to obtain accurate data on the location of residences near the chemical plants. Residences are usually located through review of USGS and other maps of a local area; for this effort, however, field studies were conducted to precisely identify the location of residences in key areas so that modeling results could be as accurate as possible. In many cases, the closest residences are within 200 m of the property lines of the major facilities.

Figures 2 through 5 show the neighborhood-level grid used by the modeling analysis to evaluate concentrations in critical subareas of the four study zones. Each of these receptors is located in a residential area. The modeling also produced estimates of general concentrations within a larger receptor grid established for each valley zone; Figure 6 shows the 2.5 km grid and associated population data used to produce estimates of population-weighted average concentrations.

Risk Assessments

In our exposure analysis, we modeled the selected pollutants and sources within each zone for the two exposures, neighborhood exposures and average individual exposures for the zone. For our carcinogenic health effects analysis, we apply our risk assessment methodology to the concentrations predicted from the exposure assessment.

For these two types of predicted concentrations we developed several measures of risk. First, we estimated lifetime incremental individual risk for these two exposures. To develop these risks, we assumed standard EPA exposure assumptions and applied unit risk factors developed by the Carcinogen Assessment Group (CAG) within EPA. From these estimates of lifetime incremental risk, we further developed cancer incidence estimates for the population. Incidence, for the purpose of this report, is the estimated upper-bound number of cancer cases expected over a 70-year exposure period. Incidence is calculated by multiplying individual risk by the population number, and can be annualized by dividing by 70, the assumed average life span, to obtain cancer cases per year.

IV. EXPOSURE ASSESSMENTS

This section describes the exposure assessment methodology in detail. Topics covered are the following:

- Simplifying assumptions;
- Model selection;

FIGURE 2

KANAWHA VALLEY TOXICS SCREENING STUDY
KANAWHA VALLEY AIR QUALITY REPORT

APPROXIMATE LOCATIONS OF NEIGHBORHOOD RECEPTORS, MAJOR FACILITIES, AND MONITORING SITES FOR THE BELLE COMPLEX

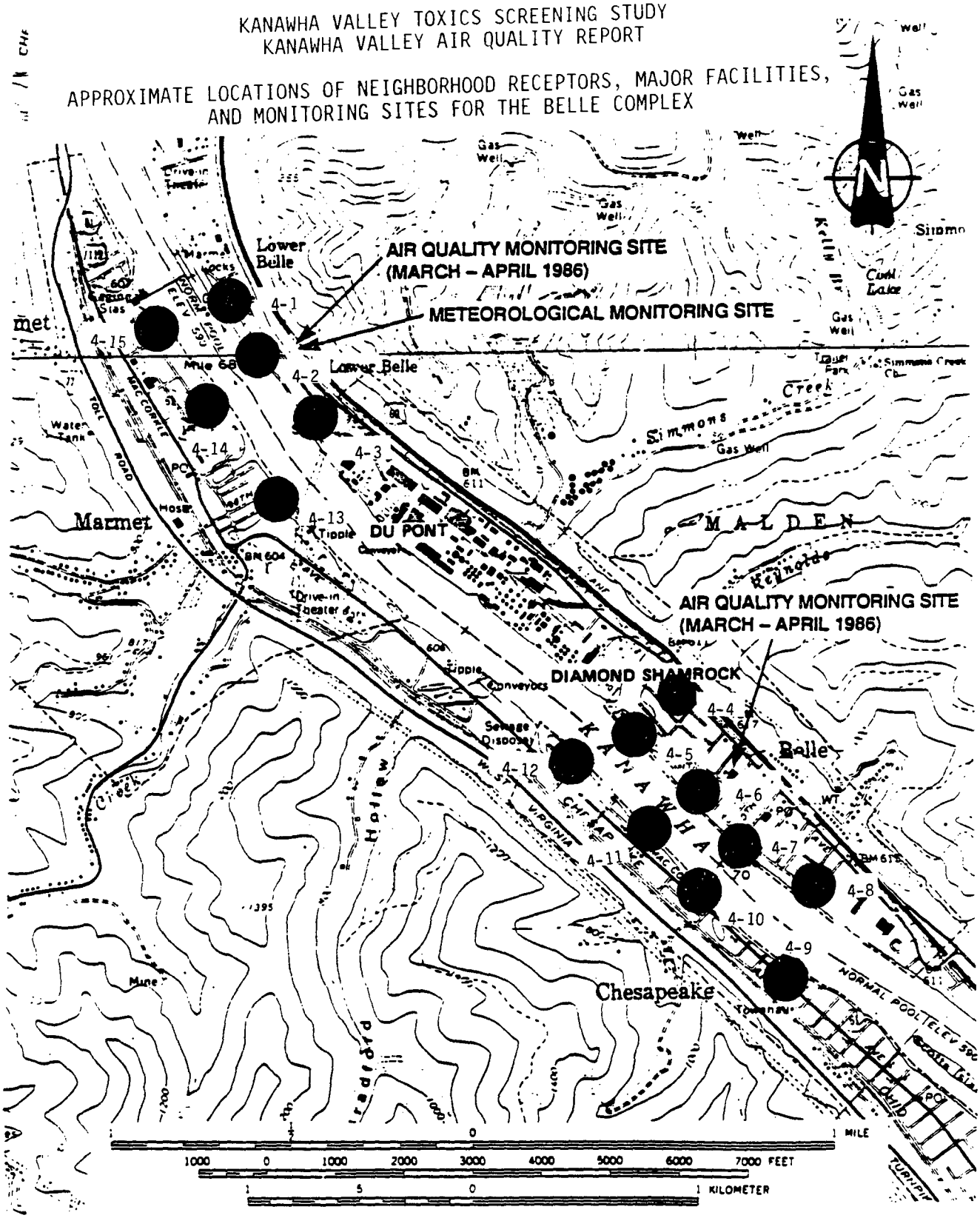


FIGURE 3

KANAWHA VALLEY TOXICS SCREENING STUDY
KANAWHA VALLEY AIR QUALITY REPORT

APPROXIMATE LOCATIONS OF NEIGHBORHOOD RECEPTORS, MAJOR FACILITIES, AND MONITORING SITES FOR THE SOUTH CHARLESTON COMPLEX

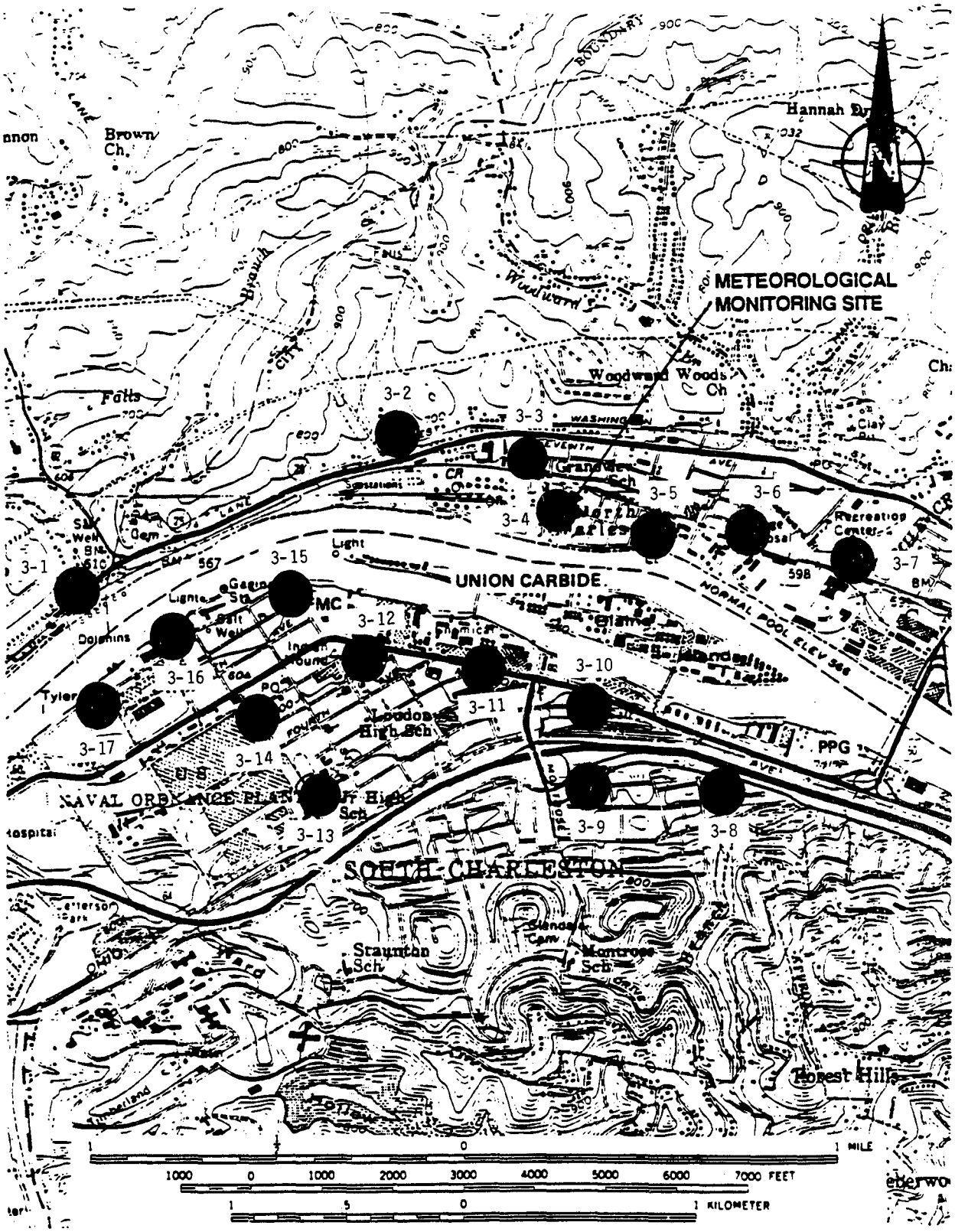


FIGURE 4

KANAWHA VALLEY TOXICS SCREENING STUDY
KANAWHA VALLEY AIR QUALITY REPORT

APPROXIMATE LOCATIONS OF NEIGHBORHOOD RECEPTORS, MAJOR FACILITIES, AND MONITORING SITES FOR THE INSTITUTE COMPLEX

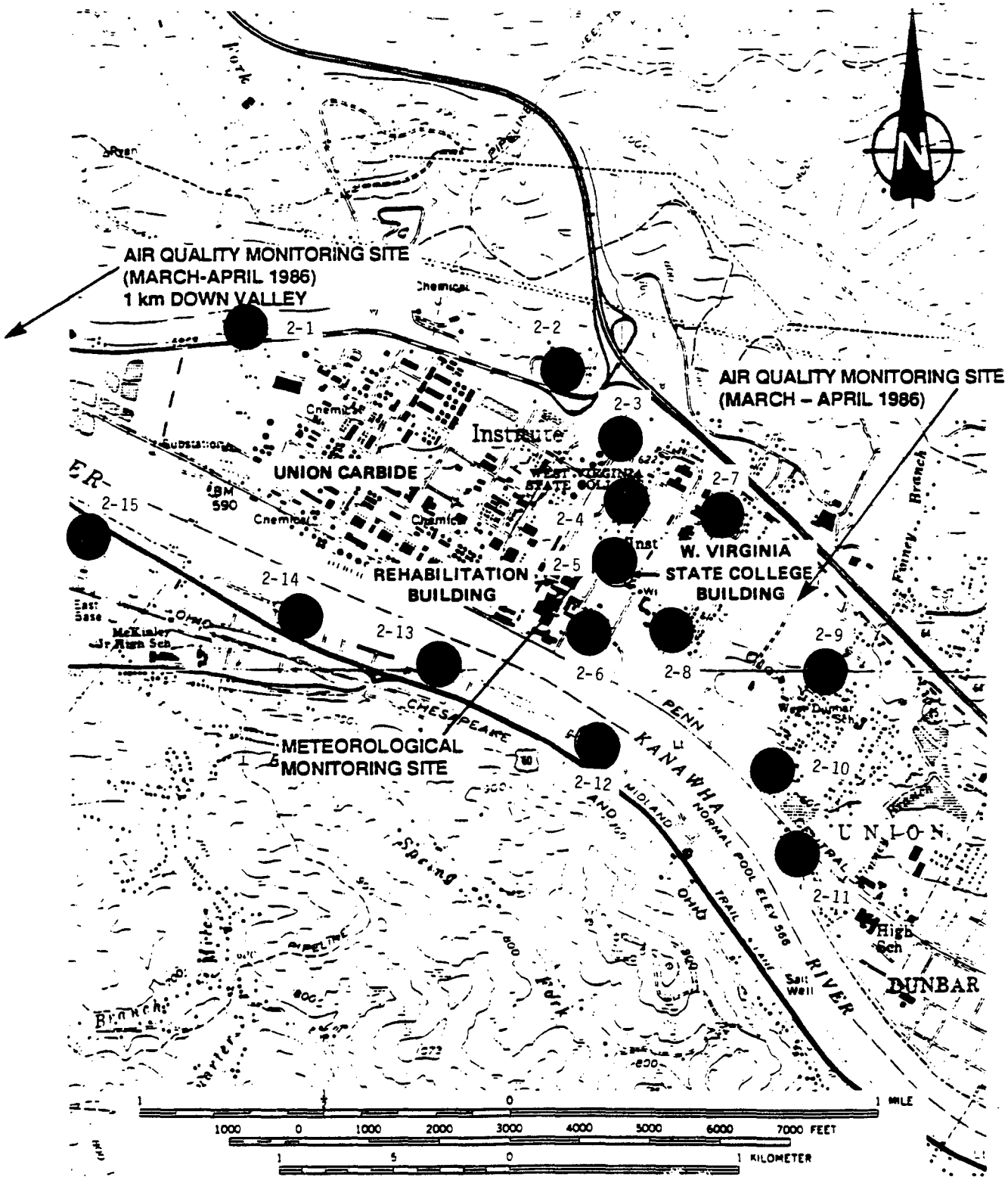


FIGURE 5

KANAWHA VALLEY TOXICS SCREENING STUDY
KANAWHA VALLEY AIR QUALITY REPORT

APPROXIMATE LOCATIONS OF NEIGHBORHOOD RECEPTORS, MAJOR FACILITIES, AND MONITORING SITES FOR THE NITRO COMPLEX

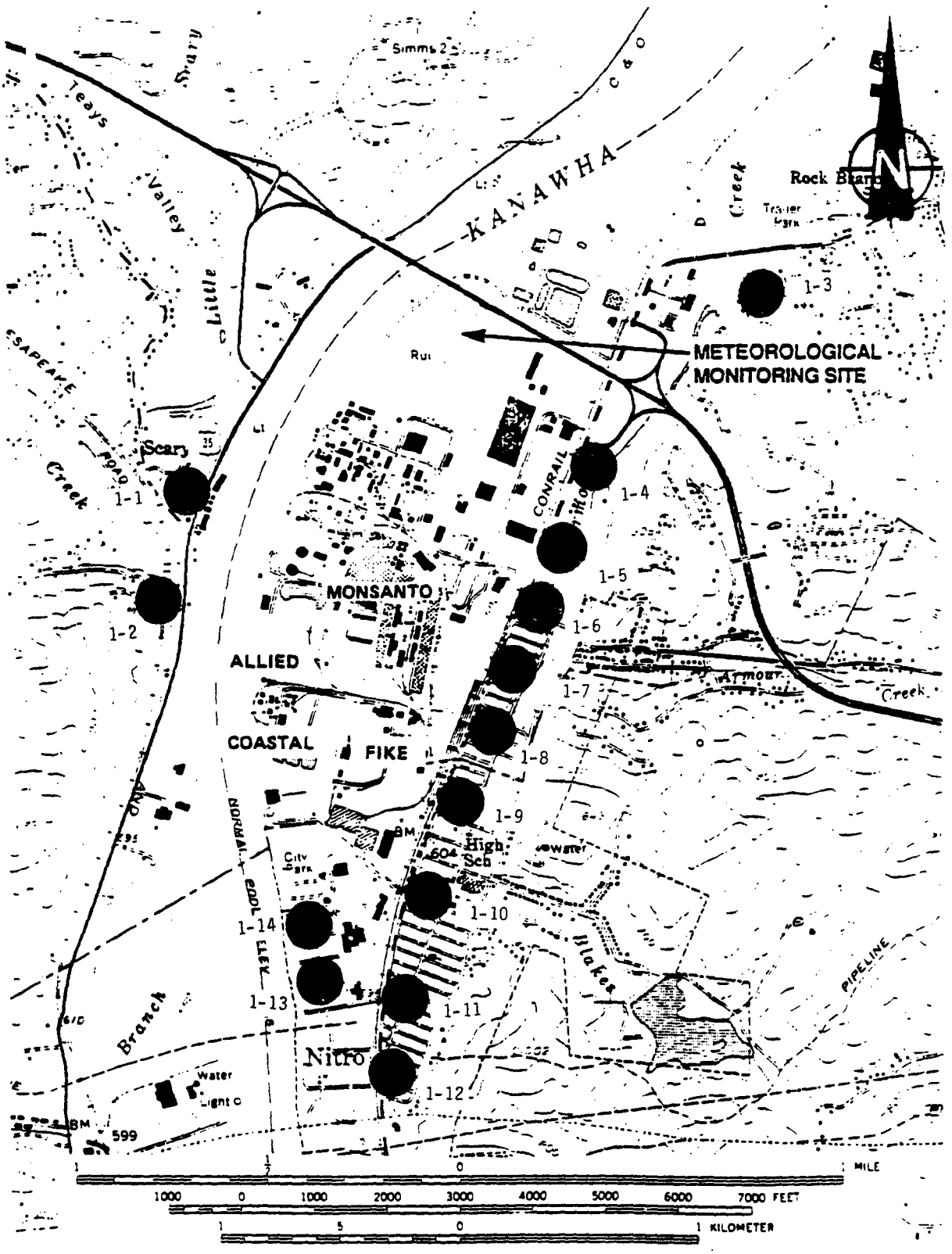
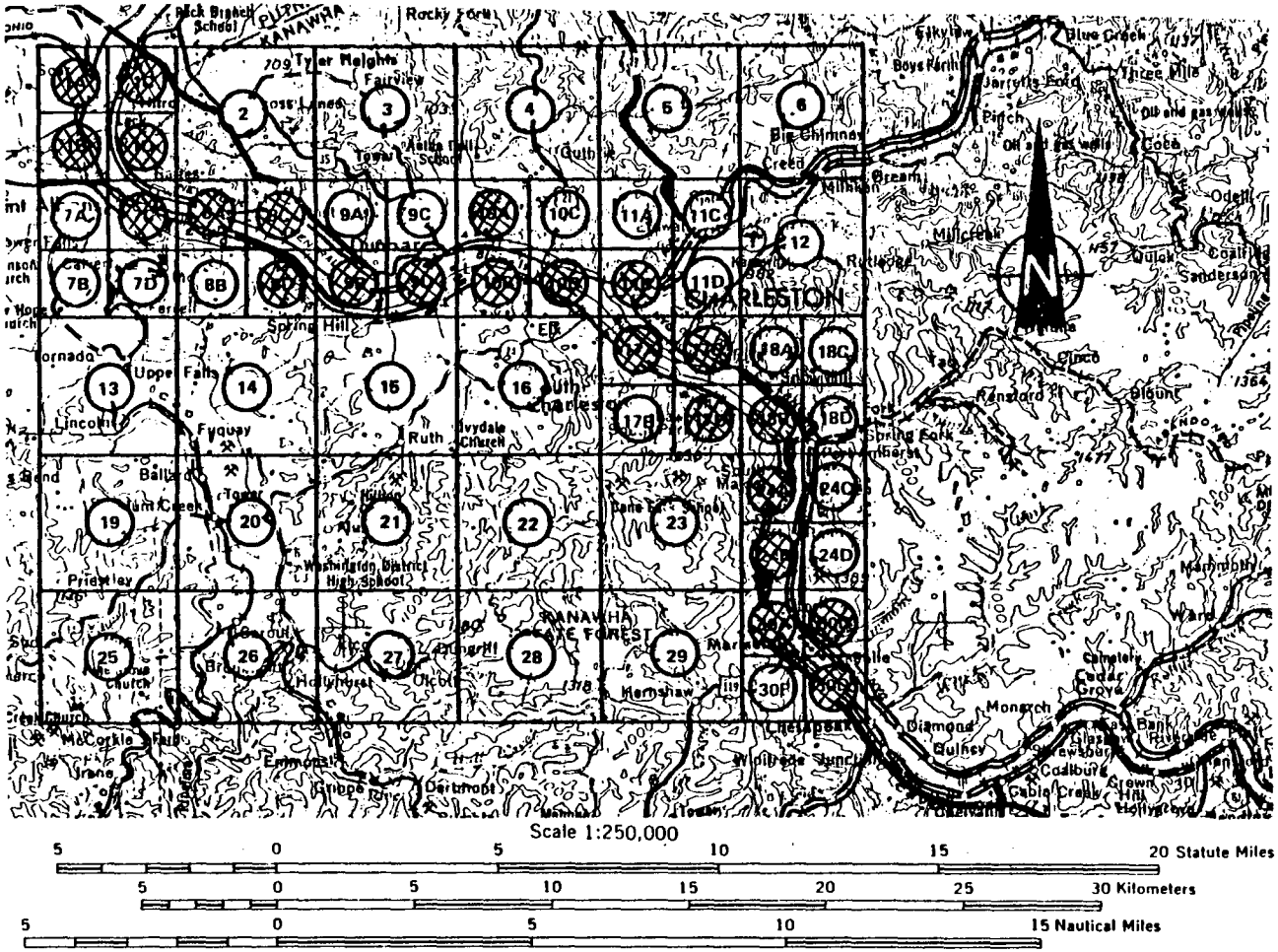


FIGURE 6

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

THE GENERAL RECEPTOR GRID AND CORRESPONDING 1980 POPULATION VALUES TO BE USED IN THE KANAWHA VALLEY STUDY TO THE AVERAGE INDIVIDUAL EXPOSURE



Cross Hatch Indicates those areas modeled

POPULATION FOR KANAWHA VALLEY AREA*

GRID NUMBER	A	B	C	D	TOTAL
1	1,360	2,800	2,790	2,290	9,240
2	-	-	-	-	7,830
3	-	-	-	-	4,400
4	-	-	-	-	2,650
5	-	-	-	-	2,500
6	-	-	-	-	2,630
7	750	750	3,000	1,570	6,070
8	7,150	1,580	1,490	870	11,090
9	2,160	7,720	2,070	6,170	18,120
10	2,450	5,640	5,960	9,640	23,690
11	7,570	6,510	710	2,500	17,290
12	-	-	-	-	2,120
13	-	-	-	-	3,280
14	-	-	-	-	3,730
15	-	-	-	-	6,500
16	-	-	-	-	7,920
17	8,890	1,660	3,780	1,760	16,090
18	950	4,530	340	340	6,160
19	-	-	-	-	900
20	-	-	-	-	1,080
21	-	-	-	-	1,320
22	-	-	-	-	1,320
23	-	-	-	-	1,110
24	1,950	1,440	320	300	4,010
25	-	-	-	-	900
26	-	-	-	-	900
27	-	-	-	-	900
28	-	-	-	-	900
29	-	-	-	-	560
30	2,730	140	1,870	3,010	7,750
				TOTAL	172,960

*DATA TAKEN FROM U.S. DEPARTMENT OF COMMERCE BUREAU OF THE CENSUS, 1983.

- Model inputs;
- Uncertainties and limitations;
- Results;
- Sensitivity analysis; and
- Ambient monitoring programs.

Simplifying Assumptions

Perhaps the most important simplifying assumption made in this study is that estimates of pollutant concentrations in ambient outside air are useful indicators of potential health risks. Such an assumption implicitly assumes that people are continuously exposed to ambient concentrations predicted for the neighborhood in which they reside.

Seven other major simplifying assumptions were used in the exposure assessment:

Meteorological conditions within each of the study zones were assumed to be homogeneous: While meteorological conditions actually vary within each valley zone, conditions throughout each zone were considered to be represented by data from the single station sited by this study within each zone. (For a discussion of the design of the meteorological monitoring system, see Technical Appendix B.)

Valley zones were assumed to have no influence on each other for the characterization of concentrations in the neighborhoods in proximity to the industrial facilities: In the Gaussian dispersion modeling, pollution transport between zones was assumed to be zero. In the sensitivity analysis, box model estimates, based on the total emissions throughout the study area, were made to provide a conservative estimate of annual average concentration to which the population is exposed, considering interzone transport. If average concentrations were to be addressed in more detail, however, extensive tracer studies would be needed to document transport and dispersion among valley zones, as well as model modifications to account for the restriction of horizontal plume spread.

County-wide area source emissions were based on national estimates: In addition to the pollutants emitted by the chemical facilities, the Kanawha Valley, like any metropolitan area, is exposed to toxic air pollutants associated with commercial and residential development. These include emissions from automobiles and trucks, solvent use (such as in dry cleaning), heating, gasoline marketing, and waste oil combustion. Since it would have been impractical to compile a detailed inventory of such sources, and since county-wide area source emissions are often quite similar from one location to another, emissions were estimated at the county level based on national averages. These estimates were then apportioned within the study area for modeling purposes on the basis of population density.

Atmospheric decay of pollutants was, with one exception, assumed to be zero: When released into the air, organic compounds can react with both ambient air and surface substances, thereby decreasing the compounds' concentration in the ambient air. Some pollutants are affected more than others; Table 4 presents available half-life data for the chemicals being modeled.

Half-life refers to the length of time (presented here in hours) that it would take, on average, to reduce a given pollutant's mass within a plume by 50 percent. As Table 4 indicates, the chemicals of interest to this study have half-lives much longer than typical transport times for the distances encountered in the valley, especially for the neighborhoods adjacent to the plants. For example, assuming a typical wind speed of 2 m/sec, it would usually take only a few minutes for pollutants to reach most nearby receptors and only about one to two hours to travel the 10 km to 15 km length of the longest valley zones.

For the purposes of this study, each pollutant was therefore assumed to have an infinite half-life. This term, therefore does not significantly affect modeling results. Furthermore, this assumption allows concentrations to be linearly scaled by emission rate, thereby facilitating data processing. An exception was made, however, for allyl chloride, which has a half-life of 4.8 hours (EPA, December 1985). The effect of decay for even this pollutant is relatively minor. Formaldehyde, on the other hand, which also has a relatively short half-life, was modeled conservatively assuming zero decay based on the upper end of the range shown in Table 4. (For a quantitative assessment of the sensitivity of this term, see also "Sensitivity Analysis" below.)

The population within the study area is assumed to live on the valley floor and not on the hillsides of the valley: A modification to the modeling protocol would have been needed to estimate pollutant concentrations at hillside receptors. Since it is likely that much of the population that is exposed to relatively high concentrations of these pollutants does live on the valley floor, no such modification was attempted. Limited modeling analysis was performed, however, to determine the magnitude of the concentration for hillside receptors. This analysis, contained in the sensitivity analysis section, indicates that predicted hillside concentrations are lower than those of the valley floor.

When apportioning population to the modeling grid, this study assumed that a census tract that overlapped into two or more grids could be apportioned based on the percentage of areas of the census tract in each modeling grid: Population enters the calculation of exposure in two ways: (1) it was used to apportion county-wide area source emissions into the modeling grids, and (2) the concentration of each grid is weighted by

TABLE 4

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

Atmospheric Half-life Values (Hours) for Pollutants Modeled

Pollutants	Half-Life (Hours) Low	High
Acrylonitrile	93.6	-
Benzene	144.0	-
Carbon Tetrachloride	192720.0	-
Chloroform	1920.0	-
Ethylene oxide	96.0	-
Formaldehyde	2.4	19.2
Methylene chloride	1380.0	-
Vinyl chloride	28.8	-
Vinylidene chloride	48.0	-
Ethylene chloride	864.0	3048.0
Allyl chloride	4.8	-
Trichloroethylene	88.8	-
1,3-Butadiene*	3.0	-
Propylene oxide	148.8	-

* This value applies to atmospheric half-life in the presence of full sunlight (Federal Register, October 10, 1985, 50 FR41466). The use of this value directly would have acted to understate risk. Zero atmospheric decay was therefore assumed for 1.3-Butadiene in order to conservatively estimate concentration.

Source: Office of Emergency and Remedial Response, Office of Solid Waste and Emergency Response, EPA, 1985.

the population of that grid to estimate weighted average exposures. Uniform population density was assumed within census tracts. For census tracts that include portions of the valley wall, or beyond, this is not likely to be a conservative assumption. Based on the apportionment method, approximately 100,000 people are found to live on the valley floor, between Belle and Nitro. It is not clear what percentage of the residents actually live on the valley floor.

A number of source categories were excluded from the analysis because they were assumed to be relatively insignificant or we were unable to quantify releases from such sources.

These source categories include:

- Volitilization from Waste Water treatment plants.
- Volatilization from the Kanawha River.
- Emission of point source metals and other inorganic pollutants not covered by the WVAPCC inventory.
- Emissions from abandoned hazardous waste disposal sites and, in some cases, active waste management facilities.
- Emissions from small facilities that were not covered in the WVAPCC inventory.
- Emissions associated with accidental releases.

Model Selection

Numerous EPA dispersion models could have been used for this study. Based on a review of the features of the models included in the "User's Network of Applied Modeling of Air Pollution" (UNAMAP)[4], ISCLT (Bowers and Bjorklund 1979) was selected as the primary model to be used for this study as the basis of risk assessment. ISCLT is EPA's recommended model for complex industrial sources for flat terrain applications (EPA 1986b), and since all receptors modeled in this application are on the valley floor, complex terrain was not an issue in model selection. In addition, however, LONGZ (Bjorklund et al. 1982) was used as a supplement to ISCLT because it offers alternative treatments (discussed below) for modeling area sources and handling turbulent intensity data. ISCLT's results are reported in this volume; LONGZ's results can be found in Technical Appendix A.

Both ISCLT and LONGZ fully meet the study's first objective of characterizing concentrations in neighborhoods nearest the chemical plants. They are less reliable in estimating annual average exposures to the average individual due to possible transport and valley wall

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4 UNAMAP V was the most current version available for use in this study at the time the modeling was performed.

reflection of pollutants throughout each valley zone the study's second objective. For this reason, a generic "box model" approach was used in addition to ISCLT and LONGZ to estimate upper bound average annual concentrations throughout the valley to account for interzonal transport.[5]

Area Sources: Since ISCLT uses a "virtual point" concept to represent area sources, it treats emissions from a particular area source as if they were all released at a location sufficiently upwind of the source so that the horizontal spread of the plume would approximate the dimensions of the area source at the location of the source. For this reason, it cannot properly compute estimates of concentrations at receptors located within an area source. Although it would be technically possible to minimize this problem by repeatedly subdividing area sources, it would be impractical to do this on as large a scale as this study required. LONGZ, on the other hand, uses an alternative area source procedure that offered the potential for better characterization of area sources. For the risk assessment, we used box model concentrations for exposure from county-wide area sources.[6]

Turbulent Intensity Data: ISCLT is well suited for addressing the complex industrial sources on the valley floor, but it cannot evaluate site-specific dispersion conditions. Although LONGZ uses similar treatments for handling industrial sources, it also is able to accept turbulent intensity data such as those collected by this study's meteorological monitoring network. Site-specific turbulent intensity data have the potential to improve model performance because, especially during stable nighttime air flow, dispersion conditions within the valley can vary markedly from flat terrain settings. ISCLT would not be able to distinguish between these two conditions.[7]

Model Inputs

Principal inputs to air dispersion analysis are (1) meterological data, (2) emissions data, (3) receptors, and (4) classification of land use. This section discusses these inputs in turn.

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5 Refer to Technical Appendix F for a discussion of the input data and approach used for the box model analysis.

6 Ultimately, the box model's (Hanna 1982) characterization of area sources were found to be the most conservative and were therefore used in the risk assessment.

7 Unfortunately, LONGZ's empirical formulae for relating turbulent intensity data to plume dimensions do not appear to be representative of near-surface releases. This weakness results in LONGZ's predicting concentrations lower than those predicted by ISCLT by a factor of roughly 2 to 3 for Institute and Belle, where turbulent intensity data were incorporated into the modeling analysis. Refer to Technical Appendix A for further details on this issue.

Meteorological Data: Atmospheric stability data are used in air dispersion modeling to estimate the width and vertical extent of a plume as a function of downwind distance. They provide a measure of atmospheric mixing, which acts to spread a plume along its horizontal and vertical axes. Turbulent intensity data can be used in modeling to provide a more direct measure of horizontal and vertical dispersion than is possible through the use of default stability data.

To develop such data for the purposes of this study, wind speed, wind direction, and the standard deviation of horizontal wind direction were monitored in four valley zones at heights ranging from 10 m to 20 m. Vertical wind speeds were measured in two valley zones, Belle and Institute, to help characterize vertical turbulent intensity; these measurements were used to estimate vertical dispersion coefficients for the LONGZ model runs (see discussion above).

The primary recording system was a data logger located at the WVAPCC headquarters. The data logger was connected to the remote sites by telemetry, such that the data could be displayed on a real-time basis or archived. At each remote station, data were also collected on strip chart or paper tape as a backup.

As noted earlier, the study subdivided the study area into four zones so that the valley orientation within each zone was relatively uniform. A meteorological monitoring site was at a representative location within each zone. A six-month data set was collected in all four zones from December 6, 1985, to June 6, 1986. Technical Appendix B describes the design and results of the meteorological monitoring program in detail.

A quality assurance plan that specified the tolerances and operating procedures for the meteorological monitoring program was written and approved (EPA 1986). Part of this plan specified that an independent systems audit would be performed annually. The first audit was performed in April 1986, with the system generally found to be operating within tolerance.

Ambient temperature data input to the model was collected at Kanawha Airport. This input was not generally a sensitive parameter because plume rise is not a factor except for stack releases, which, in this study, were found to be a relatively minor component. Although some differences in ambient temperature would be expected between the airport and the valley floor, even for stack emissions the effect on model output of these differences should be small.

Emissions Data: All the point source emissions data used in this study were developed by the WVAPCC in its 1984 survey of air emissions from West Virginia manufacturers.

The goal of this survey was explicity to obtain data to assess the impact of toxic air pollutants on air quality. It included all sources emitting more than 100 tons/year of criteria pollutants, with some exclusions. Power plants were not surveyed because their organic toxic pollutant releases are expected to be low. The most significant power plant in the area is the Amos facility, located approximately 4 km northwest of Nitro. Since its effective release heights are high and its expected organic toxic emissions are relatively low, no effort was made to estimate independent emissions data for this plant for the purposes of this study. The WVAPCC inventory also excluded small processing plants and blending facilities; they were omitted because an earlier WVAPCC inventory found them to be insignificant in relation to the major chemical facilities.

WVAPCC's questionnaire was sent to approximately 40 companies, primarily chemical manufacturers, petroleum refineries, and miscellaneous firms that potentially emit significant quantities of volatile organics. Within the study area, 17 facilities were sent questionnaires, and all responded. The seven facilities in the study area that emit pollutants of concern for this study are shown in Table 2.

Three major categories of sources were inventoried by the WVAPCC using a wide variety of methods:

> Process emissions - Industries were allowed to calculate process emissions by any technique that they felt would provide the best estimate. A variety of approaches were used, including use of ideal gas laws, computer model emissions estimates, and test data. References were requested for any method used.
>
> Combustion emissions - These emissions were based on the "Compilation of Air Emission Factors" AP-42, (USEPA 1984), supplemented with data to describe the specific volatile organic emissions.
>
> Fugitive emissions - Fugitive emissions are those that are not released from specific vents or stacks, such as emissions from leaking pipes and loading/unloading emissions. The WVAPCC instructed industries to estimate emissions based on specific EPA emission factors (USEPA 1986). Tank emissions were estimated by standard formulas (USEPA 1984).

Of these, equipment leaks within fugitive emissions were the most difficult to quantify in many cases. WVAPCC offered four different options for approaching this problem of estimating equipment leaks. In increasing levels of refinement, these were:

Option 1 - The number of pumps are counted, and a standard ratio of pumps to valves, flanges, and other components is assumed. Loss of product is then estimated based on EPA emission factors (EPA 1986), assuming a generic percentage of leaking components.

Option 2 - This option is the same as Option 1, except the component counts include open ended lines, compressors, relief valves, sampling connections, valves, and flanges in addition to pumps.

Option 3 - Option 3 requires field measurements to determine the percentage of sources within a facility that leak, i.e., leak/ no-leak approach. Once the leak/no-leak percentage is determined for each component, EPA emission factors (leaking and non-leaking) are used to estimate emissions (EPA 1986).

Option 4 - Option 4 involves performing facility-specific testing to determine the emissions estimates. This option would require detailed pollutant-specific monitoring of numerous release points and would be resource- and time-intensive.

Most facilities used Options 1 or 2 as the basis for the emissions estimates. In many cases, Option 1 was used because accurate component counts were not available. Only the Rhone Poulenc plant and Dupont used the leak/no-leak approach (Option 3) and then only for a selected number of units, totaling 14. None of the Option 3-generated data serve as the basis for the estimate of emissions of the 14 point source pollutants being evaluated in this study, i.e., all of the releases of the 14 point source pollutants addressed in this study are based on emission estimates using Option 1 or Option 2 data. Option 4 was not used for any emissions within the study area.

The data generated by the WVAPCC survey are voluminous. For the 17 facilities in the study region, a total of 2,258 release points and 570 pollutants were included. Since it was not feasible to verify all of the emissions data, the WVAPCC reviewed the data sets for reasonableness and spot-checked computations for release points that emitted large quantities of highly potent pollutants. For the Rhone Poulenc plant, the WVAPCC also requested backup calculations for 15 to 20 release points. An independent review was provided by the Office of Air Quality Planning and Standards of EPA for this special case.

For the purposes of this study, WVAPCC's emissions data were divided into daytime and nighttime releases if data were available for the hours of operation. This is important for two reasons: (1) release rates during the nighttime hours can be subject to poor dispersion conditions, and (2) emissions from some key sources are greatly reduced or even eliminated during the nighttime. This consideration is especially

important for the Kanawha Valley because of the likelihood of significant nocturnal drainage flows and stable conditions, which result in distinct daytime and nighttime flow patterns. Separation of daytime and nighttime emissions provides a more realistic treatment, especially from facilities or processes not in operation during the nighttime hours.

There are two limitations to consider concerning the use of the WVAPCC inventory for this study. First, the inventory includes recent and future voluntary reductions proposed by industry that have not been verified by WVAPCC or EPA (Farley 1986); current emissions of some pollutants may therefore have been underestimated. Second, there is some disagreement between industry and EPA concerning the accuracy of the emissions factors used to estimate fugitive sources. Industry argues that the emissions inventory overestimates some pollutant emissions by an order of magnitude or more because it relies on inappropriate emissions factors. To date, no supporting data have been received by the WVAPCC or EPA to confirm this position (Beard 1986).

An important aspect of the modeling assessment was the grouping of major facilities into logical modeling units. This permits air pollutant concentrations to be directly attributed to various components within a specific facility complex. For this study, every release point in the 1984 WVAPCC survey was assigned to a common process and modeling unit based on data provided by the WVAPCC. At the process level, a manageable number of release specifications were selected so that every release point for a given process could be assigned to one set of release specifications. For example, if a process had several stacks, a building with numerous vents, and a product storage area, the modeling analysis could be facilitated by modeling the stacks as separate sources, the vents as one building source, and the storage area as one area source. The computer software system linked modeled data with the extensive emissions inventory which has resolution to the release point level. Figure 7 provides an example of how source groupings were made.

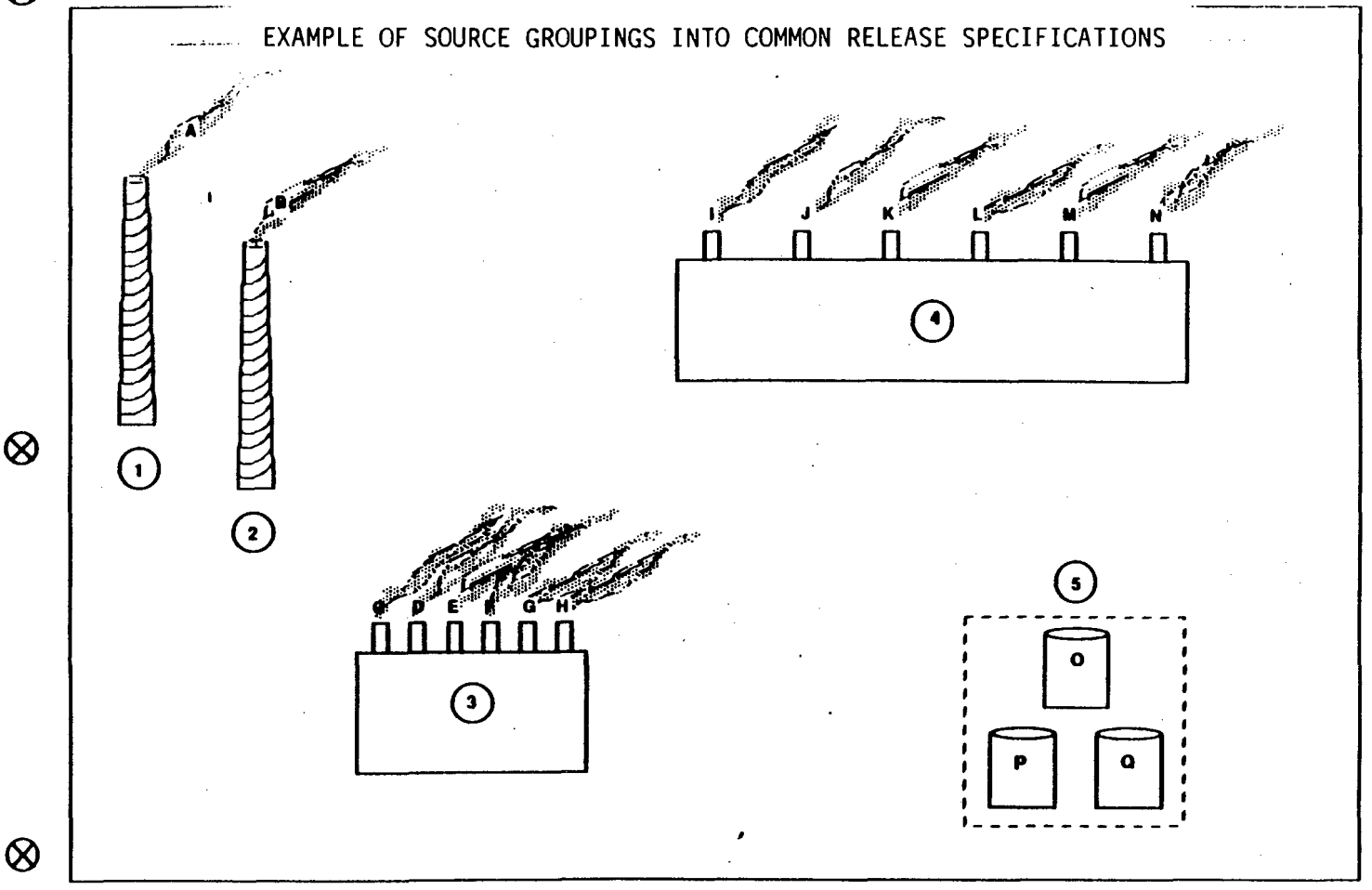
Since the WVAPCC inventory covers only industrial sources, this study independently estimated county-wide area sources for the study area. County-wide estimates were made for emissions of specific pollutants from solvent use, road vehicles, gasoline marketing, heating, and waste oil consumption. These emissions were then apportioned into a modeling grid along the valley floor. (Refer to Technical Appendix D for details on this methodology.)

Receptors: A limited number of receptors have been used in this study. The maximum values shown for the receptors in this study will probably be lower than those associated with a denser array of receptors, because denser spacing of receptors would increase the probability of locating a receptor near the offsite absolute maximum concentration.

FIGURE 7

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT



GROUP	SOURCE TYPE	SPECIFIC RELEASE POINTS
1	STACK	A
2	STACK	B
3	VOLUME SOURCE (BUILDING)	C,D,E,F,G,H
4	VOLUME SOURCE (BUILDING)	I,J,K,L,M,N
5	AREA SOURCE	O,P,Q

For each receptor within a valley zone, annual average concentrations were estimated based on the impacts from each source that emits pollutants addressed in this study. Then the impacts from each industrial source within each valley zone were summed at each receptor to estimate a total for annual average ambient concentrations. Two types of receptors were modeled. First, a set of receptors was placed at pre-selected locations in neighborhoods expected to be more affected by releases. Second, a uniform grid was established in each zone as shown in Figure 6. Four equally spaced receptors within a grid were used to compute an average concentration for each grid cell (2.5 km by 2.5 km). The exposure to the average individual is based on the concentrations that were weighted by population in each grid. To determine the exposure to the average exposed individual, these population-weighted concentrations are added and the sum is divided by the total population of the zone.

The approach used to link concentration fields with population is consistent with the approaches used in the EPA Graphical Exposure Modeling System (GEMS) and Human Exposure Model (HEM). One exception is the treatment in HEM for receptors within 3.5 km of a source. Within 3.5 km, HEM assigns population to model receptors, a different approach from the treatment used here or in GEMS. The effect of this difference on population-weighted averages is likely to be small.

Classification of Land Use: For the purposes of dispersion modeling, an area can be characterized as "rural" or "urban"; the distinction can be made on the basis of either population density or land use. This study used the preferred land-use method (Auer 1978) of characterizing all areas within a 3 km radius of each industrial zone. The results are as follows:

Valley Zone	Percent Urban	Percent Rural
Belle	6	94
Charleston	25	75
Institute	14	86
Nitro	14	86

Since all areas were considerably more than 50 percent rural, all areas were classified as rural for modeling purposes.

Summary of Input Data: Technical Appendix E documents the specific input files used for the ISCLT modeling analysis.

Uncertainties and Limitations

Modeled ambient concentrations provide the basis, with toxicological information, for estimating potential life-time incremental risks to

individuals and the population within this study. It is important to understand the limitations of the modeled concentrations that provide the basis for calculating these risk estimates.

Uncertainty in Emissions: The modeling results are critically dependent on the emissions inventory compiled for 1984 by the State of West Virginia. The inventory details the pollutants and the various associated quantities for the numerous release points in the Kanawha Valley with estimates of emissions based on generally accepted engineering calculation procedures performed by the sources themselves. Some comments have been made regarding the applicability of generic EPA emissions factors and techniques to specific processes in the Kanawha Valley. Fugitive emissions factors and techniques developed for the chemical industry overall have been used for estimating emissions from specific process units. In some instances, the resulting estimates for fugitive emissions represent a large contribution to the modeled ambient concentrations of the pollutants being studied. Industry has suggested that, for some pollutants, emissions estimates for fugitive releases may be an order of magnitude or more higher than actual emissions.

Fugitive emissions for these pollutants are, at times, the predominant contributors to the predicted ambient concentrations. This report attempts to separate out fugitive emissions for the major pollutants within the study to understand the importance of such releases for the risk assessment.

In addition, the emissions inventory is based on the calendar year 1984. Although the WVAPCC has updated the emissions for several pollutants to reflect the more current operating status of the major facilities, most emissions reflect operating status as of 1984. Furthermore, voluntary reductions reflected in the inventory have not yet been documented to EPA.

Uncertainty in Modeling: Gaussian modeling results are generally considered to be accurate within a factor of two on a seasonal or annual basis. For neighborhoods adjacent to the industrial complexes, a factor of two would still appear to be reasonable. As downwind distance increases, the Gaussian model treatment would act to systematically underestimate concentration because of the lack of a term in the models to represent valley wall reflection. Therefore, as distance increases, it would be expected that the accuracy of Gaussian modeling would decrease.

The Gaussian modeling analysis does not consider interzone transport of pollutants. Although this does not significantly affect the analysis of concentrations in neighborhoods surrounding the industrial facilities, interzone transport could affect the estimate of the average individual exposure throughout the study area. Valley wall reflection and flow

between valley zones will act to provide higher concentrations than the ISCLT and LONGZ modeling performed for each zone independently. By using a box model approach, which assumes uniform concentration within the volume of the full extent of the valley within the study area (i.e., 45 km long, by 1 km wide and 0.3 km high), it is possible to conservatively estimate population-weighted concentrations. This study performed a bounding analysis to determine estimates of upper-bound concentrations of studied pollutants based on a box model approach, which accounts for such transport.

Dispersion Modeling Results

This section presents the results of the annual average model analysis. Tables 5 through 8 present the predicted annual average concentrations from industrial facilities for each pollutant in neighborhoods within each valley zone. Figures 2 through 5, previously shown, identify the locations of each neighborhood receptor. Population-weighted average concentrations for industrial sources are presented in Tables 9 through 12 for each pollutant and valley zone. The minimum and maximum concentrations based on this uniform grid system (four receptors per 2.5 km grid zone) are also displayed.

Because ISCLT cannot adequately model county-wide area sources, which in this case are 2500 m wide, the concentrations for county-wide area source emissions are not given in Tables 5 to 8. Box model estimates were used to compute conservative estimates of county-wide area sources. Table 13 presents these results. Refer to Technical Appendix F for a comparison of the conservatism in the box model compared to a Gaussian treatment.

Because of the conservative assumptions used to specify the vertical extent of the box, in this case 100 m, and the assumption of no loss of mass across the valley walls, the box model results are likely to be higher than actual impacts. LONGZ, however, can predict impacts for county-wide area sources. LONGZ results can therefore be used to show the relative contributions from industrial and county-wide area sources based on Gaussian modeling. (Refer to Technical Appendix A.)

Sensitivity Analyses

Sensitivity analyses were conducted to explore the effects of model inputs on predicted concentrations. The following were considered: source release type (area source, building source, or point source), atmospheric decay, and dispersion coefficients. Sensitivity analyses were made only for the Belle, Charleston/South Charleston, and Institute zones because concentrations in the Nitro zone are relatively low compared to those in the other three. In addition, uncertainties with

TABLE 5

KANAWHA VALLEY TOXICS SCREENING STUDY
KANAWHA VALLEY AIR QUALITY REPORT

PREDICTED CONCENTRATIONS IN ug/m^3
TOTAL CONCENTRATION AT NEIGHBORHOOD RECEPTOR SITE

BELLE

COMPOUND

	CARBON TETRACHLO	CHLOROFORM	FORMALDEHYDE	METHYLENE CHLORI
4- 1 (ISCLT)	2.3038	7.16591	.475676	9.24479
4- 2 (ISCLT)	2.59149	8.12297	.529156	10.8371
4- 3 (ISCLT)	3.01067	9.68793	.589595	13.094
4- 4 (ISCLT)	28.4684	99.8666	1.00269	117.002
4- 5 (ISCLT)	3.70601	13.6867	.405809	20.4641
4- 6 (ISCLT)	3.81577	13.3193	.374677	17.4906
4- 7 (ISCLT)	2.84737	9.62734	.301089	11.6548
4- 8 (ISCLT)	2.03969	6.84242	.261475	8.21528
4- 9 (ISCLT)	.956009	3.1887	.136497	3.94895
4-10 (ISCLT)	1.23694	4.14253	.155636	5.00111
4-11 (ISCLT)	1.3549	4.61279	.210001	6.10983
4-12 (ISCLT)	.833441	2.69129	.194972	3.53382
4-13 (ISCLT)	1.46127	4.97915	.197985	6.31872
4-14 (ISCLT)	1.74765	5.6652	.272773	7.02454
4-15 (ISCLT)	1.57092	5.00544	0.28446	6.20416

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

TABLE 5 (CONTINUED)

KANAWHA VALLEY TOXICS SCREENING STUDY
KANAWHA VALLEY AIR QUALITY REPORT

PREDICTED CONCENTRATIONS IN ug/m3
CONTRIBUTED BY FACILITY

Occidental

	CARBON TETRACHLO	CHLOROFORM	METHYLENE CHLORI
4- 1 (ISCLT)	2.00777	7.02782	8.14668
4- 2 (ISCLT)	2.2751	7.9746	9.26035
4- 3 (ISCLT)	2.71599	9.54677	11.1735
4- 4 (ISCLT)	28.0881	99.653	116.706
4- 5 (ISCLT)	3.51881	13.5832	20.1872
4- 6 (ISCLT)	3.64452	13.2317	17.2873
4- 7 (ISCLT)	2.71233	9.56027	11.4977
4- 8 (ISCLT)	1.92609	6.78704	8.08942
4- 9 (ISCLT)	.889854	3.15667	3.86211
4-10 (ISCLT)	1.1658	4.10745	4.88817
4-11 (ISCLT)	1.25786	4.56439	5.9541
4-12 (ISCLT)	.727827	2.63973	3.35816
4-13 (ISCLT)	1.3902	4.94213	6.13353
4-14 (ISCLT)	1.59405	5.59151	6.5572
4-15 (ISCLT)	1.40853	4.92877	5.72872

Dupont Belle

COMPOUND

	CARBON TETRACHLO	CHLOROFORM	FORMALDEHYDE	METHYLENE CHLORI
4- 1 (ISCLT)	.296034	0.13809	.475676	1.0981
4- 2 (ISCLT)	.316385	.148369	.529156	1.57674
4- 3 (ISCLT)	0.29468	.141163	.589595	1.92049
4- 4 (ISCLT)	.380304	.203617	1.00269	.295919
4- 5 (ISCLT)	.187191	.103467	.405809	.276911
4- 6 (ISCLT)	0.17125	.087613	.374677	.203292
4- 7 (ISCLT)	.135042	.067073	.301089	.157014
4- 8 (ISCLT)	.113601	.055378	.261475	.125859
4- 9 (ISCLT)	.066156	.032037	.136497	0.08684
4-10 (ISCLT)	.071136	.035078	.155636	.112937
4-11 (ISCLT)	.097043	.048399	.210001	.155726
4-12 (ISCLT)	.105615	.051557	.194972	.175659
4-13 (ISCLT)	.071074	.037021	.197985	.185191
4-14 (ISCLT)	.153607	.073685	.272773	.467343
4-15 (ISCLT)	.162336	.076679	0.28446	.475445

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

TABLE 6

KANAWHA VALLEY TOXICS SCREENING STUDY
KANAWHA VALLEY AIR QUALITY REPORT

PREDICTED CONCENTRATIONS IN ug/m^3
TOTAL CONCENTRATION AT NEIGHBORHOOD RECEPTOR SITE

CHARLESTON/SOUTH CHARLESTON

COMPOUND

	ACRYLONITRILE	ETHYLENE OXIDE	METHYLENE CHLORIDE	PROPYLENE OXIDE	VINYLIDENE CL (1)
3- 1 (ISCLT)	.561643	2.82028	0.03708	5.24214	.010551
3- 2 (ISCLT)	0.34327	1.70196	0.01786	3.44946	.005825
3- 3 (ISCLT)	0.45022	1.40272	.017318	4.78489	.005583
3- 4 (ISCLT)	.891218	4.57533	.019908	14.8434	.010041
3- 5 (ISCLT)	1.22263	3.66757	.025567	5.71892	.015237
3- 6 (ISCLT)	0.9229	2.3589	.025446	3.61081	.010749
3- 7 (ISCLT)	.721844	1.70707	.027619	2.26287	.008678
3- 8 (ISCLT)	10.12	3.14244	0.06748	3.31826	.017876
3- 9 (ISCLT)	3.83297	4.87921	.083575	4.06942	0.02458
3-10 (ISCLT)	4.39437	9.65421	.053463	7.92983	.038734
3-11 (ISCLT)	11.2708	27.2998	.046699	15.0134	.072409
3-12 (ISCLT)	13.4238	46.0279	.035404	40.7283	.286163
3-13 (ISCLT)	5.30795	16.1771	.076571	10.4349	.062088
3-14 (ISCLT)	4.82406	27.0914	.048931	23.2062	.089732
3-15 (ISCLT)	1.35267	14.1306	.028777	25.4862	.025141
3-16 (ISCLT)	1.6677	11.2468	.039084	14.7202	0.03229
3-17 (ISCLT)	1.82633	10.3332	.067077	10.7268	.034677
3-18 (ISCLT)	1.76376	1.48224	.041647	1.79892	.009362

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

TABLE 6 (CONTINUED)

KANAWHA VALLEY TOXICS SCREENING STUDY
KANAWHA VALLEY AIR QUALITY REPORT

PREDICTED CONCENTRATIONS IN ug/m^3
CONTRIBUTED BY FACILITY

Carbide South Charleston

COMPOUND

	ACRYLONITRILE	ETHYLENE OXIDE	METHYLENE CHLORIDE	PROPYLENE OXIDE	VINYLIDENE CL (1)
3- 1 (ISCLT)	.560086	2.81666	7E-06	5.24214	.007705
3- 2 (ISCLT)	.342478	1.70017	6E-06	3.44946	.004407
3- 3 (ISCLT)	0.44942	1.40094	7E-06	4.78488	.004206
3- 4 (ISCLT)	.890283	4.57326	1E-05	14.8434	0.00845
3- 5 (ISCLT)	1.22148	3.66516	2E-05	5.71892	.013261
3- 6 (ISCLT)	.921744	2.35638	2E-05	3.61081	.008731
3- 7 (ISCLT)	.720571	1.70417	4E-05	2.26287	.006419
3- 8 (ISCLT)	10.1168	3.13463	8E-05	3.31825	.011943
3- 9 (ISCLT)	3.82894	4.86983	7E-05	4.06941	.017295
3-10 (ISCLT)	4.39232	9.64851	2E-04	7.92982	.034289
3-11 (ISCLT)	11.2687	27.2955	6E-05	15.0134	.068757
3-12 (ISCLT)	13.4271	46.0241	3E-05	40.7283	0.28327
3-13 (ISCLT)	5.30467	16.1697	3E-05	10.4349	.056649
3-14 (ISCLT)	4.82181	27.0862	2E-05	23.2062	.085817
3-15 (ISCLT)	1.35137	14.1275	1E-05	25.4862	.022831
3-16 (ISCLT)	1.66584	11.2424	1E-05	14.7202	.029014
3-17 (ISCLT)	1.82365	10.3286	1E-05	10.7267	.029514
3-18 (ISCLT)	1.76188	1.47758	5E-05	1.79892	.005782

Tech Center

COMPOUND

	ACRYLONITRILE	ETHYLENE OXIDE	METHYLENE CHLORIDE	PROPYLENE OXIDE	VINYLIDENE CL (1)
3- 1 (ISCLT)	.001556	.003661	.037073	4E-06	.002846
3- 2 (ISCLT)	8E-04	.001792	.017854	2E-06	.001398
3- 3 (ISCLT)	8E-04	.001779	.017311	2E-06	.001377
3- 4 (ISCLT)	9E-04	.002076	.019897	2E-06	.001591
3- 5 (ISCLT)	.001145	.002403	.025543	3E-06	.001976
3- 6 (ISCLT)	.001156	.002521	.025422	3E-06	.002018
3- 7 (ISCLT)	.001273	.002901	.027583	3E-06	.002259
3- 8 (ISCLT)	.003169	.007814	.067404	8E-06	.005933
3- 9 (ISCLT)	.004025	.009371	.083504	10E-06	.007284
3-10 (ISCLT)	.002554	.005702	.053275	6E-06	.004445
3-11 (ISCLT)	.002137	.004331	.046642	5E-06	.003652
3-12 (ISCLT)	.001709	.003798	.035379	4E-06	.002893
3-13 (ISCLT)	.003281	.007413	.076538	7E-06	.005439
3-14 (ISCLT)	.002253	.005211	.048912	5E-06	.003915
3-15 (ISCLT)	.001296	.003111	.028765	3E-06	.002311
3-16 (ISCLT)	.001861	.004363	.039073	4E-06	.003276
3-17 (ISCLT)	.002883	.006619	.067066	7E-06	.005163
3-18 (ISCLT)	.001885	.004662	.041599	5E-06	0.00358

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

TABLE 7

KANAWHA VALLEY TOXICS SCREENING STUDY
KANAWHA VALLEY AIR QUALITY REPORT

PREDICTED CONCENTRATIONS IN ug/m^3
TOTAL CONCENTRATIONS

INSTITUTE

COMPOUND

	ACRYLONITRILE	BENZENE	CHLOROFORM	ETHYLENE OXIDE	FORMALDEHYDE	METHYLENE CHLORIDE	PROPYLENE OXIDE	1,3-BUTADIENE
2- 1 (ISCLT)	6.56565	.031644	7.48184	14.4919	.012949	2.59974	10.5372	21.2178
2- 2 (ISCLT)	1.31098	0.00545	3.03608	6.48205	.007104	1.18378	5.11137	3.01951
2- 3 (ISCLT)	.862691	.004247	6.26104	7.30372	.010967	3.00909	4.4901	1.4678
2- 4 (ISCLT)	.721332	.003876	6.38188	6.28709	.014694	2.42746	3.66392	1.06844
2- 5 (ISCLT)	0.62025	.003911	4.66446	5.07301	.014976	1.4437	3.14118	.849235
2- 6 (ISCLT)	0.50318	.003022	2.96079	3.76416	.012407	1.28641	2.40557	.747657
2- 7 (ISCLT)	0.48574	.003017	2.69098	3.16921	.007407	.879308	2.09304	.679536
2- 8 (ISCLT)	.401021	.002667	1.98069	2.59141	.007036	.609127	1.74668	.528887
2- 9 (ISCLT)	.281138	.002083	1.0754	1.52315	.003418	0.31214	1.06631	.351693
2-10 (ISCLT)	.250313	.001876	.897757	1.34982	0.00299	.275497	.918914	.314766
2-11 (ISCLT)	.199069	.001539	.662593	.978149	.001953	.200141	.632414	.248728
2-12 (ISCLT)	0.38929	.002484	2.2374	2.44371	.006321	.712993	1.58022	.568193
2-13 (ISCLT)	.843106	.004155	3.04252	5.19491	.038778	.512306	4.42911	1.4104
2-14 (ISCLT)	1.02914	.006388	2.58381	12.9568	.040425	.593078	3.8409	1.08941
2-15 (ISCLT)	2.48409	.024218	5.83403	18.977	.034426	1.35598	9.61468	1.12071

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

TABLE 8

KANAWHA VALLEY TOXICS SCREENING STUDY
KANAWHA VALLEY AIR QUALITY REPORT

TOTAL CONCENTRATIONS

NITRO

COMPOUND

	ACRYLONITRILE	BENZENE	CARBON TETRACHLORIDE	CHLOROFORM	FORMALDEHYDE	METHYLENE CHLORIDE	VINYL CHLORIDE	ETHYLENE CHLORIDE	ALLYL CHLORIDE	TRICHLOROETHYLENE
1- 1 (ISCLT)	2E-04	3E-05	3E-05	3E-05	.668361	2E-05	2E-05	10E-04	.021722	1.06686
1- 2 (ISCLT)	1E-04	3E-05	3E-05	3E-05	0.3782	1E-05	1E-05	8E-04	.020097	0.78389
1- 3 (ISCLT)	3E-04	5E-05	5E-05	5E-05	.457446	3E-05	3E-05	.001785	.046332	1.63816
1- 4 (ISCLT)	7E-04	1E-04	1E-04	1E-04	.984112	6E-05	6E-05	.004265	.115492	4.87016
1- 5 (ISCLT)	.001024	2E-04	2E-04	2E-04	.956076	9E-05	9E-05	.006249	.172869	4.71002
1- 6 (ISCLT)	.001607	3E-04	3E-04	3E-04	0.71295	1E-04	1E-04	.010399	.301345	2.76505
1- 7 (ISCLT)	.003813	7E-04	7E-04	7E-04	.697067	3E-04	3E-04	.021522	.582253	1.36783
1- 8 (ISCLT)	.005915	.001067	.001067	.001067	.859498	5E-04	5E-04	.033418	.893875	1.1818
1- 9 (ISCLT)	.002759	5E-04	5E-04	5E-04	.449896	2E-04	2E-04	.020166	.689724	.877357
1-10 (ISCLT)	.001114	2E-04	2E-04	2E-04	.256381	9E-05	9E-05	.007882	.256527	.713345
1-11 (ISCLT)	6E-04	9E-05	9E-05	9E-05	.172398	5E-05	5E-05	.003984	.129957	.553317
1-12 (ISCLT)	4E-04	6E-05	6E-05	6E-05	.136385	3E-05	3E-05	.002699	.087008	.464994
1-13 (ISCLT)	4E-04	7E-05	7E-05	7E-05	.175402	3E-05	3E-05	.002772	.088565	.474901
1-14 (ISCLT)	5E-04	8E-05	8E-05	8E-05	.227896	4E-05	4E-05	.003549	.119677	.543989

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

TABLE 8 (CONTINUED)

PREDICTED CONCENTRATIONS IN ug/m^3

BY FACILITY

NITRO

Artel

COMPOUND

	ACRYLONITRILE	BENZENE	CARBON TETRACHLORIDE	CHLOROFORM	FORMALDEHYDE	METHYLENE CHLORIDE	VINYL CHLORIDE	ETHYLENE CHLORIDE	ALLYL CHLORIDE
1- 1 (ISCLT)	2E-04	3E-05	3E-05	3E-05	0.02133	2E-05	2E-05	10E-04	.021722
1- 2 (ISCLT)	1E-04	3E-05	3E-05	3E-05	.015996	1E-05	1E-05	8E-04	.020097
1- 3 (ISCLT)	3E-04	5E-05	5E-05	5E-05	.032522	3E-05	3E-05	.001785	.046332
1- 4 (ISCLT)	7E-04	1E-04	1E-04	1E-04	0.07637	6E-05	6E-05	.004265	.115492
1- 5 (ISCLT)	.001024	2E-04	2E-04	2E-04	.110699	9E-05	9E-05	.006249	.172869
1- 6 (ISCLT)	.001607	3E-04	3E-04	3E-04	.185525	1E-04	1E-04	.010399	.301345
1- 7 (ISCLT)	.003813	7E-04	7E-04	7E-04	.398581	3E-04	3E-04	.021522	.582253
1- 8 (ISCLT)	.005915	.001067	.001067	.001067	.614097	5E-04	5E-04	.033418	.893875
1- 9 (ISCLT)	.002759	5E-04	5E-04	5E-04	.284377	2E-04	2E-04	.020166	.689724
1-10 (ISCLT)	.001114	2E-04	2E-04	2E-04	.110308	9E-05	9E-05	.007882	.256527
1-11 (ISCLT)	6E-04	9E-05	9E-05	9E-05	.056183	5E-05	5E-05	.003984	.129957
1-12 (ISCLT)	4E-04	6E-05	6E-05	6E-05	.038293	3E-05	3E-05	.002699	.087008
1-13 (ISCLT)	4E-04	7E-05	7E-05	7E-05	.040934	3E-05	3E-05	.002772	.088565
1-14 (ISCLT)	5E-04	8E-05	8E-05	8E-05	.055317	4E-05	4E-05	.003549	.119677

Monsanto

COMPOUND

	FORMALDEHYDE	TRICHLOROETHYLENE
1- 1 (ISCLT)	0.64703	1.06686
1- 2 (ISCLT)	.362204	0.78389
1- 3 (ISCLT)	.424924	1.63816
1- 4 (ISCLT)	.907742	4.87016
1- 5 (ISCLT)	.845377	4.71002
1- 6 (ISCLT)	.527425	2.76505
1- 7 (ISCLT)	.298487	1.36783
1- 8 (ISCLT)	.245401	1.1818
1- 9 (ISCLT)	.165519	.877357
1-10 (ISCLT)	.146073	.713345
1-11 (ISCLT)	.116215	.553317
1-12 (ISCLT)	.098092	.464994
1-13 (ISCLT)	.134468	.474901
1-14 (ISCLT)	.172579	.543989

TABLE 9

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

PRELIMINARY RISK SCREENING RESULTS
INTENDED FOR POLICY DEVELOPMENT

CONCENTRATIONS TO THE AVERAGE EXPOSED INDIVIDUAL
(ug/m^3)

BELLE

Population for Belle: 15,530

COMPOUND	MINIMUM MAIN GRID CONCENTRATION	AVERAGE EXPOSED INDIVIDUAL CONCENTRATION (POPULATION-WEIGHTED)	MAXIMUM MAIN GRID CONCENTRATION
Carbon tetrachloride	8.70E-02	2.36E+00	1.33E+01
Chloroform	2.78E-01	7.86E+00	4.50E+01
Formaldehyde	1.48E-02	4.76E-01	2.97E+00
Methylene chloride	3.16E-01	9.19E+00	5.22E+01

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987.

TABLE 10

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

PRELIMINARY RISK SCREENING RESULTS
INTENDED FOR POLICY DEVELOPMENT

CONCENTRATIONS TO THE AVERAGE EXPOSED INDIVIDUAL
(ug/m^3)

CHARLESTON/SOUTH CHARLESTON

Population for Charleston/South Charleston: 51,750

COMPOUND	MINIMUM MAIN GRID CONCENTRATION	AVERAGE EXPOSED INDIVIDUAL CONCENTRATION (POPULATION-WEIGHTED)	MAXIMUM MAIN GRID CONCENTRATION
Acrylonitrile	8.40E-02	9.28E-01	7.53E+00
Ethylene oxide	1.50E-01	2.19E+00	3.01E+01
Methylene chloride	1.37E-02	5.01E-01	1.09E+01
Propylene oxide	1.80E-01	2.09E+00	2.03E+01
Vinylidene chloride (1,1-Dichloroethene)	1.79E-03	2.76E-02	3.61E-01

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987.

TABLE 11

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

PRELIMINARY RISK SCREENING RESULTS
INTENDED FOR POLICY DEVELOPMENT

CONCENTRATIONS TO THE AVERAGE EXPOSED INDIVIDUAL
(ug/m^3)

INSTITUTE

Population for Institute: 22,390

COMPOUND	MINIMUM MAIN GRID CONCENTRATION	AVERAGE EXPOSED INDIVIDUAL CONCENTRATION (POPULATION-WEIGHTED)	MAXIMUM MAIN GRID CONCENTRATION
Acrylonitrile	9.71E-02	1.61E+00	6.74E+00
Benzene	8.29E-04	1.74E-02	8.90E-02
Chloroform	2.55E-01	2.41E+00	1.96E+01
Ethylene oxide	4.13E-01	5.81E+00	3.06E+01
Formaldehyde	7.14E-04	9.21E-03	1.40E-01
Methylene chloride	6.94E-02	6.67E-01	2.94E+00
Propylene oxide	2.77E-01	3.57E+00	3.84E+01
1,3-Butadiene	1.03E-01	1.28E+00	6.53E+00

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987.

TABLE 12

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

PRELIMINARY RISK SCREENING RESULTS
INTENDED FOR POLICY DEVELOPMENT

CONCENTRATIONS TO THE AVERAGE EXPOSED INDIVIDUAL
(ug/m^3)

NITRO

Population for Nitro: 9,990

COMPOUND	MINIMUM MAIN GRID CONCENTRATION	AVERAGE EXPOSED INDIVIDUAL CONCENTRATION (POPULATION-WEIGHTED)	MAXIMUM MAIN GRID CONCENTRATION
Acrylonitrile	5.02E-05	8.40E-04	4.01E-03
Benzene	8.52E-06	1.47E-04	7.03E-04
Carbon tetrachloride	8.52E-06	1.47E-04	7.03E-04
Chloroform	8.52E-06	1.47E-04	7.03E-04
Formaldehyde	3.95E-02	4.08E-01	1.59E+00
Methylene chloride	4.26E-06	7.36E-05	3.52E-04
Vinyl chloride (chloroethene)	4.26E-06	7.36E-05	3.52E-04
Ethylene chloride-EDC	3.45E-04	5.23E-03	2.51E-02
Allyl chloride	9.62E-03	1.51E-01	7.39E-01
Trichloroethylene	1.28E-01	1.77E+00	1.01E+01

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987.

Table 13

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

COUNTY-WIDE AREA SOURCES BASED ON BOX MODEL ANALYSIS

	Emissions (County-Wide Area Sources) (metric tons)				Modeled Concentration (ug/m^3)			
					Box Model			
Pollutant Zone	Belle	Charleston	Institute	Nitro	Belle	Charleston	Inst.	Nitro
Benzene	14.4084	41.7199	18.7094	7.31173	3.0	4.8	2.4	0.9
Perchloroethylene	12.5941	36.4665	16.3535	6.39104	2.4	4.2	2.1	0.9
Methylene chloride	7.03098	20.3584	9.12978	3.56796	1.5	2.4	1.2	0.3
Formaldehyde	6.6573	19.0323	8.5351	3.33555	1.2	2.1	1.2	0.3
Trichloroethylene	1.67983	4.86398	2.18127	0.85245	0.3	0.6	2.9×10^{-1}	1.0×10^{-1}
1,3-Butadiene	0.10648	0.308316	0.138265	0.054035	2.1×10^{-2}	3.6×10^{-2}	1.8×10^{-2}	6.6×10^{-3}
Ethylene chloride	0.037012	0.10717	0.048061	0.018782	7.2×10^{-3}	1.2×10^{-2}	6.3×10^{-3}	2.3×10^{-3}
Ethylene bromide	0.017362	0.050272	0.022545	0.008811	3.3×10^{-3}	5.7×10^{-3}	3.0×10^{-3}	1.1×10^{-3}
Arsenic	0.012881	0.037296	0.016725	0.006536	2.6×10^{-3}	4.2×10^{-3}	2.2×10^{-3}	7.8×10^{-4}
Benzo(a)pyrene	0.005412	0.015671	0.007028	0.002746	1.1×10^{-3}	1.7×10^{-3}	9.0×10^{-4}	3.0×10^{-4}
Cadmium	0.004375	0.012667	0.005681	0.00222	8.7×10^{-4}	1.4×10^{-3}	7.5×10^{-4}	2.7×10^{-4}
Beryllium	0.00040	0.001265	0.00060	0.0002	7.8×10^{-5}	1.4×10^{-4}	7.8×10^{-5}	2.4×10^{-5}

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987.

TABLE 13 (CONTINUED)

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

INCREMENTAL CONCENTRATIONS FROM AREA SOURCE CATEGORIES

	ROAD VEHICLES				SOLVENT USAGES				HEATING			
POLLUTANT	B*	I*	C*	N*	B	I	C	N	B	I	C	N
Benzene	2.9	2.3	4.6	0.9	0.0	0.0	0.0	0.0	1.3×10^{-2}	1.0×10^{-2}	2.0×10^{2}	3.8×10^{3}
Perchlorehylene	0.0	0.0	0.0	0.0	2.4	2.1	4.2	0.9	0.0	0.0	0.0	0.0
Methylene chloride	0.0	0.0	0.0	0.0	1.5	1.2	2.4	0.3	0.0	0.0	0.0	0.0
Formaldehyde	0.7	0.7	1.3	0.2	0.0	0.0	0.0	0.0	0.5	0.5	0.8	0.1
Trichloroethylene	0.0	0.0	0.0	0.0	0.3	0.3	0.6	0.1	0.0	0.0	0.0	0.0
1,3-Butadiene	2.1×10^{-2}	1.8×10^{-2}	3.6×10^{-2}	6.6×10^{-3}	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ethylene chloride	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ethylene bromide	2.6×10^{-3}	2.4×10^{-3}	4.5×10^{-3}	8.6×10^{-4}	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Arsenic	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	2.4×10^{-3}	2.1×10^{-3}	3.9×10^{-3}	7.3×10^{-4}
Benzo(a)pyrene	4.5×10^{-4}	4.0×10^{-4}	7.6×10^{-4}	1.4×10^{-4}	0.0	0.0	0.0	0.0	6.0×10^{-4}	5.3×10^{-4}	1.0×10^{-3}	1.9×10^{-4}
Cadmium	1.6×10^{-4}	1.4×10^{-4}	2.5×10^{-4}	4.9×10^{-5}	0.0	0.0	0.0	0.0	6.8×10^{-4}	5.8×10^{-4}	1.1×10^{-3}	2.1×10^{-4}
Beryllium	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	7.8×10^{-5}	6.5×10^{-5}	1.1×10^{-4}	2.4×10^{-5}

*B, I, C, N represent Belle, Institute, Charleston, and Nitro respectively.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987.

TABLE 13 (CONTINUED)
KANAWHA VALLEY TOXICS SCREENING STUDY
KANAWHA VALLEY AIR QUALITY REPORT
INCREMENTAL CONCENTRATIONS FROM AREA SOURCE CATEGORIES

	GASOLINE MARKETING				WASTE OIL BURNING			
POLLUTANT	B*	I	C	N	B	I	C	N
Benzene	9.2×10^{-2}	6.1×10^{-2}	0.1	2.8×10^{-2}	8.3×10^{-5}	6.4×10^{-6}	1.2×10^{-5}	2.5×10^{-6}
Perchlorethylene	0.0	0.0	0.0	0.0	1.9×10^{-5}	2.6×10^{-5}	3.5×10^{-5}	8.4×10^{-6}
Methylene chloride	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Formaldehyde	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Trichloroethylene	0.0	0.0	0.0	0.0	1.8×10^{-5}	2.7×10^{-5}	4.9×10^{-5}	8.2×10^{-6}
1,3-Butadiene	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ethylene chloride	7.2×10^{-3}	6.3×10^{-3}	1.2×10^{-2}	2.3×10^{-3}	0.0	0.0	0.0	0.0
Ethylene bromide	7.0×10^{-4}	6.4×10^{-4}	1.2×10^{-3}	2.3×10^{-4}	0.0	0.0	0.0	0.0
Arsenic	0.0	0.0	0.0	0.0	1.6×10^{-4}	1.3×10^{-4}	2.5×10^{-4}	4.8×10^{-5}
Benzo(a)pyrene	0.0	0.0	0.0	0.0	3.9×10^{-6}	2.7×10^{-6}	4.6×10^{-6}	9.8×10^{-7}
Cadmium	0.0	0.0	0.0	0.0	4.0×10^{-5}	2.6×10^{-5}	5.5×10^{-5}	1.1×10^{-5}
Beryllium	0.0	0.0	0.0	0.0	1.6×10^{-5}	1.3×10^{-5}	2.2×10^{-5}	4.8×10^{-6}

*B, I, C, N represent Belle, Institute, Charleston, and Nitro respectively.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987.

respect to impacts on hillside receptors and the significance of interzone transport were explored through additional quantitative analysis using techniques described below.

For the purposes of the sensitivity analyses, a 100 m by 100 m industrial area source was assumed to exist in the center of each zone. A common set of upwind and downwind receptor locations was also specified.

The results of the sensitivity runs are summarized on Table 14. The following can be inferred:

Release Types: For sensitivity evaluations of release types (area source, building source, or point source) the following additional release specifications were used.

Building source dimensions: 50 m x 50 m x 30 m.

Stack parameters: 5 m (height), 0.1 m (diameter), 298 K (temperature), 0.1 m/sec (flow rate).

Within the first kilometer of plume travel, the study found that treatment of source type can significantly influence model outputs. For the examples shown here, modeling the test release as a building source was found to produce nearby concentrations much lower than those derived from the stack or area source treatments. This can be attributed to the relatively high initial horizontal and vertical dispersion produced by a building source. Based on these comparisons, modeling vent releases as building sources rather than as stack sources can reduce estimated concentrations within 200 m of the source by as much as a factor of 3 or 4 for a large building. Past one kilometer, however, the importance of the initial release conditions decreases.

Atmospheric Decay: Allyl choride, with a half-life of 4.8 hours, was modeled with the fastest decay rate of any pollutant in this study. As shown in Table 14, atmospheric decay for this chemical is, as expected, relatively unimportant for the distances and decay rates associated with pollutants under study here. The decay term produces little differences in the results, especially for the neighborhoods within a kilometer of the industrial clusters. A half-life of one hour or less would be needed to make a substantial difference in the results for the scales of interest in this study.

Dispersion Coefficients: Dispersion coefficients are used in Gaussian modeling to estimate plume dimensions along the horizontal and vertical axes as a function of downwind distance and atmospheric stability. For annual averages, vertical dispersion coefficients can be input, as they were here, on the basis of site-specific meteorological

TABLE 14
KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

SENSITIVITY RESULTS
USING ISLCT
CONCENTRATIONS IN UG/CU. M.

	DOWNVALLEY DISTANCE FROM SORUCE (M)					UPVALLEY DISTANCE FROM SOURCE (M)				
	200	600	1000	2500	5000	200	600	1000	2500	5000
ZONE: BELLE										
CLASSIFICATION										
AREA	3.1	0.8	0.4	9.2E-2	3.3E-2	1.8	0.4	0.2	4.6E-2	1.6E-2
BUILDING	1.3	0.4	0.2	0.1	2.7E-2	0.8	0.2	0.1	3.6E-2	1.3E-2
POINT	4.1	0.9	0.4	0.1	3.4E-2	2.7	0.5	0.2	4.9E-2	1.6E-2
ATMOSPHERIC DECAY (HRS)										
00.5	2.6	0.6	0.2	3.2E-2	6.1E-3	1.5	0.3	0.1	1.9E-2	3.7E-3
05.0	3.1	0.8	0.3	8.1E-2	2.6E-2	1.8	0.4	0.2	4.1E-2	1.3E-2
25.0	3.1	0.8	0.4	8.9E-2	3.1E-2	1.8	0.4	0.2	4.5E-2	1.5E-2
ZONE: CHARLESTON										
AREA	1.7	0.3	0.1	3.7E-2	1.3E-2	0.7	7.9E-2	3.0E-2	6.9E-3	2.2E-3
BUILDING	0.6	0.2	9.4E-2	2.8E-2	1.1E-2	0.3	4.9E-2	2.2E-2	5.7E-3	1.9E-3
POINT	2.0	0.4	0.2	3.8E-2	1.3E-2	0.5	8.5E-2	3.4E-2	7.2E-3	2.3E-3
ATMOSPHERIC DECAY (HRS)										
00.5	1.4	0.2	8.7E-2	1.2E-2	1.9E-3	0.6	5.6E-2	1.8E-2	2.5E-3	4.3E-4
05.0	1.7	0.3	0.1	3.3E-2	1.0E-2	0.7	7.6E-2	2.8E-2	6.1E-3	1.8E-3
25.0	1.7	0.3	0.1	3.6E-2	1.2E-2	0.7	7.8E-2	3.0E-2	6.7E-3	2.1E-3
ZONE: INSTITUTE										
CLASSIFICATION										
AREA	3.7	1.0	0.4	0.1	4.1E-2	0.7	0.1	4.8E-2	1.2E-2	4.0E-3
BUILDING	1.3	0.4	0.2	8.3E-2	3.3E-2	0.3	6.2E-2	3.2E-2	9.1E-3	3.3E-3
POINT	5.2	1.2	0.5	0.2	4.2E-2	0.7	0.1	5.3E-2	1.2E-2	4.1E-3
ATMOSPHERIC DECAY (HRS)										
00.5	3.0	0.6	0.2	3.0E-2	4.5E-3	0.6	8.8E-2	3.3E-2	5.4E-3	1.1E-3
05.0	3.6	0.9	0.4	0.1	3.1E-2	0.7	0.1	4.6E-2	1.0E-2	3.3E-3
25.0	3.7	0.9	0.4	0.1	3.9E-2	0.7	0.1	4.8E-2	1.1E-2	3.8E-3

data; horizontal dimensions of the plume are specified by assuming a uniformly mixed plume within a 22.5 sector. In this study, vertical dispersion coefficients were computed based strictly on stability data in ISCLT, and turbulent intensity data in LONGZ, for valley zones with available measured data. Technical Appendix A provides a description of the sensitivity of model output to the method of treating vertical dispersion.

Hillside Receptor Analysis: The impacts of plumes on the hillside were not considered to be a major issue for the pollutants studied. To evaluate this assumption, a limited analysis was performed in the Belle zone.

This example was selected because (1) chloroform is emitted in relatively high quantities in Belle, and (2) there are residential areas up the hillside directly across from major releases of chloroform in Belle. The LONGZ model was used. As shown in Figure 8, for these emissions associated with area sources, the impacts along the hillside are one to two orders of magnitude less than maximum concentrations along the valley floor. This is attributed to two factors: (1) flow along the valley floor is far more common than crossvalley flow, and (2) crossvalley flow is generally associated with relatively strong winds and moderate dispersion.

Interzone Transport: Valley wall reflection and flow between valley zones will act to provide higher concentrations from the box model than the ISCLT and LONGZ modeling performed for each zone independently. By using a box model approach, which assumes uniform concentration within the volume of the full extent of the valley (i.e., 45 km long by 1 km wide by 0.3 km high), one can conservatively estimate average concentrations. Table 15 shows the average concentrations based on a simple box model approach for the full 45 km^2 study area, in comparsion to population-weighted average concentrations based on ISCLT. As shown, the box model approach was consistently more conservative than ISCLT.

Refer to Technical Appendix F for a summary of the inputs to the box model estimates and a comparison of the box model treatment with the preferred approach for modeling county-wide area sources in flat terrain, the Climatological Dispersion Model (CDM). Based on Technical Appendix F, it appears that the box model approach is more conservative than a Gaussian model treatment, such as CDM.

Ambient Monitoring Programs

Three ambient air quality monitoring data sets are summarized in this section: (1) preexisting data, (2) the preliminary field program, and (3) the ORD field program.

FIGURE 8

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

HILLSIDE RECEPTOR CONCENTRATIONS FOR CHLOROFORM IN THE BELLE ZONE (ug/m^3)

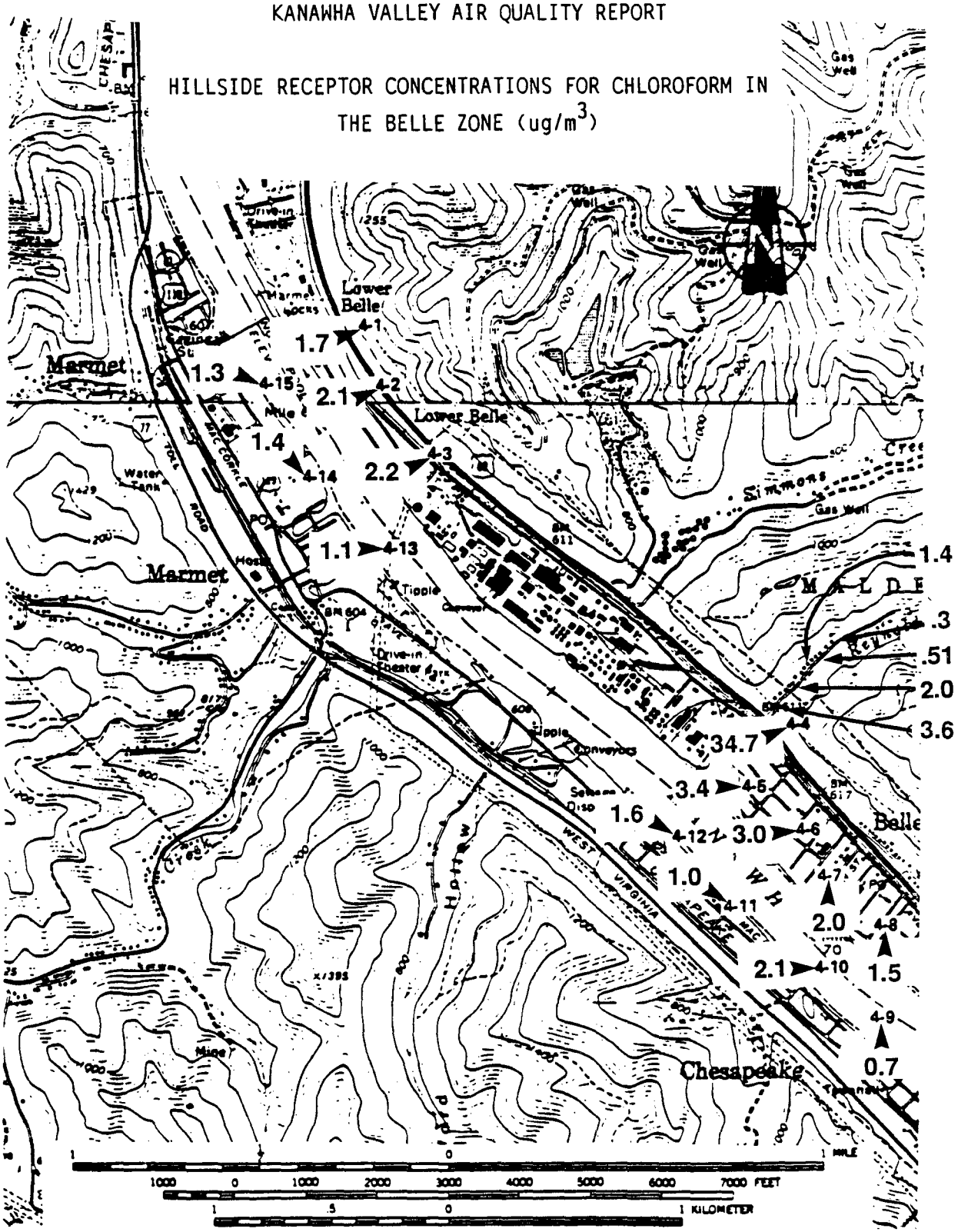


TABLE 15

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA AIR QUALITY REPORT

BOX MODEL CONCENTRATIONS FOR FULL STUDY AREA

TOTAL EMISSIONS		MODELED CONCENTRATION (ug/m^3)		Ratio:
POLLUTANT	kkg/yr	BOX MODEL	POPULATION-WEIGHTED CONCENTRATIONS BASED ON ISCLT*	Concentration (Box Model) / Concentration
Benzene	82	3.6	-	-
Perchloroethylene	71.8	3.2	-	-
Methylene chloride	145.7	6.4	-	-
Formaldehyde	50.9	2.2	-	-
Trichloroethylene	32.1	1.4	-	-
1,3-Butadiene	25.8	1.1	0.3	3.7
Ethylene chloride	0.26	0.01	-	-
Ethylene bromide	0.099	0.004	-	-
Arsenic	0.07	0.003	-	-
Benzo(a)pyrene	0.03	1.3×10^{-3}	-	-
Cadmium	0.025	1.1×10^{-3}	-	-
Beryllium	2.5×10^{-3}	1.1×10^{-4}	-	-
Carbon tetrachloride	22.9	1.0	0.4	2.5
Chloroform	134.3	5.9	1.8	3.3
Ethylene oxide	133.6	5.9	2.4	2.5
Acrylonitrile	45.7	2.0	0.8	2.5
Allyl chloride	2.0	9.0×10^{-2}	1.5×10^{-2}	6.0
Vinylidene chloride	.48	2.0×10^{-2}	1.4×10^{-2}	1.4
Vinyl chloride	5.10^{-4}	2.02×10^{-5}	7.4×10^{-6}	2.7
Propylene oxide	115.9	5.1	1.9	2.7
Average				2.8

* Comparisons are valid only for pollutants emitted primarily by industrial sources because ISCLT was not used to model county-wide area sources.

Preexisting Data: Comprehensive measured data for noncriteria pollutants were not available for the Kanawha Valley study area. Previous studies (EPA 1977, Pellizzari 1976, Pellizzari 1978) have collected limited data on selected volatile organics. Somewhat more extensive data are available for selected metals (through SAROAD). Technical Appendix G presents a summary of data collected during these programs.

Preliminary Field Program: During the week of December 9, 1985, EPA's Region III Environmental Services Division conducted an ambient air quality screening program in the four identified Kanawha Valley zones of Belle, Institute, Nitro, and Charleston/South Charleston. The screening program was short, with a limited scope and modest objectives. Details of the program are outlined in a summary report dated March 6, 1986 (Guide 1986) (refer to Technical Appendix H.1.)

This program was designed to provide real time data at selected sites within each valley zone in order to rank the importance of various pollutants and to help in selecting locations for a 45-day monitoring program, which was conducted by EPA's Office of Research and Development during March and April of 1986.

Samples were collected in 5-liter multilayer air bags and were analyzed by a Photovac Model 10A10 portable gas chromatograph (GC). Sites targeted for gas chromatograph/mass spectroscopy GC/MS confirmation were also sampled with 5-liter multilayer bags, which were returned to the EPA Central Regional Laboratory for analysis.

Unlike the later monitoring program conducted in March and April in which four fixed sites were sampled, the December field program collected samples at a large number of sites that were selected primarily to evaluate the maximum expected concentrations for the meteorological conditions present during each sampling period.

The samples that were analyzed by GC and GC/MS were not in general agreement; also, no clear pattern emerged to distinguish upwind and downwind concentrations. There is no obvious explanation for these inconsistencies. Quality control records did not indicate any significant problems in the program. Because of difficulty in interpreting these results, they were used only in a qualitative manner as input for the more detailed monitoring study that followed.

Parallel with EPA's work, the WVAPCC collected five samples during the December field program, some of which were concurrent with the Region III samples (Engle 1986). These samples were collected on Tenax and analyzed by GC/MS after being concentrated on a NuTech thermal desorption system. These samples showed much lower concentrations than the Region

III data. The results are presented in Tables 16 through 20. These results were from samples taken generally downwind of the Union Carbide South Charleston plant and the Nitro industrial complex (refer to Technical Appendix H.2 for further information).

ORD Field Program: During March and April 1986, EPA's Office of Research and Development (ORD) conducted a more detailed monitoring study at four selected sites -- two in Institute and two in Belle. The study had five objectives:

(1) To qualitatively and quantitatively confirm the presence of targeted compounds in the ambient air;

(2) To determine whether a more in-depth emissions verification of facilities and processes is needed;

(3) To assist in model selection for potential future phases of the study;

(4) To help determine whether a more detailed monitoring program is needed at a later date; and

(5) To evaluate a newly designed 24-hour cannister sampling system.

Twenty-four hour integrated air quality samples were obtained for each site in the network during this program every third day over the 45-day monitoring period (USEPA 1986c). Upvalley and downvalley monitors were placed approximately 1 km from the fencelines of the Belle and Institute complexes. The locations of these monitoring sites are shown in Figures 2 and 4. The following compounds were routinely measured and detected:

Methylene Chloride	Chloroform
Ethylene Chloride	1,1,1-Trichloroethane
Benzene	Carbon tetrachloride
Trichloroethylene	Toluene
Perchloroethylene	Chlorobenzene
o-Xylene	Vinyl chloride
Vinylidine chloride	Ethylene bromide

Refer to Technical Appendix H.3 for detailed information concerning this program.

The ORD monitoring program produced a data set that appears in general to be reasonably consistent with available emissions data and

TABLE 16

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

VOLATILE ORGANIC COMPOUNDS COLLECTED ON TENAX BY APCC

LOCATION: No. Charleston
DATE: December 6, 1985
TIME: 10:18 a.m. - 2:18 p.m.

WIND SPEED: 6 mph
WIND DIRECTION: 300° WNW

CHEMICAL COMPOUND	FLOW RATE: 200 cc/min (Vol: 48 liters)		FLOW RATE: 100 cc/min Vol: 24 liters	
	Ng*	Conc. ug/m3**	Ng*	Conc. ug/m3**
2-Chlorobutane	Trace	<0.5	Trace	<0.5
Chloroform	ND	----	ND	----
Benzene	61	1.27	36	1.5
Carbon Tetrachloride	Trace	<0.5	Trace	<0.5
Toluene	73	1.52	50	2.08
Tetrachloroethene	ND	----	ND	----
Ethyl benzene	Trace	<0.5	23	0.96
Benzaldehyde	112	2.33	77	3.21
Mesitylene	Trace	<0.5	Trace	<0.5
Dichlorobenzene	233	4.85	413	17.2

* Nanograms deteched after correction for blank.
** ug/m^3 = Ng/liter
NOTE: ND denotes Nondetectable

TABLE 17

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

VOLATILE ORGANIC COMPOUNDS COLLECTED ON TENAX BY APCC

LOCATION: Institute
DATE: December 9, 1985
TIME: 3:00 p.m. - 4:00 p.m.

WIND SPEED: 5.5 mph
WIND DIRECTION: 250°-255° WSW

CHEMICAL COMPOUND	FLOW RATE: 400 cc/min (Vol: 24 liters)		FLOW RATE: 200 cc/min Vol: 12 liters	
	Ng*	Conc. ug/m3**	Ng*	Conc. ug/m3**
2-Chlorobutane	Trace	<0.5	ND	----
Chloroform	ND	----	ND	----
Benzene	45	1.87	19	1.58
Carbon tetrachloride	ND	----	ND	----
Toluene	161	6.70	43	3.58
Tetrachloroethene	ND	----	ND	----
Ethyl benzene	22	0.92	Trace	<0.5
Benzaldehyde	121	5.04	60	5.00
Mesitylene	Trace	0.50	Trace	<0.5
Dichlorobenzene	37	1.54	ND	----

* Nanograms deteched after correction for blank.

** ug/m^3 = Ng/liter

NOTE: ND denotes Nondetectable

TABLE 18

KANAWHA VALLEY TOXICS SCREENING STUDY
KANAWHA VALLEY AIR QUALITY REPORT

VOLATILE ORGANIC COMPOUNDS COLLECTED ON TENAX BY APCC

LOCATION: Belle
DATE: December 10, 1985
TIME: 10:00 p.m. - 11:00 p.m.

WIND SPEED: 4.5 mph
WIND DIRECTION: 155°-165° WSW

CHEMICAL COMPOUND	FLOW RATE: 400 cc/min (Vol: 24 liters)		FLOW RATE: 200 cc/min Vol: 12 liters	
	Ng*	Conc. ug/m3**	Ng*	Conc. ug/m3**
2-Chlorobutane	13	0.54	32	2.66
Chloroform	36	0.50	Trace	<0.5
Benzene	71	2.96	33	2.75
Carbon tetrachloride	33	1.37	Trace	<0.5
Toluene	91	3.79	30	2.50
Tetrachloroethene	ND	----	ND	----
Ethyl benzene	ND	----	NDe	----
Benzaldehyde	61	2.54	62	5.16
Mesitylene	Trace	<0.5	Trace	0.54
Dichlorobenzene	ND	----	ND	----

* Nanograms deteched after correction for blank.
** ug/m^3 = Ng/liter
NOTE: ND denotes Nondetectable

TABLE 19

KANAWHA VALLEY TOXICS SCREENING STUDY
KANAWHA VALLEY AIR QUALITY REPORT

VOLATILE ORGANIC COMPOUNDS COLLECTED ON TENAX BY APCC

LOCATION: Nitro+
DATE: December 11, 1985
TIME: 11:40 a.m. - 12:40 p.m.

WIND SPEED: 2-4.8 mph
WIND DIRECTION: 210°-220° WSW

CHEMICAL COMPOUND	FLOW RATE: 400 cc/min (Vol: 24 liters)		FLOW RATE: 200 cc/min Vol: 12 liters	
	Ng*	Conc. ug/m3**	Ng*	Conc. ug/m3**
2-Chlorobutane	303	12.6	98	8.16
Chloroform	206	8.58	113	9.42
Benzene	206	8.58	94	7.83
Carbon tetrachloride	Trace	<0.5	24	2.00
Toluene	1044	43.5	309	25.7
Tetrachloroethene	1111	46.3	613	51.0
Ethyl benzene	422	17.6	75	6.25
Benzaldehyde	127	5.29	71	5.92
Mesitylene	120	5.00	30	2.54
Dichlorobenzene	161	6.70	101	8.42

* Nanograms deteched after correction for blank.
** ug/m^3 = Ng/liter
+ Gress Equip. Co., Inc., Hubb Industrial Park
NOTE: ND denotes Nondetectable

TABLE 20

KANAWHA VALLEY TOXICS SCREENING STUDY
KANAWHA VALLEY AIR QUALITY REPORT

VOLATILE ORGANIC COMPOUNDS COLLECTED ON TENAX BY APCC

LOCATION: Nitro+
DATE: December 11, 1985
TIME: 11:40 a.m. - 12:40 p.m.

WIND SPEED: 2-4.8 mph
WIND DIRECTION: 210°-220° WSW

CHEMICAL COMPOUND	FLOW RATE: 400 cc/min (Vol: 24 liters)		FLOW RATE: 200 cc/min Vol: 12 liters	
	Ng*	Conc. ug/m3**	Ng*	Conc. ug/m3**
2-Chlorobutane	63	3.15	25	2.5
Chloroform	87	4.35	59	5.9
Benzene	57	2.85	46	4.6
Carbon tetrachloride	32	1.60	19	1.9
Toluene	87	4.35	41	4.1
Tetrachloroethene	ND	----	ND	----
Ethyl benzene	ND	----	ND	----
Benzaldehyde	28	1.40	45	4.5
Mesitylene	Trace	<0.5	Trace	<0.5
Dichlorobenzene	ND	----	ND	----

* Nanograms deteched after correction for blank.
** ug/m^3 = Ng/liter
+ State Hygenic Lab, 167-11th Ave., S. Chas. WV
NOTE: ND denotes Nondetectable

meteorological conditions for the four pollutants that were compared with meteorological data for the sampling days.[8] The measured data generally show the highest concentrations during periods with drainage flows;[9] some days with persistent flow oriented along the valley also showed relatively high concentrations. During days with persistent flow that resulted in monitoring sites being upwind of the industrial complexes, concentrations were generally low, as would be expected.

For the pollutants present in the highest concentrations, methylene chloride, chloroform, toluene, carbon tetrachloride and "unknown compound," Table 21 summarizes the measured data by site and provides an interpretation of the meteorological data. The results shown in Table 21 indicate that the average concentrations at Site 4 were found to be higher than those at Site 3. Site 3 is approximately 0.45 km downvalley of the Belle industrial complex and should therefore be more affected by stable drainage flows. Site 4 is located approximately 0.8 km upvalley of the industrial complex. The higher concentrations found at Site 4 are consistent with the available emissions data, which show emissions of chloroform and methylene chloride to be towards the upvalley border of the complex.

Conversely, the toluene and chloroform concentrations at Site 2, which is located approximately 2.5 km downvalley of the edge of the Institute complex, were generally higher than those at Site 1, which is approximately 0.9 km upvalley. Table 22 presents a summary of the means and maximum/minimum values across all sites.

In interpreting these data, it is important to consider the way in which the monitoring sites were selected for this 45-day program. Although an important goal of this study is to characterize pollutant concentrations in the neighborhoods closest to the plants, it was undesirable to locate the monitoring sites directly in these neighborhoods. Since the facility complexes are very large, a station near a fenceline of a plant could easily fail to intercept plumes emitted by sources located cross-wind of the monitor, especially since the samples were collected during a relatively short period of time, i.e., 15 days over a 45-day period. By locating the stations an average of one

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8 Drainage flow is a condition that can occur in the Kanawha Valley during the night if the sky is clear and regional winds are light. Under these conditions, the air in contact with the valley walls and floor cools and sinks, flowing down the valley. Within this flow, air near the ground surface is stable; pollutants emitted within it are dispersed slowly.

9 Other pollutants are emitted in substantial quantities in the study area, including 1,3-butadiene and acrylonitrile. These pollutants were not included in the monitoring program, however.

Table 21

KANAWHA VALLEY TOXICS SCREENING STUDY
KANAWNA VALLEY AIR QUALITY REPORT

DETAILED SUMMARY OF MEASURED DATA SET
(MARCH 4, 1986-APRIL 11, 1986)

SAMPLING DAY	CONCENTRATIONS (PPB) #3 METHYLENE CHLORIDE				#4 CHOROFORM				#10 TOLUENE				"UNKNOWN COMPOUND" **				GENERAL DESCRIPTION OF METEREOLOGICAL CONDITIONS	DISPERSION CONDITIONS BELLE 0100-0500	BELLE 1300-1700	INSTITUTE 0100-0500	INSTITUTE 1300-1700
SITE →	1	2	3	4	1	2	3	4	1	2	3	4	1	2	3	4		σθ − σφ	σθ − σφ	σθ − σφ	σθ − σφ
3/4/86	0.50	0.24	0.54	3.08	0.11	NF	0.06	0.55	0.72	0.86	0.95	1.10		9.30			REGIONAL FLOW GENERALLY OUT OF 240° - 270°	48.0, 8.9	56.0, 6.9	10.8, 4.8	33.4, 8.9
3/6/86	0.61	0.25	0.24	4.10	0.12	0.01	0.01	1.66	0.57	0.50	0.58	1.29		–			REGIONAL FLOW GENERALLY 250°- 270°	16.0, 3.6	37.6, 10.9	24.8, 5.5	11.9, 7.2
3/9/86	0.33	0.83	0.25	0.57	0.05	2.49	4.55	0.08	0.57	1.76	3.36	1.18		9.06			FLOW ACT OF 230°- 260° DURING AFTERNOON, DRAINAGE FLOW 2000-0900	8.9, 4.4	51.2, 7.1	14.5, 3.4	18.6, 7.0
3/12/86	0.88	1.78	25.04	0.40	0.60	3.61	4.89	0.06	1.77	5.45	4.01	0.97	6.80	13.0			REGIONAL FLOW GENERALLY OUT OF 120°- 150°	61.9, 5.3	10.2, 3.6	18.2, 7.0	20.9, 5.5
3/16/86	0.72	0.29	0.60	2.85	0.05	0.03	0.10	1.36	2.44	0.58	1.26	1.47		–			REGIONAL FLOW GENERALLY 340° - 030°	*, *	42.0, *	31.0, 4.5	19.9, 7.3
3/18/86	0.60	0.97	11.96	0.51	0.14	2.30	2.82	0.08	2.71	5.11	2.03	0.64		5.90	9.76	4.90	REGIONAL FLOW GENERALLY 140°- 200° UNTIL 2200, THEN 240°- 330°	9.8, 6.2	23.9, 8.9	10.3, 3.0	23.5, 4.0
3/21/86	0.59	0.27	0.60	1.25	0.46	0.10	0.18	1.34	1.97	0.80	1.34	2.03	3.80	4.70			FLOW OUT OF 270°- 330° IN AFTERNOON WEAK DRAINAGE FLOW 2100-0900, LOW WINDS	11.1, 3.7	39.6, 11.9	18.7, 4.1	40.4, 10.4
3/24/86	1.13	1.42	6.09	1.14	0.41	5.27	3.27	0.53	2.91	14.16	2.38	2.03	6.60	9.80			270°- 330° UNTIL 2000, DRAINAGE UNTIL 1000	30.8, 6.1	37.6, 16.1	31.4, 5.6	29.3, 13.2
3/27/86	0.89	1.21	2.33	9.92	1.03	2.71	0.52	2.56	4.88	4.59	1.98	1.55	4.80	6.00			300°- 360° UNTIL 2100, DRAINAGE UNTIL 0900	26.8, 6.3	21.4, 8.9	34.5, 8.9	25.6, 8.9
3/30/86	1.72	1.46	8.74	3.31	2.89	2.14	3.74	0.96	7.38	7.98	4.70	3.26	11.1	11.4			GENERALLY 250°- 270° UNTIL 2000, DRAINAGE UNTIL 1000	7.1, 4.3	56.4, 15.5	6.5, 2.6	24.6, 11.3
4/2/86	1.02	0.60	4.82	3.52	0.85	1.56	1.95	4.32	2.80	2.96	2.33	2.76	6.10	5.30			300°- 360° UNTIL 2400, DRAINAGE UNTIL 0900	54.0, 3.9	12.4, 9.5	49.0, 6.2	32.2, 13.6
4/4/86	1.43	2.60	4.61	3.21	0.76	5.19	1.77	1.29	3.32	9.48	2.45	2.50	–				270°- 300° UNTIL 2000, DRAINAGE UNTIL 0900	24.3, 2.3	41.3, 11.0	17.4, 4.8	21.5, 10.6
4/7/86	1.14	0.59	3.35	5.61	0.63	0.38	2.47	2.93	4.27	2.23	1.36	1.08	7.20	7.40		11.5	290°- 300° UNTIL 2000, DRAINAGE UNTIL 0800	12.4, 4.1	33.6, 12.4	47.7, 2.3	21.0, 9.9
4/9/86	1.29	0.20	0.24	2.95	0.98	0.01	0.02	0.75	3.96	0.39	0.38	0.54	–	–	–	–	REGIONAL FLOW 250°- 300°	14.9, 6.7	23.5, 9.8	13.1, 8.5	27.0, 6.7
4/11/86	1.41	0.86	4.77	10.61	2.03	1.13	1.32	6.16	8.60	3.01	1.35	1.30	–	6.90	12.2	11.0	270°- 290° UNTIL DRAINAGE FLOW DEVELOPS AT 2200, PERSISTENT UNTIL 0900	49.1, 14.4	36.5, 11.8	15.2, 9.3	15.3, 7.2
MEAN	0.95	0.90	5.99	3.54	0.74	1.80	1.84	1.64	3.25	3.99	2.03	1.63	4.7	6.3	7.4	6.9					

* Data Unavailable
** Same retention time and peak shape as ethylene oxide, however, a positive identification was not made. Incomplete coverage is available for this compound.

PAGE 1 of 2

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

Table 21 (Continued)

KANAWHA VALLEY TOXICS SCREENING STUDY
KANAWHA VALLEY AIR QUALITY REPORT

SAMPLING DAY	CONCENTRATIONS (PPB) #8 CARBON TETRACHLORIDE				GENERAL DESCRIPTION OF METEREOLOGICAL CONDITIONS	DISPERSION CONDITIOI			
						BELLE		INSTITUTE	
						0100-0500	1300-1700	0100-0500	1300-1700
SITE →	1	2	3	4		σθ – σφ	σθ – σφ	σθ – σφ	σθ – σφ
3/4/86	0.09	0.11	0.11	0.16	REGIONAL FLOW GENERALLY OUT OF 240 ° - 270 °	48.0, 8.9	56.0, 6.9	10.8, 4.8	33.4, 8.9
3/6/86	0.14	0.11	0.11	0.28	REGIONAL FLOW GENERALLY 250 ° - 270 °	16.0, 3.6	37.6, 10.9	24.8, 5.5	11.9, 7.2
3/9/86	1.00	0.13	0.88	0.13	FLOW ACT OF 230 ° - 260 ° DURING AFTERNOON, DRAINAGE FLOW 2000-0900	8.9, 4.4	51.2, 7.1	14.5, 3.4	18.6, 7.0
3/12/86	0.15	0.17	1.70	0.09	REGIONAL FLOW GENERALLY OUT OF 120 ° - 150 °	61.9, 5.3	10.2, 3.6	18.2, 7.0	20.9, 5.5
3/16/86	0.11	0.10	0.11	0.30	REGIONAL FLOW GENERALLY 340 ° - 030 °	*, *	42.0, *	31.0, 4.5	19.9, 7.3
3/18/86	0.13	0.15	1.05	0.11	REGIONAL FLOW GENERALLY 140 ° - 200 ° UNTIL 2200, THEN 240 ° - 330 °	9.8, 6.2	23.9, 8.9	10.3, 3.0	23.5, 4.0
3/21/86	0.12	0.13	0.11	0.22	FLOW OUT OF 270 ° - 330 ° IN AFTERNOON WEAK DRAINAGE FLOW 2100-0900, LOW WINDS	11.1, 3.7	39.6, 11.9	18.7, 4.1	40.4, 10.4
3/24/86	0.16	0.21	0.45	0.17	270 ° - 330 ° UNTIL 2000, DRAINAGE UNTIL 1000	30.8, 6.1	37.6, 16.1	31.4, 5.6	29.3, 13.2
3/27/86	0.13	0.17	0.26	0.53	300 ° - 360 ° UNTIL 2100, DRAINAGE UNTIL 0900	26.8, 6.3	21.4, 8.9	34.5, 8.9	25.6, 8.9
3/30/86	0.62	0.39	1.04	0.42	GENERALLY 250 ° - 270 ° UNTIL 2000, DRAINAGE UNTIL 1000	7.1, 4.3	56.4, 15.5	6.5, 2.6	24.6, 11.3
4/2/86	0.16	0.13	0.39	0.50	300 ° - 360 ° UNTIL 2400, DRAINAGE UNTIL 0900	54.0, 3.9	12.4, 9.5	49.0, 6.2	32.2, 13.6
4/4/86	0.18	0.20	0.43	0.23	270 ° - 300 ° UNTIL 2000, DRAINAGE UNTIL 0900	24.3, 2.3	41.3, 11.0	17.4, 4.8	21.5, 10.6
4/7/86	0.11	0.11	0.76	0.39	290 ° - 300 ° UNTIL 2000, DRAINAGE UNTIL 0800	12.4, 4.1	33.6, 12.4	47.7, 2.3	21.0, 9.9
4/9/86	0.11	0.11	0.11	0.22	REGIONAL FLOW 250 ° - 300 °	14.9, 6.7	23.5, 9.8	13.1, 8.5	27.0, 6.7
4/11/86	0.15	0.13	0.54	1.44	270 ° - 290 ° UNTIL DRAINAGE FLOW DEVELOPS AT 2200, PERSISTENT UNTIL 0900	49.1, 14.4	36.5, 11.8	15.2, 9.3	15.3, 7.2
MEAN	0.22	0.16	0.54	0.35					

*Data Unavailable
**Same retention time and peak shape as ethylene oxide; however, a positive identification was not made. Coverage is incomplete for this compound.

Source: Regulatory Integration Office, Office of Policy Analysis, EPA, 1987

TABLE 22

KANAWHA VALLEY TOXICS SCREENING STUDY
KANAWHA VALLEY AIR QUALITY REPORT

AVERAGE AND RANGE OF CONCENTRATIONS FOR TARGET COMPOUNDS WITH ORD MONITORING PROGRAM
(March 4, 1986-April 11, 1986)

Average and range (PPB) of target compounds

compound	mean	minimum	maximum
dichloromethane	2.82	0.0	24.71
trichloromethane	1.58	0.0	6.11
1,2-dichloroethane	0.03	0.0	0.20
1,1,1-trichloroethane	0.34	0.2	0.72
benzene	0.89	0.4	1.64
tetrachloromethane	0.30	0.1	1.70
trichloroethene	0.02	0.0	0.13
toluene	2.99	0.5	16.54
tetrachloroethene	0.07	0.0	0.28
chlorobenzene	0.04	0.0	0.32
o-xylene	0.34	0.0	0.84

Source: Report on the Air Monitoring in the Kanawha Valley, West Virginia, Fitz-Simons, Lumpkin, McClenny.

kilometer from the plants, the natural channeling of air flow in the valley tended to mix the plumes and ensure a more reliable identification of the full range of pollutants being emitted. Unfortunately, measured data collected at these sites do not provide direct indications of concentrations at the locations likely to experience maximum concentrations. The one-kilometer distance was chosen as the best practical tradeoff between (1) assessing plant-level emissions data via comparisons of modeled and measured data, and (2) measuring concentrations in neighborhoods most affected by these releases.

Quantitative Comparison of Measured and Modeled Data: One way of comparing the general magnitude of measured and modeled concentrations is to compare the limited measured data with six-month average modeled values for the model receptors closest to the monitoring sites. Table 23 compares the six-month average modeled values with the limited measured data. The closest receptors to the monitoring sites were averaged to provide a point of comparison. Clearly, in order to definitively compare the results, the specific sites and monitoring periods would need to be modeled based on daily-specific emissions data. These rough comparisons should not be interpreted as a model performance evaluation.

Modeled and measured values for chloroform, carbon tetrachloride, and methylene chloride were found to be in reasonable agreement based on these rough comparisons. These results are based on ISCLT. Only industrial sources were modeled in these comparisons. If county-wide concentrations and general background values were also included, the modeled concentrations would likely increase by up to a few micrograms per cubic meters for chloroform, methylene chloride, and carbon tetrachloride.

The comparisons for the modeled ethylene oxide data and the unknown measured compound (with the same retention time as ethylene oxide) showed poor agreement. While the measured and modeled values for the "unknown compound," which was hypothesized to be ethylene oxide, at Sites 1 and 2 were found to be in reasonably close agreement, still higher values were found for Sites 3 and 4, where the inventory shows no emissions of this compound. Based on this extremely limited and uncertain data set, it appears that the unknown compound may not be ethylene oxide, or that possibly there is an interfering compound in the Belle zone. Alternatively, if the measured data reasonably represent the magnitude of ethylene oxide concentrations, the emissions data in the Belle zone are not including these releases.

The preceding comparisons provide a rough indication of how well the modeling approach represents transport and dispersion within the study area, and, for each pollutant, how reasonably the emissions data represent actual concentrations via dispersion modeling. Dispersion models and measured data are typically not used to assess emissions. For

Table 23

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

COMPARISON OF MEASURED (3/86 - 4/86)
AND MODELED (12/85 - 6/86)
DATA BASED ON ORD MONITORING PROGRAM

	Concentration[10] (ug/m3)							
	Institute				Belle			
	Site 1 (Upvalley)		Site 2 (Downvalley)		Site 3 (Downvalley)		Site 4 (Upvalley)	
Pollutant	mod.	mon.	mod.	mon.	mod.	mon.	mod.	mon.
Chloroform	1.9	3.6	4.5	8.7	8.1	8.9	11.5	8.0
Methylene Chloride	0.6	3.3	1.3	3.1	10.8	20.8	14.6	12.3
"Ethylene Oxide" (unknown comp.)[11]	2.4	8.4	12.8	11.3	0	13.3	0	12.4
Carbon Tet.	0	1.4	0	1.0	2.6	3.4	3.3	2.2

10 Based on ISCLT for the closest receptors: Site 1 (2-7, 2-8, 2-9); Site 2 (8A.1 - 8A.4); Site 3 (4-2); and Site 4 (4-6, 4-7).

11 Ethylene oxide (unknown compound) averages are based on very limited data: Site 1 (n=10); Site 2 (n=14); Site 3 (n=3); and Site 4 (n=4).

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987.

criteria pollutants, it is often assumed that the emissions inventory is accurate; comparisons of measured and modeled data are made to determine the validity of the modeling analysis. For this study, where annual average impacts at the residential areas near major facilities are a key objective, model performance in terms of representing transport and dispersion is expected to be within a factor of two on a seasonal or annual basis. Substantially greater uncertainty exists in the emissions data, especially for pollutants involving a large percentage of fugitive releases. Evaluating emissions is therefore of prime importance for this study.

The objectives of the ORD field program were generally achieved. The following repeats the objectives and provides brief comments concerning the achievement of each objective:

(1) To qualitatively and quantitatively confirm the presence of targeted compounds in the ambient air. In general, this objective was met except for uncertainty regarding the identification of ethylene oxide.

(2) To determine if a more in-depth emissions verification of facilities and processes is needed. To date, only rough comparisons have been made. Tentative results indicate, for example, that modeled and measured results for chloroform, carbon tetrachloride, and methylene chloride are reasonably consistent for nearby receptors. However, measured data for an unknown compound with same retention time as ethylene oxide did not match the modeled values very well. If a more complete data set were compiled, including process status data for sampling days, a more quantitative comparison could be made.

(3) To assist in model selection for potential future phases of the study. Quantitative analysis of measured data in comparison to alternative model formulations has not yet been performed.

(4) To help determine if a more detailed monitoring program is needed at a later date. The results suggest that monitoring methods applicable for the ambient air are needed for ethylene oxide, 1,3-butadiene, and propylene oxide. In addition, follow-up long-term monitoring in the immediate vicinity of major facilities would be helpful in more definitively estimating ambient exposures.

(5) To evaluate a newly designed 24-hour cannister sampling system. There appears to be a reasonable comparison between the results of Tenax and the cannister sampling system.

V. RISK ASSESSMENT

This section presents the results of the cancer risk assessment screening methodology of the air quality analysis of the Kanawha Valley Toxics Study. This methodology is based on the exposure modeling analysis of a limited set of pollutants performed in support of this study. This modeling analysis is documented in detail in the exposure assessment (Section IV) of this report. For this risk assessment analysis, we estimated upper-bound, lifetime, incremental carcinogenic risks by combining estimates of individual and average population exposures to a limited set of pollutants, based on our modeling analysis, with estimates of the cancer potency of pollutants. We designed our analysis to allow comparison of potential risks among this limited set of pollutants and sources within this analysis. We can also compare these potential risks from airborne pollutants to those risks posed by pollutants within other media.

The estimates of upper-bound cancer risk are primarily based on three factors: (1) modeled ambient air concentrations (annual averages); (2) assumptions regarding the relation of actual exposure to the modeled concentrations; and (3) cancer potency unit risk estimates of the compounds studied. As noted in the exposure assessment, we estimated two types of exposures for our risk assessment. We first evaluated the exposure of selected neighborhoods immediately surrounding the major facilities within the study area. We had pre-selected these neighborhoods for analysis and believed them to be the most affected by emissions from these facilities. Second, we performed dispersion modeling to estimate the exposure to the average individual from selected pollutants within valley zones.

Risk Assessment Methodology

This study uses a risk assessment screening methodology to evaluate and compare, in a very limited fashion, the potential health risks from exposure to a limited set of potentially toxic pollutants. The purpose of this methodology is to permit a comparison of risks across pollutants, sources, and pathways and to provide a general sense of the risk a substance may present. <u>This methodology does not allow one to make a definitive statement concerning the absolute risk posed by a particular pollutant, source, or exposure pathway</u>.

This study is not designed to draw conclusions regarding the observed cancer incidence in the community and environmental exposures. To draw such conclusions, one would need to conduct an epidemological study and control for factors such as smoking and socioeconomic class.

Risk to an individual is defined as the increased probability that an individual exposed to one or more pollutants will experience a particular adverse health effect, in this case cancer, during his or her lifetime. Several indices of carcinogenic risk are used in these analyses, including increased estimated lifetime risk to an individual and increased incidence of cancers in the exposed population, i.e., the upper-bound projected increased cases across the entire population within the study area.

This risk screening methodology involves both a qualitative and quantitative assessment of the potential carcinogenicity of the selected pollutants. As a screening study, this analysis employs both types of assessments. EPA's Carcinogen Assessment Group (CAG) reviews the evidence of carcinogenicity for selected pollutants and qualitatively categorizes the chemical based on the weight of scientific evidence. It also classifies pollutants as human carcinogens (Group A), probable human carcinogens (Group B), possible human carcinogens (Group C), not classifiable because of inadequate evidence (Group D), and not carcinogenic to humans (Group E).

For those chemicals in groups A, B, and C, CAG provides quantitative, conservative upper-bound estimates of carcinogenic unit risk factors. A unit risk factor allows the calculation of the estimated increased lifetime individual risk posed by exposure to a chemical, given standard exposure assumptions. To calculate individual risk, a chemical's unit risk factor is multiplied by the estimated concentration of the pollutant to which an individual is exposed.

The concentration used in this calculation of risk is the predicted annual average concentration of the chemical in the ambient air. For this analysis, we used the predicted model concentration to calculate individual risk. The unit risk factor that provides a measure of the carcinogenic potency assumes that an average person weighs 70 kg and breathes 20 m^3 of air each day for 70 years.

SAMPLE CALCULATION
POSSIBLE RISK OF CANCER ASSOCIATED WITH INHALATION OF CHLOROFORM IN AMBIENT AIR

Estimated Average Individual Lifetime Risk	=	Unit Risk Factor x Concentration
Cancer Unit Risk Factor for the Inhalation of Chloroform	=	2.3×10^{-5} $(ug/m^3)^{-1}$
Concentration	=	For this example assume 1 ug/m^3 of chloroform in the ambient air
Estimated Average Individual Lifetime Risk	=	2.3×10^{-5} $(ug/m^3)^{-1} \times 1$ (ug/m^3)
	=	2.3×10^{-5}

This estimated average individual risk, based on an upper-bound unit risk factor, indicates that the increased risk of cancer is not likely to exceed a two in a one hundred thousand probability over a 70-year lifetime of constant exposure to this concentration of chloroform. Given the upper-bound nature of the unit risk factor, the actual risk could be considerably lower. Table 24 presents the unit risk values for the selected pollutants for the air analysis of the Kanawha Valley Toxics Study.

Incidence, another index of risk, is the probable number of cancer cases expected over a 70-year period when a population is exposed to the average individual exposure concentration. Incidence is calculated by multiplying risk to the average exposed individual by the number of people exposed and can be annualized by dividing by 70 to obtain cases per year.

Risk Assessment Limitations

There are several limitations to the risk assessment methodology. It is important to emphasize these points to allow proper interpretation of the estimate of risk presented in this report.

- The estimate of individual cancer risk and aggregate cancer incidence from exposure to toxics should not be interpreted as precise or absolute estimates of present or future health effects. The simplifying assumptions and uncertainties in both

TABLE 24

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

UPPER-BOUND CANCER UNIT RISK VALUES FOR SELECTED TOXICS IN THE AMBIENT AIR

POLLUTANT	INHALATION POTENCY VALUE $(ug/m^3)^{-1}$	GROUPING BASED ON EPA CRITERIA
1,3-Butadiene	2.8×10^{-4}	B 2
Carbon Tetrachloride	1.5×10^{-5}	B 2
Chloroform	2.3×10^{-5}	B 2
Ethylene oxide	1×10^{-4}	B 1
Perchlorethylene	4.8×10^{-7}	B 2
Acrylonitrile	6.8×10^{-5}	B 1
Methylene chloride	4.1×10^{-6}	B 2
Arsenic	4.3×10^{-3}	A
Benzo(a)pyrene	3.3×10^{-3}	B 2
Benzene	8×10^{-6}	A
Beryllium	2.4×10^{-3}	B 2
Cadmium	1.8×10^{-3}	B 1
Ethylene bromide	2.17×10^{-4}	B 2
Ethylene chloride	2.6×10^{-5}	B 2
Trichloroethylene	1.3×10^{-6}	B 2
Allyl chloride	2.2×10^{-8}	B 2
Vinylidene chloride	5.0×10^{-5}	C
Vinyl chloride	2.6×10^{-6}	A

SOURCE: EPA Carcinogen Assessment Group, 1986. Potency values are plausible upper-bound estimates of human health effects.

the toxicology and exposure components are simply too great to justify a high level of confidence in the precision of the results. The estimates of cancer risks are approximations of the upper-bound potential risks to human health and are designed to permit comparison across different sources, pollutants, and exposure pathways.

- The cancer unit risk factors used in this study are consistently conservative in the direction of overestimating risk for the particular pollutants and exposure pathways we have assessed.

- We may understate risks to the extent that we have not considered all pollutants and sources and have not considered workplace or consumer exposure. Synergistic or antagonistic effects of pollutants are not considered, and there may be carcinogens that have not received a potency value from CAG. Also, the scope of the study does not include a consideration of non-cancer health effects.

- The results from this risk assessment are based primarily on existing knowledge. As new scientific data and techniques become available, these results may change.

VI. RESULTS

In the following sections, we present the results of our risk assessment. We first discuss the results of the individual risks at the neighborhood sites identified within the vicinity of the facilities. These results are based on modeled concentrations presented in Tables 5 - 8 and the unit risk factors shown in Table 24. For this analysis, we identify those neighborhoods of relatively higher risks. For these sites, we present the primary pollutants and facilities, in terms of risk and percentage contribution to total risk, contributing to the estimated risks within these neighborhoods. For the neighborhood with the highest relative risk, we have determined the contribution of fugitive emissions of the primary pollutant contributing to the total risk. Technical Appendix J provides the risk and percentage contribution of fugitive emissions for all the sites. The risk results presented for the neighborhoods only reflect potential risk from point source facilities. Risks from county-wide area sources are not included.

Second, we present the risk posed to the average exposed individual from the selected point source pollutants. These results are based on modeled concentrations presented in Tables 9 - 12. We then identify the percentage contribution of pollutants and facilities to the risk to the average exposed individual. We also calculate incidence by pollutant and facility within the zone.

Finally, we present risks posed by county-wide area sources predicted by the box model. These results are based on model concentrations presented in Table 13.

Belle Zone

We modeled air emissions from two facilities, Dupont and Occidental, within the Belle zone.

These facilities emitted three of the studied pollutants: carbon tetrachloride, methylene chloride and chloroform. Formaldehyde is also emitted. The risk estimates for formaldehyde are presented in Technical Appendix I. For these pollutants and these facilities, we present the results for our risk screening analysis, first for the exposures within selected neighborhoods and then for the exposure to the average individual within the Belle zone.

Risks in Neighborhood Sites with Suspected Highest Exposures from Point Sources: Table 25 presents the estimated upper-bound lifetime incremental risk posed by each pollutant and the total estimated risk from these pollutants at neighborhoods we selected as likely to have relatively high exposures from point sources. The total risk is the sum of the risk posed by each pollutant (we assume the cancer risks are additive). Figure 9 maps the estimated risks for the neighborhoods surrounding the facilities. For these sites, site 4-4 has the highest estimated risk from these pollutants. The total estimated lifetime incremental risk is 3×10^{-3} using upper-bound potency values. This is a three in a thousand probability of contracting cancer during a 70-year lifetime exposure from these concentrations of chemicals. There are approximately 600 people living within this vicinity (1980 Census). According to Table 25, chloroform contributes almost three-fourths of the estimated risk. Carbon tetrachloride and methylene chloride contribute about one-eighth and one-sixth of the total risk, respectively, at this neighborhood.

The other surrounding neighborhood concentrations present upper-bound lifetime risks between 9×10^{-5} to 5×10^{-4} individual risk from these pollutants. This risk runs from a nine in a hundred thousand probability to a six in a ten thousand probability of contracting cancer during a 70-year lifetime exposure to these chemicals. At all sites, chloroform is the most significant contributor to the total risk.

Table 26 presents the estimated lifetime individual risk by pollutant contributed individually by the Dupont and Occidental facilities. The total column for each facility is the sum of the risk posed by each pollutant emitted by the facility. Occidental contributes the overwhelming majority of the risk from these selected pollutants.

TABLE 25

KANAWHA VALLEY TOXICS SCREENING STUDY
KANAWHA VALLEY AIR QUALITY REPORT
PRELIMINARY RISK SCREENING RESULTS INTENDED FOR POLICY DEVELOPMENT

UPPER-BOUND ESTIMATES OF LIFETIME CANCER RISKS IN NEIGHBORHOODS SURROUNDING FACILITIES FROM POINT SOURCE POLLUTANTS

BELLE

COMPOUND

	CARBON TETRACHLO	CHLOROFORM	METHYLENE CHLORI	TOTAL
4- 1 (ISCLT)	3E-05	2E-04	4E-05	2E-04
4- 2 (ISCLT)	4E-05	2E-04	4E-05	3E-04
4- 3 (ISCLT)	5E-05	2E-04	5E-05	3E-04
4- 4 (ISCLT)	4E-04	.002297	5E-04	.003204
4- 5 (ISCLT)	6E-05	3E-04	8E-05	5E-04
4- 6 (ISCLT)	6E-05	3E-04	7E-05	4E-04
4- 7 (ISCLT)	4E-05	2E-04	5E-05	3E-04
4- 8 (ISCLT)	3E-05	2E-04	3E-05	2E-04
4- 9 (ISCLT)	1E-05	7E-05	2E-05	1E-04
4-10 (ISCLT)	2E-05	10E-05	2E-05	1E-04
4-11 (ISCLT)	2E-05	1E-04	3E-05	2E-04
4-12 (ISCLT)	1E-05	6E-05	1E-05	9E-05
4-13 (ISCLT)	2E-05	1E-04	3E-05	2E-04
4-14 (ISCLT)	3E-05	1E-04	3E-05	2E-04
4-15 (ISCLT)	2E-05	1E-04	3E-05	2E-04

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

FIGURE 9

Kanawha Valley Toxics Screening Study
Kanawha Valley Air Quality Report
Preliminary Risk Screening Results Intended for Policy Development

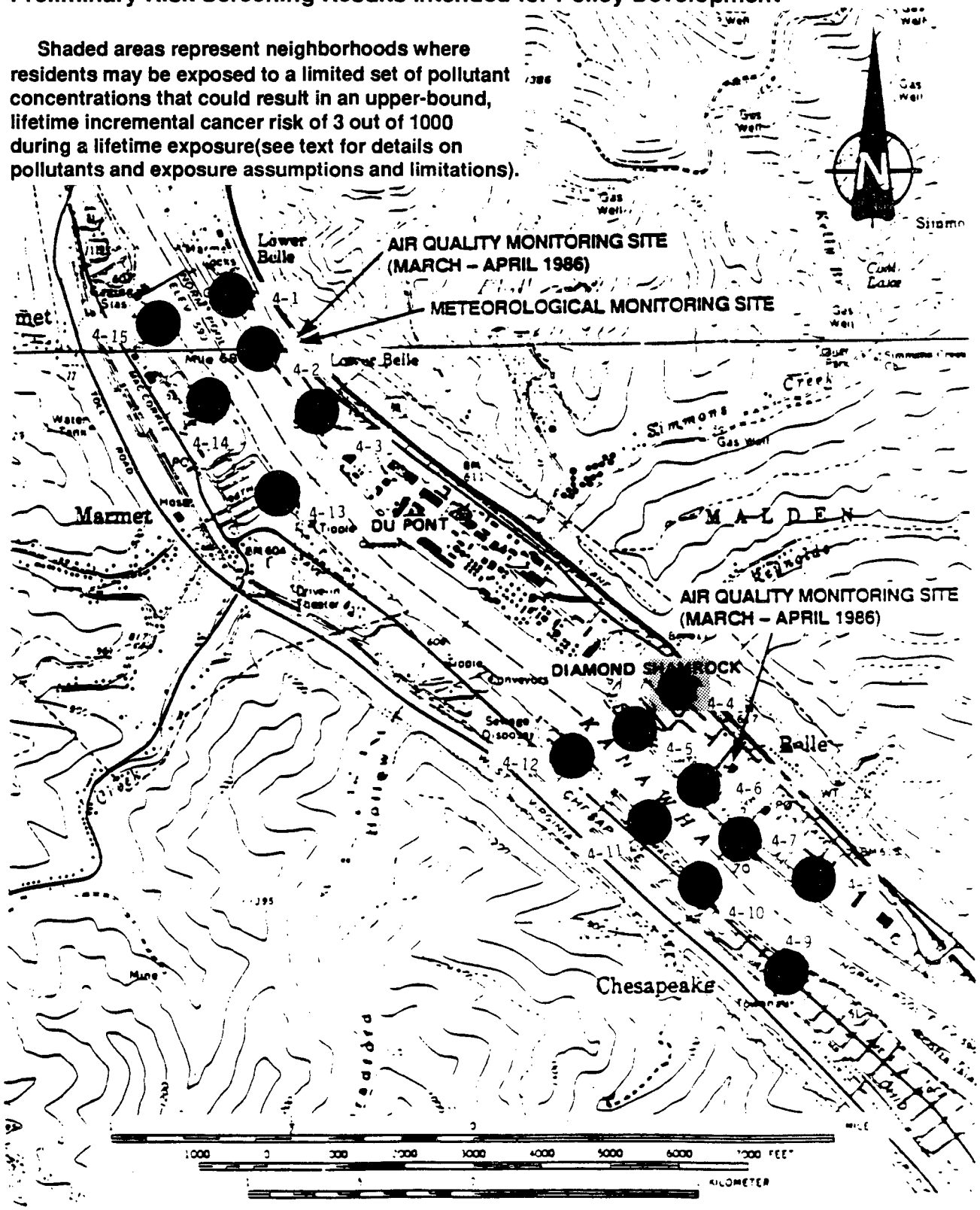

TABLE 26

KANAWHA VALLEY TOXICS SCREENING STUDY
KANAWHA VALLEY AIR QUALITY REPORT
PRELIMINARY RISK SCREENING RESULTS INTENDED FOR POLICY DEVELOPMENT

UPPER-BOUND ESTIMATES OF LIFETIME CANCER RISK CONTRIBUTED BY FACILITIES IN NEIGHBORHOODS SURROUNDING FACILITIES

BELLE

DuPont

COMPOUND

	CARBON TETRACHLO	CHLOROFORM	METHYLENE CHLORI	TOTAL
4- 1 (ISCLT)	4E-06	3E-06	5E-06	1E-05
4- 2 (ISCLT)	5E-06	3E-06	6E-06	1E-05
4- 3 (ISCLT)	4E-06	3E-06	8E-06	2E-05
4- 4 (ISCLT)	6E-06	5E-06	1E-06	1E-05
4- 5 (ISCLT)	3E-06	2E-06	1E-06	6E-06
4- 6 (ISCLT)	3E-06	2E-06	8E-07	5E-06
4- 7 (ISCLT)	2E-06	2E-06	6E-07	4E-06
4- 8 (ISCLT)	2E-06	1E-06	5E-07	3E-06
4- 9 (ISCLT)	10E-07	7E-07	4E-07	2E-06
4-10 (ISCLT)	1E-06	8E-07	5E-07	2E-06
4-11 (ISCLT)	1E-06	1E-06	6E-07	3E-06
4-12 (ISCLT)	2E-06	1E-06	7E-07	3E-06
4-13 (ISCLT)	1E-06	9E-07	8E-07	3E-06
4-14 (ISCLT)	2E-06	2E-06	2E-06	6E-06
4-15 (ISCLT)	2E-06	2E-06	2E-06	6E-06

Occidental

COMPOUND

	CARBON TETRACHLO	CHLOROFORM	METHYLENE CHLORI	TOTAL
4- 1 (ISCLT)	3E-05	2E-04	3E-05	2E-04
4- 2 (ISCLT)	3E-05	2E-04	4E-05	3E-04
4- 3 (ISCLT)	4E-05	2E-04	5E-05	3E-04
4- 4 (ISCLT)	4E-04	.002292	5E-04	.003192
4- 5 (ISCLT)	5E-05	3E-04	8E-05	4E-04
4- 6 (ISCLT)	5E-05	3E-04	7E-05	4E-04
4- 7 (ISCLT)	4E-05	2E-04	5E-05	3E-04
4- 8 (ISCLT)	3E-05	2E-04	3E-05	2E-04
4- 9 (ISCLT)	1E-05	7E-05	2E-05	1E-04
4-10 (ISCLT)	2E-05	9E-05	2E-05	1E-04
4-11 (ISCLT)	2E-05	1E-04	2E-05	1E-04
4-12 (ISCLT)	1E-05	6E-05	1E-05	9E-05
4-13 (ISCLT)	2E-05	1E-04	3E-05	2E-04
4-14 (ISCLT)	2E-05	1E-04	3E-05	2E-04
4-15 (ISCLT)	2E-05	1E-04	2E-05	2E-04

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

We have isolated the risk posed by the fugitive emissions of chloroform at site 4-4. Approximately five-sixths of the chloroform emissions are fugitives at site 4-4. Technical Appendix J presents fugitive contributions at the other neighborhood sites.

Risks to the Average Exposed Individual from Point Sources and in the Zone: Table 27 presents the risks to the average exposed individual within the Belle zone from these point source pollutants. According to Table 27, the total lifetime individual risk is 2.5 X 10^{-4} from carbon tetrachloride, chloroform, and methylene chloride. This is a two and one-half out of ten thousand probability of contracting cancer during a 70-year lifetime exposure to these concentrations of pollutants. Chloroform contributes most of this risk. Table 28 cites risks (and percent of total risk) by pollutant and facility. Chloroform contributes 71 percent of the total risk. Carbon tetrachloride and methylene chloride jointly contribute approximately 29 percent of the risk. According to Table 28, Occidental contributes approximately 98 percent of the total risk from these pollutants; Dupont contributes about two percent of the total risk.

Table 29 presents the contribution of fugitive and non-fugitive sources to the total risk to the average exposed individual from point sources. According to Table 29, fugitive sources contribute almost 85 percent of the risk posed by these pollutants to the average exposed individual.

Tables 30 and 31 present annual incidence by point source pollutant and by facility within the Belle zone. Belle has a population of 15,530 (Census 1980). We calculate a total annual incidence of .056 cases per year within the Belle zone from point source pollutants. Chloroform is the major pollutant contributing to this incidence; Occidental is the major facility.

Risks to the Average Exposed Individual from County-Wide Area Sources: Finally, in Table 32 we present lifetime, upper-bound risks to the average exposed individual from area sources. We calculated these risk estimates from the predicted concentrations from the box model. The upper-bound risk to the average exposed individual is approximately 5.3 X 10^{-5}, or five in a one hundred thousand probability of contracting cancer during a 70-year lifetime exposure from these county-wide area source pollutants. The incidence for county-wide area sources is .012 cases per year.

Charleston/South Charleston Zone

We modeled air emissions from two facilities, Union Carbide-South Charleston and the Union Carbide Technical Center. These facilities

TABLE 27

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

PRELIMINARY RISK SCREENING RESULTS
INTENDED FOR POLICY DEVELOPMENT

TOTAL UPPER-BOUND ESTIMATES OF LIFETIME CANCER
RISKS TO THE AVERAGE EXPOSED INDIVIDUAL
ACROSS POINT SOURCE POLLUTANTS

BELLE

POLLUTANT (Weight of Evidence)[1]	UPPER-BOUND RISKS TO THE AVERAGE EXPOSED INDIVIDUAL
Carbon Tetrachloride (B2)	3.5×10^{-5}
Chloroform (B2)	1.8×10^{-4}
Methylene Chloride (B2)	3.8×10^{-5}
Total	2.5×10^{-4}

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

1 Weight-of Evidence rating derived by CAG, based on EPA's classification system: A = proven human carcinogen; B = probable human carcinogen (B1 indicates limited evidence from human studies, B2 indicates sufficient evidence from animal studies but inadequate evidence from human studies); C = possible human carcinogen; D = not classifiable, and E = no evidence of carcinogenicity.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

TABLE 28

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

PRELIMINARY RISK SCREENING RESULTS
INTENDED FOR POLICY DEVELOPMENT

CONTRIBUTION OF FACILITIES AND POINT SOURCE
POLLUTANTS TO TOTAL UPPER-BOUND ESTIMATES
OF RISKS TO THE AVERAGE EXPOSED INDIVIDUAL
(BY RISK AND PERCENT OF TOTAL POINT SOURCE RISK)

BELLE

POLLUTANT (Weight of Evidence)[1]	FACILITY		
	Dupont (%)	Occidental (%)	Total Risk (%)
Carbon Tetrachloride (B2)	2.5×10^{-6} (1%)	3.3×10^{-5} (12.9%)	3.6×10^{-5} (13.9%)
Chloroform (B2)	1.9×10^{-6} (1%)	1.8×10^{-4} (70.4%)	1.8×10^{-4} (71.4%)
Methylene Chloride (B2)	1.0×10^{-6} (0.4%)	3.7×10^{-5} (14.4%)	3.8×10^{-5} (14.8%)
Total	5.4×10^{-6} (2.4%)	2.5×10^{-4} (97.7%)	2.5×10^{-4} (100%)

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

1 Weight-of Evidence rating derived by CAG, based on EPA's classification system: A = proven human carcinogen; B = probable human carcinogen (B1 indicates limited evidence from human studies, B2 indicates sufficient evidence from animal studies but inadequate evidence from human studies); C = possible human carcinogen; D = not classifiable, and E = no evidence of carcinogenicity.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

TABLE 29

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

PRELIMINARY RISK SCREENING RESULTS
INTENDED FOR POLICY DEVELOPMENT

CONTRIBUTION OF FUGITIVE AND NON-FUGITIVE POINT SOURCES TO TOTAL UPPER-BOUND ESTIMATES OF RISK TO THE AVERAGE EXPOSED INDIVIDUAL BY RISK AND PERCENT OF TOTAL POINT SOURCE RISK

BELLE

POLLUTANT (Weight of Evidence)[1]	FUGITIVE SOURCES	NON-FUGITIVE SOURCES
Carbon Tetrachloride (B2)	3.2×10^{-5} (12.7%)	3.0×10^{-6} (1.2%)
Chloroform (B2)	1.5×10^{-4} (60.4%)	2.7×10^{-5} (10.8%)
Methylene Chloride (B2)	2.9×10^{-5} (11.5%)	8.4×10^{-6} (3.3)
Total	2.1×10^{-4} (84.6%)	3.8×10^{-5} (15.3%)

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

1 Weight-of Evidence rating derived by CAG, based on EPA's classification system: A = proven human carcinogen; B = probable human carcinogen (B1 indicates limited evidence from human studies, B2 indicates sufficient evidence from animal studies but inadequate evidence from human studies); C = possible human carcinogen; D = not classifiable, and E = no evidence of carcinogenicity.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

TABLE 30

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

PRELIMINARY RISK SCREENING RESULTS
INTENDED FOR POLICY DEVELOPMENT

UPPER-BOUND ESTIMATES
OF EXCESS ANNUAL CANCER INCIDENCE
ACROSS POINT SOURCE POLLUTANTS

BELLE

POLLUTANT (Weight of Evidence)[1]	UPPER-BOUND ANNUAL CASES
Carbon Tetrachloride (B2)	.008
Chloroform (B2)	.040
Methylene Chloride (B2)	.008
TOTAL	.056

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

1 Weight-of Evidence rating derived by CAG, based on EPA's classification system: A = proven human carcinogen; B = probable human carcinogen (B1 indicates limited evidence from human studies, B2 indicates sufficient evidence from animal studies but inadequate evidence from human studies); C = possible human carcinogen; D = not classifiable, and E = no evidence of carcinogenicity.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

TABLE 31

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

PRELIMINARY RISK SCREENING RESULTS
INTENDED FOR POLICY DEVELOPMENT

UPPER-BOUND ESTIMATES OF EXCESS ANNUAL CANCER
INCIDENCE ACROSS FACILITIES

BELLE

FACILITY	UPPER-BOUND ANNUAL CASES
Dupont	.001
Occidental	.055
TOTAL	.056

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

TABLE 32

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

PRELIMINARY RISK SCREENING RESULTS
INTENDED FOR POLICY DEVELOPMENT

UPPER-BOUND ESTIMATES OF LIFETIME
CANCER RISKS TO THE AVERAGE EXPOSED INDIVIDUAL
ACROSS COUNTY-WIDE AREA SOURCES

BELLE

AREA SOURCE	UPPER BOUND LIFE-TIME CANCER RISK
Gas Marketing	1.1×10^{-6}
Heating	1.3×10^{-5}
Road Vehicles	3.1×10^{-5}
Solvent Use	7.7×10^{-6}
Waste Oil Burning	6.8×10^{-7}
TOTAL	5.3×10^{-5}

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

emitted four of the selected pollutants: acrylonitile, ethylene oxide, methylene chloride and vinylidene chloride. Propylene oxide is also emitted. Risk estimates for propylene oxide are presented in Technical Appendix I. For these pollutants and facilities, we present the results of our risk screening analysis, first for the exposure within selected neighborhoods and then for the exposure to the average individual within the Charleston/South Charleston zone.

Risks in Neighborhood Sites with Suspected Highest Exposures from Point Sources: Table 33 presents the estimated upper-bound lifetime incremental risk posed by each pollutant and the total estimated risk from these point source pollutants at neighborhoods we selected as likely to have relatively high exposure. The total risk is the sum of the risk of each pollutant (we assume that cancer risks are additive). Figure 10 maps estimated risks for the neighborhoods surrounding the facilities.

According to Table 33, nine sites within South Charleston, south and southwest of the Union Carbide-South Charleston facility, have predicted ambient air concentrations of these select pollutants that should present a total upper-bound risk estimate of 1 to 5 X 10^{-3} lifetime incremental risk. This is a one out of a thousand probability to a five out of a thousand probability of contracting cancer during a 70-year lifetime exposure to these chemicals at these sites. These sites cluster in South Charleston within a neighborhood that contains approximately 2,700 people (1980 Census). The remaining neighborhood sites could be exposed to a range of lifetime incremental risks of 2 X 10^{-4} to 8 X 10^{-4}.

For site 3-12 with the highest risk relative to other sites (5.5 X 10^{-3}), ethylene oxide contributes four-fifths of the total risk. Acrylonitile contributes about one-sixth of the total upper-bound incremental risk.

Table 34 presents the contribution of risk by the two facilities. Within this neighborhood of site 3-12, Union Carbide-South Charleston contributes nearly all of the risk.

For ethylene oxide and acrylonitile, we can estimate the contribution of fugitive and non-fugitive sources to the risk posed by these chemicals at site 3-12. Approximately one-fifth of the ethylene oxide concentration is provided by fugitive sources. Fugitive emissions contribute more than four-fifths of the risk posed by acrylonitile at site 3-12. Technical Appendix J presents fugitive contributions to the remaining sites.

TABLE 33

KANAWHA VALLEY TOXICS SCREENING STUDY
KANAWHA VALLEY AIR QUALITY REPORT
PRELIMINARY RISK SCREENING RESULTS INTENDED FOR POLICY DEVELOPMENT

UPPER-BOUND ESTIMATES OF LIFETIME CANCER RISKS IN NEIGHBORHOODS SURROUNDING FACILITIES FROM POINT SOURCE POLLUTANTS

CHARLESTON/SOUTH CHARLESTON)

COMPOUND

	ACRYLONITRILE	ETHYLENE OXIDE	METHYLENE CHLORIDE	VINYLIDENE CL (1)	TOTAL
3- 1 (ISCLT)	4E-05	3E-04	2E-07	5E-07	3E-04
3- 2 (ISCLT)	2E-05	2E-04	7E-08	3E-07	2E-04
3- 3 (ISCLT)	3E-05	1E-04	7E-08	3E-07	2E-04
3- 4 (ISCLT)	6E-05	5E-04	8E-08	5E-07	5E-04
3- 5 (ISCLT)	8E-05	4E-04	1E-07	8E-07	5E-04
3- 6 (ISCLT)	6E-05	2E-04	1E-07	5E-07	3E-04
3- 7 (ISCLT)	5E-05	2E-04	1E-07	4E-07	2E-04
3- 8 (ISCLT)	7E-04	3E-04	3E-07	9E-07	.001004
3- 9 (ISCLT)	3E-04	5E-04	3E-07	1E-06	8E-04
3-10 (ISCLT)	3E-04	10E-04	2E-07	2E-06	.001266
3-11 (ISCLT)	8E-04	0.00273	2E-07	4E-06	0.0035
3-12 (ISCLT)	9E-04	.004603	1E-07	1E-05	0.00553
3-13 (ISCLT)	4E-04	.001618	3E-07	3E-06	.001982
3-14 (ISCLT)	3E-04	.002709	2E-07	4E-06	.003042
3-15 (ISCLT)	9E-05	.001413	1E-07	1E-06	.001506
3-16 (ISCLT)	1E-04	.001125	2E-07	2E-06	0.00124
3-17 (ISCLT)	1E-04	.001034	3E-07	2E-06	0.00116
3-18 (ISCLT)	1E-04	1E-04	2E-07	5E-07	3E-04

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

FIGURE 10

Kanawha Valley Toxics Screening Study
Kanawha Valley Air Quality Report

Preliminary Risk Screening Results Intended for Policy Development

Shaded areas represent neighborhoods where residents may be exposed to a limited set of pollutant concentrations that could result in an upper-bound, lifetime incremental cancer risk of 1 to 5 out of 1,000 during a lifetime exposure(see text for details on pollutants and exposure assumptions and limitations).

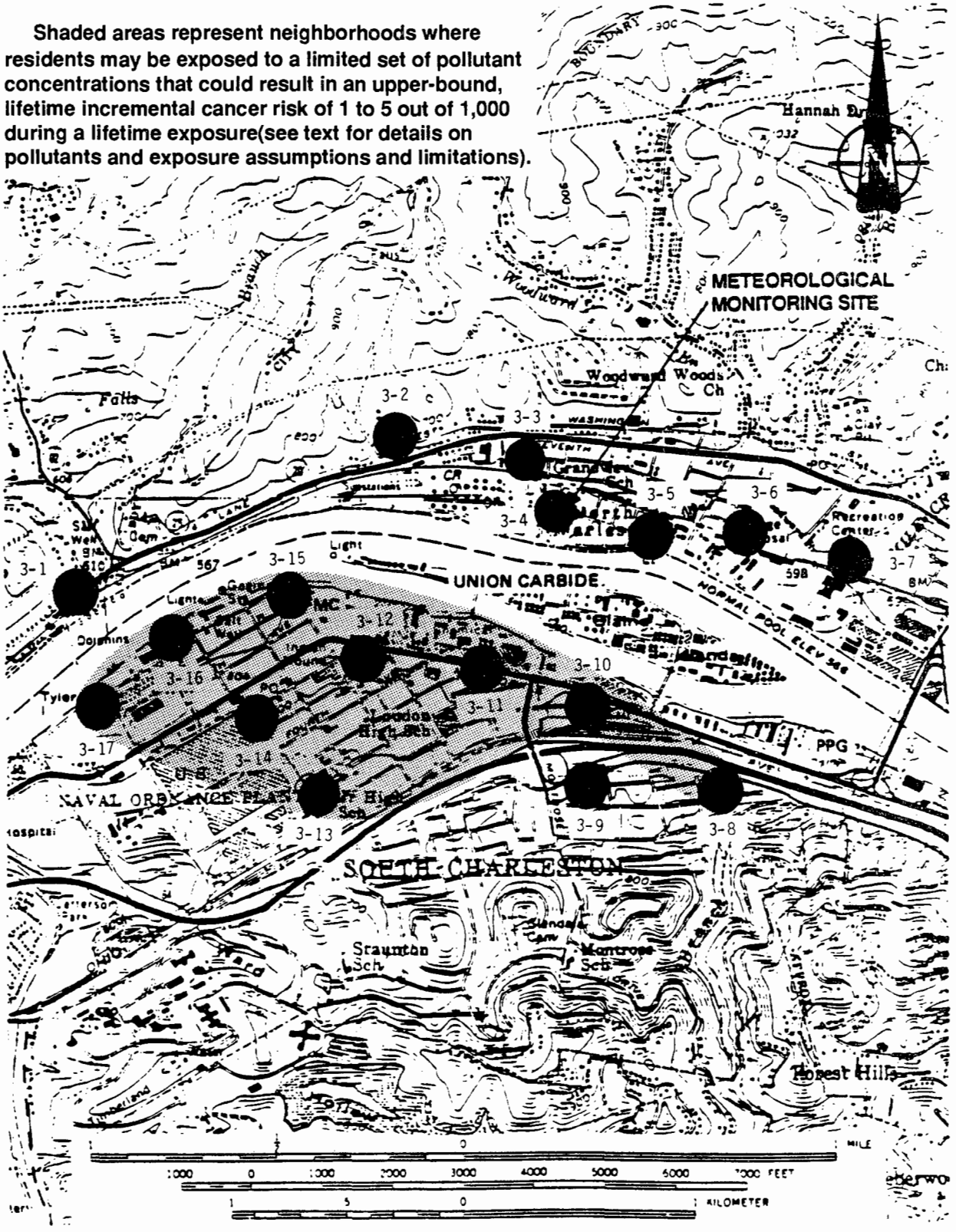

TABLE 34

KANAWHA VALLEY TOXICS SCREENING STUDY
KANAWHA VALLEY AIR QUALITY REPORT
PRELIMINARY RISK SCREENING RESULTS INTENDED FOR POLICY DEVELOPMENT

UPPER-BOUND ESTIMATES OF LIFETIME CANCER CONTRIBUTED BY FACILITIES IN NEIGHBORHOODS SURROUNDING FACILITIES

CHARLESTON/SOUTH CHARLESTON

Union Carbide - South Charleston

	COMPOUND				
	ACRYLONITRILE	ETHYLENE OXIDE	METHYLENE CHLORIDE	VINYLIDENE CL (1)	TOTAL
3- 1 (ISCLT)	4E-05	3E-04	3E-11	4E-07	3E-04
3- 2 (ISCLT)	2E-05	2E-04	3E-11	2E-07	2E-04
3- 3 (ISCLT)	3E-05	1E-04	3E-11	2E-07	2E-04
3- 4 (ISCLT)	6E-05	5E-04	4E-11	4E-07	5E-04
3- 5 (ISCLT)	8E-05	4E-04	1E-10	7E-07	5E-04
3- 6 (ISCLT)	6E-05	2E-04	10E-11	4E-07	3E-04
3- 7 (ISCLT)	5E-05	2E-04	1E-10	3E-07	2E-04
3- 8 (ISCLT)	7E-04	3E-04	3E-10	6E-07	.001002
3- 9 (ISCLT)	3E-04	5E-04	3E-10	9E-07	7E-04
3-10 (ISCLT)	3E-04	10E-04	8E-10	2E-06	.001265
3-11 (ISCLT)	8E-04	0.00273	2E-10	3E-06	.003499
3-12 (ISCLT)	9E-04	.004602	1E-10	1E-05	0.00553
3-13 (ISCLT)	4E-04	.001617	1E-10	3E-06	.001981
3-14 (ISCLT)	3E-04	.002709	8E-11	4E-06	.003041
3-15 (ISCLT)	9E-05	.001413	5E-11	1E-06	.001506
3-16 (ISCLT)	1E-04	.001124	4E-11	1E-06	.001239
3-17 (ISCLT)	1E-04	.001033	5E-11	1E-06	.001158
3-18 (ISCLT)	1E-04	1E-04	2E-10	3E-07	3E-04

Tech Center

	COMPOUND				
	ACRYLONITRILE	ETHYLENE OXIDE	METHYLENE CHLORIDE	VINYLIDENE CL (1)	TOTAL
3- 1 (ISCLT)	1E-07	4E-07	2E-07	1E-07	8E-07
3- 2 (ISCLT)	5E-08	2E-07	7E-08	7E-08	4E-07
3- 3 (ISCLT)	5E-08	2E-07	7E-08	7E-08	4E-07
3- 4 (ISCLT)	6E-08	2E-07	8E-08	8E-08	4E-07
3- 5 (ISCLT)	8E-08	2E-07	1E-07	10E-08	5E-07
3- 6 (ISCLT)	8E-08	3E-07	1E-07	1E-07	5E-07
3- 7 (ISCLT)	9E-08	3E-07	1E-07	1E-07	6E-07
3- 8 (ISCLT)	2E-07	8E-07	3E-07	3E-07	2E-06
3- 9 (ISCLT)	3E-07	9E-07	3E-07	4E-07	2E-06
3-10 (ISCLT)	2E-07	6E-07	2E-07	2E-07	1E-06
3-11 (ISCLT)	1E-07	4E-07	2E-07	2E-07	10E-07
3-12 (ISCLT)	1E-07	4E-07	1E-07	1E-07	8E-07
3-13 (ISCLT)	2E-07	7E-07	3E-07	3E-07	2E-06
3-14 (ISCLT)	2E-07	5E-07	2E-07	2E-07	1E-06
3-15 (ISCLT)	9E-08	3E-07	1E-07	1E-07	6E-07
3-16 (ISCLT)	1E-07	4E-07	2E-07	2E-07	9E-07
3-17 (ISCLT)	2E-07	7E-07	3E-07	3E-07	1E-06
3-18 (ISCLT)	1E-07	5E-07	2E-07	2E-07	9E-07

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

Risks to the Average Exposed Individual in the Zone from Point Source Pollutants: We present risk to the average exposed individual for the Charleston/South Charleston zone in Table 35. The total risk posed by these point source pollutants, which assumes additivity of cancer risks, is close to 3 X 10^{-4} upper-bound incremental risk during a 70-year lifetime exposure from these pollutants.

Table 36 displays the contribution by pollutant and facility to the risk to the average exposed individual. According to Table 36, Union Carbide-South Charleston contributes nearly all the risk from these pollutants.

For pollutants, ethylene oxide contributes three-fourths of the risk, and acrylonitile contributes more than one-fifth of the risk.

Table 37 presents the contribution of fugitive and non-fugitive sources to the total risk to the average exposed individual. According to Table 37, fugitive sources contribute 36 percent of the total risk to the average exposed individual.

Tables 38 and 39 present annual incidence by pollutant and facility within the Charleston/South Charleston zone. Charleston and South Charleston have a population of 51,750. We calculate a total incidence of .211 cases per year within the Charleston/South Charleston Zone. Ethylene oxide and acrylonitile from the Union Carbide-South Charleston facility are the primary pollutants contributing to this incidence.

Risks to the Average Exposed Individual from County-Wide Area Sources: Finally, in Table 40 we present lifetime upper-bound individual risks from area sources. We calculated these risk estimates from the predicted concentrations from the box model. Compared to the total risks from the two point sources, the risks from area sources are lower than the risks from point sources for the selected pollutants. The total risk from these county-wide area sources is 8 X 10^{-5}; the annual incidence from county-wide area sources is .059.

Institute Zone

We modeled air emissions from one facility, Rhone Poulenc, within the Institute zone. The facility emitted six of the selected pollutants: acrylonitrile, benzene, chloroform, ethylene oxide, methylene chloride and 1,3-butadiene. Formaldehyde and propylene oxide are also emitted. These pollutants are discussed in Technical Appendix I. We present the results of our preliminary risk screening analysis, first for the exposure within selected neighborhoods and then for the exposure to the average individual within the Institute Zone.

TABLE 35

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

PRELIMINARY RISK SCREENING RESULTS
INTENDED FOR POLICY DEVELOPMENT

TOTAL UPPER-BOUND ESTIMATES OF LIFETIME CANCER
RISKS TO THE AVERAGE EXPOSED INDIVIDUAL
ACROSS POINT SOURCE POLLUTANTS

CHARLESTON/SOUTH CHARLESTON

POLLUTANT (Weight of Evidence)[1]	UPPER-BOUND RISKS TO THE AVERAGE EXPOSED INDIVIDUAL
Acrylonitrile (B1)	6.3×10^{-5}
Ethylene Oxide (B1)	2.2×10^{-4}
Methylene Chloride (B2)	2.0×10^{-6}
Vinylidene Chloride (C)	1.4×10^{-6}
Total	2.9×10^{-4}

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

1 Weight-of Evidence rating derived by CAG, based on EPA's classification system: A = proven human carcinogen; B = probable human carcinogen (B1 indicates limited evidence from human studies, B2 indicates sufficient evidence from animal studies but inadequate evidence from human studies); C = possible human carcinogen; D = not classifiable, and E = no evidence of carcinogenicity.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

TABLE 36

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

PRELIMINARY RISK SCREENING RESULTS
INTENDED FOR POLICY DEVELOPMENT

CONTRIBUTION OF FACILITIES AND POINT SOURCE POLLUTANTS TO TOTAL UPPER-BOUND ESTIMATES OF RISK TO THE AVERAGE EXPOSED INDIVIDUAL BY RISK AND PERCENT OF TOTAL POINT SOURCE RISK

CHARLESTON/SOUTH CHARLESTON

POLLUTANT (Weight of Evidence)[1]	FACILITY		TOTAL RISK (%)
	Union Carbide	Union Carbide Tech-Center	
Acrylonitrile (B1)	6.2×10^{-5} (21.9%)	5.5×10^{-7} (.2%)	6.3×10^{-5} (22.1%)
Ethylene Oxide (B1)	2.2×10^{-4} (75.9%)	2.1×10^{-6} (.8%)	2.2×10^{-4} (76.7%)
Methylene chloride (B2)	9.7×10^{-11} (<.1%)	2.0×10^{-6} (.7%)	2.0×10^{-6} (0.7%)
Vinylidene chloride (C)	4.2×10^{-7} (.1%)	9.6×10^{-7} (.3%)	1.4×10^{-6} (.4%)
Total	2.8×10^{-4} (97.9%)	5.6×10^{-6} (2%)	2.9×10^{-4} (100%)

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

1 Weight-of Evidence rating derived by CAG, based on EPA's classification system: A = proven human carcinogen; B = probable human carcinogen (B1 indicates limited evidence from human studies, B2 indicates sufficient evidence from animal studies but inadequate evidence from human studies); C = possible human carcinogen; D = not classifiable, and E = no evidence of carcinogenicity.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

TABLE 37

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

PRELIMINARY RISK SCREENING RESULTS INTENDED FOR POLICY DEVELOPMENT

CONTRIBUTION OF FUGITIVE AND NON-FUGITIVE POINT SOURCES TO TOTAL UPPER-BOUND ESTIMATES OF RISK TO THE AVERAGE EXPOSED INDIVIDUAL BY RISK AND PERCENT OF TOTAL POINT SOURCE RISK

CHARLESTON/SOUTH CHARLESTON

POLLUTANT (WEIGHT OF EVIDENCE)	FUGITIVE SOURCES	NON-FUGITIVE SOURCES
Acrylonitrile (B1)	3.6×10^{-5} (12.6%)	2.7×10^{-5} (9.5%)
Ethylene oxide (B1)	6.8×10^{-5} (23.7%)	1.5×10^{-4} (52.9%)
Methylene chloride (B2)	-	2.0×10^{-6} (0.7%)
Vinylidene chloride (C)	9.9×10^{-7} (0.3%)	3.9×10^{-7} (0.1%)
TOTAL	1.0×10^{-4} (36.6%)	1.8×10^{-4} (63.2%)

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

Weight-of Evidence rating derived by CAG, based on EPA's classification system: A = proven human carcinogen; B = probable human carcinogen (B1 indicates limited evidence from human studies, B2 indicates sufficient evidence from animal studies but inadequate evidence from human studies); C = possible human carcinogen; D = not classifiable, and E = no evidence of carcinogenicity.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

TABLE 38

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

PRELIMINARY RISK SCREENING RESULTS
INTENDED FOR POLICY DEVELOPMENT

UPPER-BOUND ESTIMATES OF
EXCESS ANNUAL CANCER INCIDENCE
ACROSS FACILITIES

CHARLESTON/SOUTH CHARLESTON

FACILITY	UPPER-BOUND ANNUAL CASES
Union Carbide-South Charleston	.207
Union Carbide-Technical Center	.004
Total	.211

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

TABLE 39

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

PRELIMINARY RISK SCREENING RESULTS
INTENDED FOR POLICY DEVELOPMENT

UPPER-BOUND ESTIMATES OF
EXCESS ANNUAL CANCER INCIDENCE
ACROSS POLLUTANTS

CHARLESTON/SOUTH CHARLESTON

POLLUTANT (Weight of Evidence)[1]	UPPER-BOUND ANNUAL CASES
Acrylonitrile (B1)	.046
Ethylene Oxide (B1)	.163
Methylene Chloride (B2)	.001
Vinylidene Chloride (C)	.001
Total	.211

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

1 Weight-of Evidence rating derived by CAG, based on EPA's classification system: A = proven human carcinogen; B = probable human carcinogen (B1 indicates limited evidence from human studies, B2 indicates sufficient evidence from animal studies but inadequate evidence from human studies); C = possible human carcinogen; D = not classifiable, and E = no evidence of carcinogenicity.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

TABLE 40

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

PRELIMINARY RISK SCREENING RESULTS
INTENDED FOR POLICY DEVELOPMENT

UPPER-BOUND ESTIMATES OF LIFETIME
CANCER RISKS TO THE AVERAGE EXPOSED INDIVIDUAL
ACROSS COUNTY-WIDE AREA SOURCES

CHARLESTON/SOUTH CHARLESTON

AREA SOURCE	UPPER BOUND LIFE-TIME CANCER RISK
Gas Marketing	1.4×10^{-6}
Solvent Use	1.3×10^{-5}
Heating	1.3×10^{-5}
Road Vehicles	5.1×10^{-5}
Waste Oil Burning	1.2×10^{-6}
Total	8.0×10^{-5}

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

Risks in Neighborhood Sites with Suspected Highest Exposures from Point Source Pollutants: Table 41 presents the estimated upper-bound lifetime incremental risk posed by each pollutant and the total estimated risk from these pollutants, which is the sum of the risks posed by each pollutant. Figure 11 maps the estimated risks from point source pollutants for the neighborhoods surrounding the facility.

According to Table 41, seven sites surrounding the Union Carbide facility have predicted ambient air concentrations of these pollutants that could pose an upper-bound risk estimate of 1 to 8 X 10^{-3} lifetime incremental risk using upper-bound potency values. This is a one to eight in a thousand probability of contracting cancer over a lifetime exposure from these pollutants. For the most affected site, site 2-1, there is an eight in a thousand chance of contracting cancer from a 70-year exposure to these concentrations.

The total population within these most affected neighborhoods is approximately 1,300 people (1980 Census). The modeled concentrations for the remaining sites surrounding the facility pose a total risk on the order of 10^{-4} lifetime risks from these pollutants.

At site 2-1, the site most affected by these pollutants, 1,3-butadiene contributes approximately three-fourths of the total risk at this site. Ethylene oxide contributes close to one-fifth of the total risk. Acrylonitrile, methylene chloride, benzene, and chloroform pose the remaining risk.

Nearly all of the 1,3-butadiene risk is from fugitive emissions.

Risks to the Average Exposed Individual from Point Source Pollutants: Table 42 presents the risks for the average exposed individual within the Institute zone.

According to Table 42, the total lifetime individual incremental risk is 1.1 X 10^{-3} from a 70-year exposure from the point source pollutants. This is an upper-bound estimate of one in a thousand chance of contracting cancer from these pollutants during a lifetime exposure to these concentrations.

Ethylene oxide contributes over one-half of the total risk posed by these pollutants. 1,3-Butadiene contributes close to one-third of the total estimated risk. Acrylonitrile contributes approximately one-tenth of the total risk and chloroform, methylene chloride, and benzene contribute the remaining risk.

Table 43 presents the contribution of fugitive and non-fugitive sources to the total risk to the average exposed individual. According to Table 43, fugitive sources contribute 70 percent of the total risk to the average exposed individual.

TABLE 41

KANAWHA VALLEY TOXICS SCREENING STUDY
KANAWHA VALLEY AIR QUALITY REPORT
PRELIMINARY RISK SCREENING RESULTS INTENDED FOR POLICY DEVELOPMENT

UPPER-BOUND ESTIMATES OF LIFETIME CANCER RISKS IN NEIGHBORHOODS SURROUNDING FACILITIES FROM POINT SOURCE POLLUTANTS

INSTITUTE

COMPOUND

	ACRYLONITRILE	BENZENE	CHLOROFORM	ETHYLENE OXIDE	METHYLENE CHLORI	1,3-BUTADIENE	TOTAL
2- 1 (ISCLT)	4E-04	3E-07	2E-04	.001449	1E-05	.005941	0.00802
2- 2 (ISCLT)	9E-05	4E-08	7E-05	6E-04	5E-06	8E-04	.001658
2- 3 (ISCLT)	6E-05	3E-08	1E-04	7E-04	1E-05	4E-04	.001356
2- 4 (ISCLT)	5E-05	3E-08	1E-04	6E-04	10E-06	3E-04	.001134
2- 5 (ISCLT)	4E-05	3E-08	1E-04	5E-04	6E-06	2E-04	9E-04
2- 6 (ISCLT)	3E-05	2E-08	7E-05	4E-04	5E-06	2E-04	7E-04
2- 7 (ISCLT)	3E-05	2E-08	6E-05	3E-04	4E-06	2E-04	6E-04
2- 8 (ISCLT)	3E-05	2E-08	5E-05	3E-04	2E-06	1E-04	5E-04
2- 9 (ISCLT)	2E-05	2E-08	2E-05	2E-04	1E-06	10E-05	3E-04
2-10 (ISCLT)	2E-05	2E-08	2E-05	1E-04	1E-06	9E-05	3E-04
2-11 (ISCLT)	1E-05	1E-08	2E-05	10E-05	8E-07	7E-05	2E-04
2-12 (ISCLT)	3E-05	2E-08	5E-05	2E-04	3E-06	2E-04	5E-04
2-13 (ISCLT)	6E-05	3E-08	7E-05	5E-04	2E-06	4E-04	.001044
2-14 (ISCLT)	7E-05	5E-08	6E-05	.001296	2E-06	3E-04	.001733
2-15 (ISCLT)	2E-04	2E-07	1E-04	.001898	6E-06	3E-04	0.00252

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

FIGURE 11

Kanawha Valley Toxics Screening Study
Kanawha Valley Air Quality Report

Preliminary Risk Screening Results Intended for Policy Development

Shaded areas represent neighborhoods where residents may be exposed to a limited set of pollutant concentrations that could result in an upper-bound, lifetime incremental cancer risk of 1 to 8 out of 1,000 during a lifetime exposure(see text for details on pollutants and exposure assumptions and limitations).

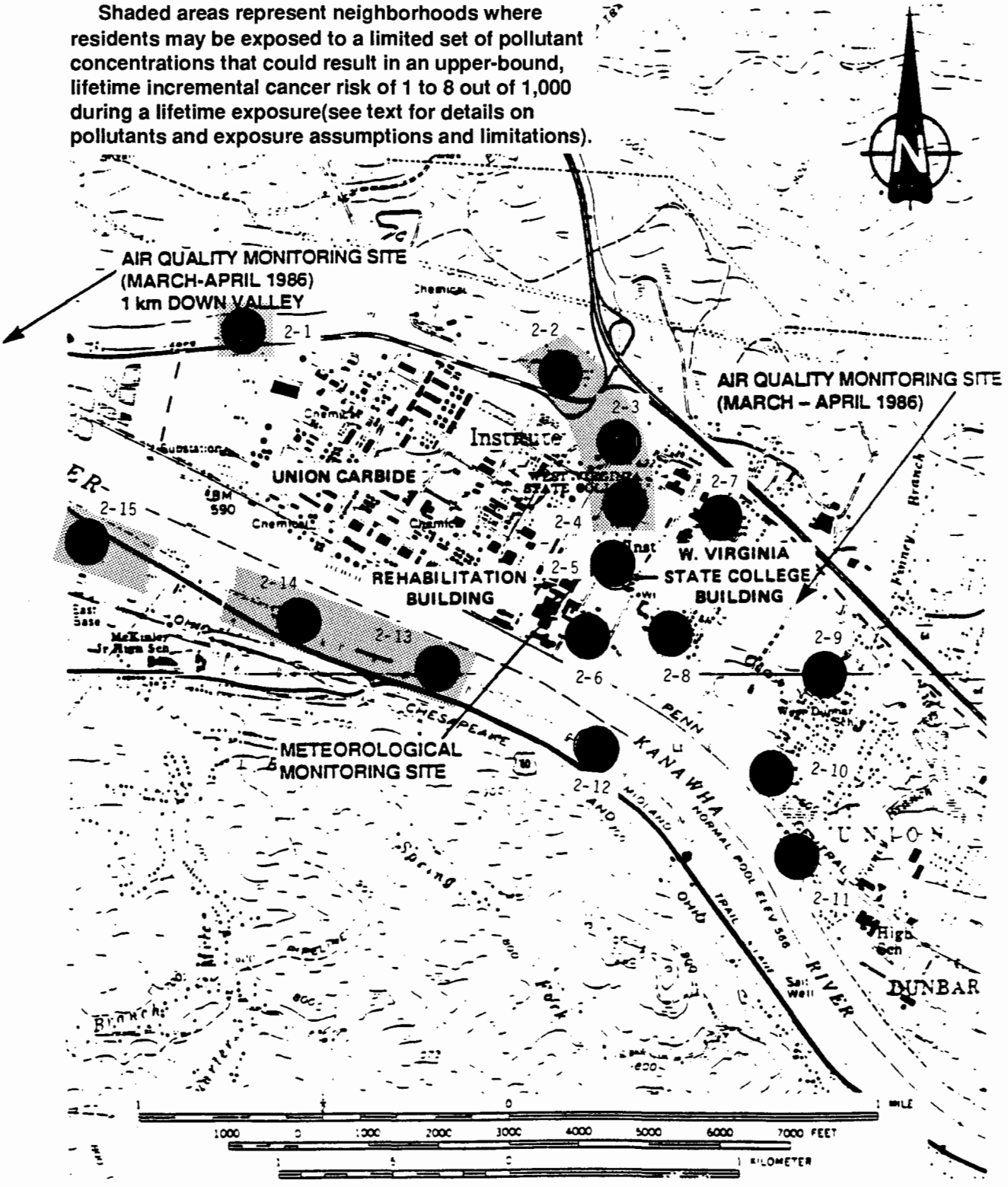

TABLE 42

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

PRELIMINARY RISK SCREENING RESULTS
INTENDED FOR POLICY DEVELOPMENT

TOTAL UPPER-BOUND ESTIMATES OF LIFETIME CANCER
RISKS TO THE AVERAGE EXPOSED INDIVIDUAL
ACROSS POINT SOURCE POLLUTANTS

INSTITUTE

POLLUTANT (Weight of Evidence)[1]	UPPER-BOUND RISKS TO THE AVERAGE EXPOSED INDIVIDUAL
Acrylonitrile (B1)	1.1×10^{-4}
Benzene (A)	1.4×10^{-7}
Chloroform (B2)	5.5×10^{-5}
Ethylene Oxide (B1)	5.8×10^{-4}
Methylene Chloride (B2)	2.7×10^{-6}
1,3-Butadiene (B2)	3.6×10^{-4}
Total	1.1×10^{-3}

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

1 Weight-of Evidence rating derived by CAG, based on EPA's classification system: A = proven human carcinogen; B = probable human carcinogen (B1 indicates limited evidence from human studies, B2 indicates sufficient evidence from animal studies but inadequate evidence from human studies); C = possible human carcinogen; D = not classifiable, and E = no evidence of carcinogenicity.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

TABLE 43

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

PRELIMINARY RISK SCREENING RESULTS
INTENDED FOR POLICY DEVELOPMENT

CONTRIBUTION OF FUGITIVE AND NON-FUGITIVE POINT SOURCES TO TOTAL UPPER-BOUND ESTIMATES OF RISK TO THE AVERAGE EXPOSED INDIVIDUAL BY RISK AND PERCENT OF TOTAL POINT SOURCE RISK

INSTITUTE

POLLUTANT (Weight of Evidence)[1]	FUGITIVE SOURCES (%)	NON-FUGITIVE SOURCES (%)
Acrylonitrile (B1)	2.9×10^{-5} (2.6%)	8.0×10^{-5} (7.3%)
Benzene (A)	-	1.4×10^{-7} (<.1%)
Chloroform (B2)	2.3×10^{-5} (2.1%)	3.2×10^{-5} (2.9%)
Ethylene oxide (B1)	3.7×10^{-4} (33.3%)	2.1×10^{-4} (19.2%)
Methylene chloride (B2)	1.4×10^{-6} (.1%)	1.3×10^{-6} (.1%)
1,3-Butadiene (B2)	3.6×10^{-4} (32.1%)	2.5×10^{-6} (.2%)
Total	7.8×10^{-4} (70.2%)	3.3×10^{-4} (29.7%)

THE UNIT RISK FACTORS USEO IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

1 Weight-of Evidence rating derived by CAG, based on EPA's classification system: A = proven human carcinogen; B = probable human carcinogen (B1 indicates limited evidence from human studies, B2 indicates sufficient evidence from animal studies but inadequate evidence from human studies); C = possible human carcinogen; D = not classifiable, and E = no evidence of carcinogenicity.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

Table 44 cites annual incidence by point source pollutant within the Institute zone. Institute has a total population of 22,390 (1980 Census). We calculated a total annual incidence of .354 cases per year, using upper-bound potency values. Ethylene oxide is the most significant of the six pollutants, contributing about one-half of the risk to the total population.

Risks to the Average Exposed Individual from County-Wide Area Sources: Finally, we present life-time incremental individual risks from county-wide area sources. We calculate these estimates from the predicted concentrations for the box model. We present average individual risks from county-wide area sources in Table 45. Compared to the total risks from Union Carbide (1.1×10^{-3}), area sources contribute an individual life-time incremental risk of 5×10^{-5}, approximately two orders of magnitude lower than the risk from the Rhone Poulenc facility. The incidence from county-wide area sources is .015.

Nitro Zone

We modeled air emissions from two facilities, Artel and Monsanto, within the Nitro zone. These facilities together emitted nine of the selected pollutants: acrylonitrile, benzene, carbon tetrachloride, chloroform, methylene chloride, vinyl chloride, ethylene chloride, allyl chloride, and trichloroethylene. Formaldehyde is also emitted in Nitro. Risk estimates for formaldehyde are presented in Technical Appendix I. For these pollutants and facilities, we present the results of our risk screening analysis, first for the exposures within selected neighborhoods and then for the exposure to the average individual within the Nitro zone.

Risks in Neighborhood Sites with Suspected Highest Exposures From Point Source Pollutants: Table 46 presents the estimated upper-bound incremental risk posed by each point source pollutant. The total risk is the sum of the risk of each pollutant (we assume that cancer risks are additive). Figure 12 maps the estimated risks for the neighborhoods surrounding the facilities.

According to Table 46 and Figure 12, ten sites have a total risk ranging from 1 to 6×10^{-6} individual lifetime incremental risk from these pollutants. This is a one to six in a million probability of contracting cancer during a 70-year exposure to these concentrations of pollutants. Many of these neighborhoods are located on the east bank of the Kanawha River between the two facilities and the valley walls. Monsanto contributes most of the total risk at these sites, primarily from the emission of trichoroethylene. Table 47 breaks out estimated risk contributed by facilities.

Risks to the Average Exposed Individual from Point Source Pollutants: We present the total risk to the average exposed individual within the

TABLE 44

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

PRELIMINARY RISK SCREENING RESULTS
INTENDED FOR POLICY DEVELOPMENT

UPPER-BOUND ESTIMATES OF EXCESS
ANNUAL CANCER INCIDENCE
ACROSS POINT SOURCE POLLUTANTS

INSTITUTE

POLLUTANT (Weight of Evidence)[1]	UPPER-BOUND ANNUAL CASES
Acrylonitrile (B1)	.035
Benzene (A)	<.0001
Chloroform (B2)	.018
Ethylene Oxide (B1)	.185
Methylene Chloride (B2)	.001
1,3-Butadiene (B2)	.115
Total	.354

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

1 Weight-of Evidence rating derived by CAG, based on EPA's classification system: A = proven human carcinogen; B = probable human carcinogen (B1 indicates limited evidence from human studies, B2 indicates sufficient evidence from animal studies but inadequate evidence from human studies); C = possible human carcinogen; D = not classifiable, and E = no evidence of carcinogenicity.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

TABLE 45

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

PRELIMINARY RISK SCREENING RESULTS
INTENDED FOR POLICY DEVELOPMENT

UPPER-BOUND ESTIMATES OF LIFETIME
CANCER RISKS TO THE AVERAGE EXPOSED INDIVIDUAL
ACROSS AREA SOURCES

INSTITUTE

AREA SOURCE	UPPER BOUND LIFE-TIME CANCER RISK
Gas Marketing	7.9×10^{-7}
Solvent Use	6.3×10^{-6}
Heating	1.3×10^{-5}
Road Vehicles	2.6×10^{-5}
Waste Oil Burning	6.5×10^{-7}
Total	4.7×10^{-5}

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

TABLE 46

KANAWHA VALLEY TOXICS SCREENING STUDY
KANAWHA VALLEY AIR QUALITY REPORT
PRELIMINARY RISK SCREENING RESULTS INTENDED FOR POLICY DEVELOPMENT

UPPER-BOUND ESTIMATES OF LIFETIME CANCER RISKS IN NEIGHBORHOODS SURROUNDING FACILITIES FROM POINT SOURCE POLLUTANTS

NITRO

COMPOUND

	ACRYLONITRILE	BENZENE	CARBON TETRACHLORIDE	CHLOROFORM	METHYLENE CHLORIDE	VINYL CHLORIDE	ETHYLENE CHLORIDE	ALLYL CHLORIDE	TRICHLOROETHYLENE	TOTAL
1- 1 (ISCLT)	1E-08	3E-10	5E-10	8E-10	7E-11	4E-11	3E-08	5E-10	1E-06	1E-06
1- 2 (ISCLT)	10E-09	2E-10	4E-10	6E-10	5E-11	3E-11	2E-08	4E-10	1E-06	1E-06
1- 3 (ISCLT)	2E-08	4E-10	8E-10	1E-09	1E-10	7E-11	5E-08	1E-09	2E-06	2E-06
1- 4 (ISCLT)	5E-08	10E-10	2E-09	3E-09	3E-10	2E-10	1E-07	3E-09	6E-06	6E-06
1- 5 (ISCLT)	7E-08	1E-09	3E-09	4E-09	4E-10	2E-10	2E-07	4E-09	6E-06	6E-06
1- 6 (ISCLT)	1E-07	2E-09	4E-09	6E-09	6E-10	4E-10	3E-07	7E-09	4E-06	4E-06
1- 7 (ISCLT)	3E-07	6E-09	1E-08	2E-08	1E-09	9E-10	6E-07	1E-08	2E-06	3E-06
1- 8 (ISCLT)	4E-07	9E-09	2E-08	2E-08	2E-09	1E-09	9E-07	2E-08	2E-06	3E-06
1- 9 (ISCLT)	2E-07	4E-09	7E-09	1E-08	9E-10	6E-10	5E-07	2E-08	1E-06	2E-06
1-10 (ISCLT)	8E-08	1E-09	3E-09	4E-09	4E-10	2E-10	2E-07	6E-09	9E-07	1E-06
1-11 (ISCLT)	4E-08	8E-10	1E-09	2E-09	2E-10	1E-10	1E-07	3E-09	7E-07	9E-07
1-12 (ISCLT)	3E-08	5E-10	10E-10	1E-09	1E-10	8E-11	7E-08	2E-09	6E-07	7E-07
1-13 (ISCLT)	3E-08	5E-10	10E-10	2E-09	1E-10	9E-11	7E-08	2E-09	6E-07	7E-07
1-14 (ISCLT)	3E-08	6E-10	1E-09	2E-09	2E-10	1E-10	9E-08	3E-09	7E-07	8E-07

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

Figure 12

Kanawha Valley Toxics Screening Study
Kanawha Valley Air Quality Report

Preliminary Risk Screening Results Intended for Policy Development

Shaded areas represent neighborhoods where residents may be exposed to a limited set of pollutant concentrations that could result in an upper-bound, lifetime incremental cancer risk of 1 to 6 out of 1,000,000 during a lifetime exposure(see text for details on pollutants and exposure assumptions and limitations).

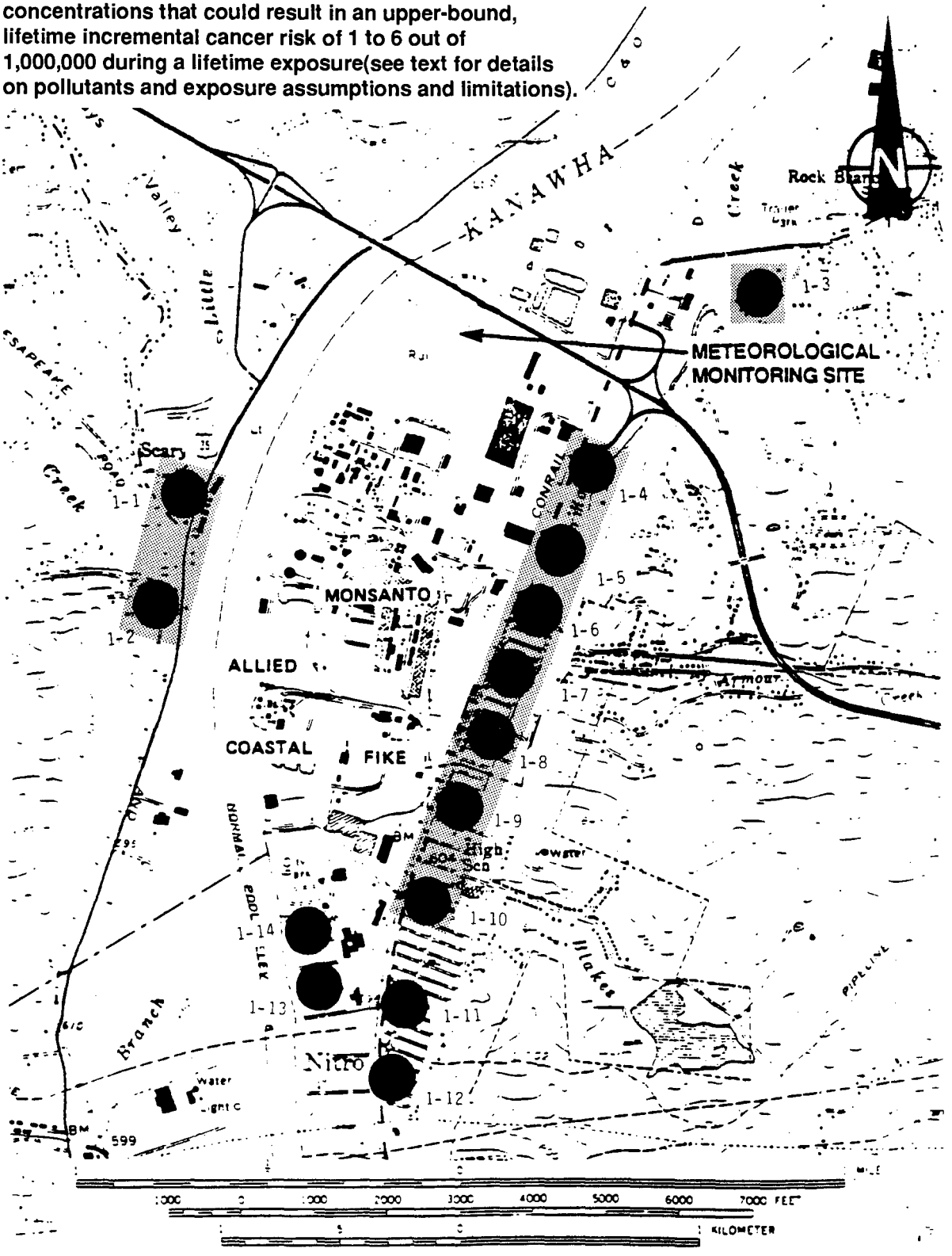

TABLE 47

KANAWHA VALLEY TOXICS SCREENING STUDY
KANAWHA VALLEY AIR QUALITY REPORT
PRELIMINARY RISK SCREENING RESULTS INTENDED FOR POLICY DEVELOPMENT

UPPER-BOUND ESTIMATES OF LIFETIME CANCER RISKS CONTRIBUTED BY FACILITIES IN NEIGHBORHOODS SURROUNDING FACILITIES

NITRO
(Page 1 of 2)

Artel

COMPOUND

	ACRYLONITRILE	BENZENE	CARBON TETRACHLORIDE	CHLOROFORM	METHYLENE CHLORIDE	VINYL CHLORIDE	METHYLENE CHLORIDE	VINYL CHLORIDE	TOTAL
1- 1 (ISCLT)	1E-08	3E-10	5E-10	8E-10	7E-11	4E-11	3E-08	5E-10	4E-08
1- 2 (ISCLT)	10E-09	2E-10	4E-10	6E-10	5E-11	3E-11	2E-08	4E-10	3E-08
1- 3 (ISCLT)	2E-08	4E-10	8E-10	1E-09	1E-10	7E-11	5E-08	1E-09	7E-08
1- 4 (ISCLT)	5E-08	10E-10	2E-09	3E-09	3E-10	2E-10	1E-07	3E-09	2E-07
1- 5 (ISCLT)	7E-08	1E-09	3E-09	4E-09	4E-10	2E-10	2E-07	4E-09	2E-07
1- 6 (ISCLT)	1E-07	2E-09	4E-09	6E-09	6E-10	4E-10	3E-07	7E-09	4E-07
1- 7 (ISCLT)	3E-07	6E-09	1E-08	2E-08	1E-09	9E-10	6E-07	1E-08	9E-07
1- 8 (ISCLT)	4E-07	9E-09	2E-08	2E-08	2E-09	1E-09	9E-07	2E-08	1E-06
1- 9 (ISCLT)	2E-07	4E-09	7E-09	1E-08	9E-10	6E-10	5E-07	2E-08	7E-07
1-10 (ISCLT)	8E-08	1E-09	3E-09	4E-09	4E-10	2E-10	2E-07	6E-09	3E-07
1-11 (ISCLT)	4E-08	8E-10	1E-09	2E-09	2E-10	1E-10	1E-07	3E-09	1E-07
1-12 (ISCLT)	3E-08	5E-10	10E-10	1E-09	1E-10	8E-11	7E-08	2E-09	1E-07
1-13 (ISCLT)	3E-08	5E-10	10E-10	2E-09	1E-10	9E-11	7E-08	2E-09	1E-07
1-14 (ISCLT)	3E-08	6E-10	1E-09	2E-09	2E-10	1E-10	9E-08	3E-09	1E-07

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

TABLE 47

KANAWHA VALLEY TOXICS SCREENING STUDY
KANAWHA VALLEY AIR QUALITY REPORT
PRELIMINARY RISK SCREENING RESULTS INTENDED FOR POLICY DEVELOPMENT

UPPER-BOUND ESTIMATES OF LIFETIME CANCER RISKS CONTRIBUTED BY FACILITIES IN NEIGHBORHOODS SURROUNDING FACILITIES

NITRO
(Page 2 of 2)

Monsanto

	COMPOUND	
	TRICHLOROETHYLEN	TOTAL
1- 1 (ISCLT)	1E-06	1E-06
1- 2 (ISCLT)	1E-06	1E-06
1- 3 (ISCLT)	2E-06	2E-06
1- 4 (ISCLT)	6E-06	6E-06
1- 5 (ISCLT)	6E-06	6E-06
1- 6 (ISCLT)	4E-06	4E-06
1- 7 (ISCLT)	2E-06	2E-06
1- 8 (ISCLT)	2E-06	2E-06
1- 9 (ISCLT)	1E-06	1E-06
1-10 (ISCLT)	9E-07	9E-07
1-11 (ISCLT)	7E-07	7E-07
1-12 (ISCLT)	6E-07	6E-07
1-13 (ISCLT)	6E-07	6E-07
1-14 (ISCLT)	7E-07	7E-07

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

Nitro zone in Table 48. The total risk presented by these pollutants is 2.5×10^{-6}. This is more than a two in a million probability of contracting cancer during a 70-year lifetime exposure from these concentrations of point source pollutants. According to Table 49, trichloroethylene from Monsanto contributes more than nine-tenths of this total risk.

Tables 50 and 51 present the annual incidence by pollutant and facility within the Nitro zone for point sources. Nitro has a population of 9,990. We calculated a total annual incidence of .0003 cases per year from these point source pollutants using upper-bound potency values.

Risks to the Average Exposed Individual from County-wide Area Sources: Finally, we present lifetime, upper-bound incremental risks from county-wide area sources in Table 52. We calculated these risk estimates from the predicted concentrations from the box model. For area sources, we observe a total risk of 1.4×10^{-5}, which is larger than the estimated risk from the selected point source pollutants. Incidence from county-wide area sources is approximately .002 cases per year.

VII. AIR ANALYSIS SUMMARY AND CONCLUSIONS

The Kanawha Valley Air Quality Report presents a conservative assessment of the potential cancer health effects that a select set of pollutants may pose to a limited population within the Kanawha Valley. Based on an emission inventory for volatile organic compounds developed by the West Virginia Air Pollution Control Commission and EPA generated estimates of county-wide area source pollutants, our analysis develops exposure concentrations for various populated areas within the study area. These exposure estimates are the basis for our risk assessment analysis of these chemicals. Using upper-bound cancer unit risk values with exposure estimates, we developed upper-bound lifetime, incremental individual risk estimates which assume a 70-year exposure to these pollutants.

We emphasize that our results are not predictors of absolute risk. We have reviewed only a small set of pollutants of our risk assessments; a complete review of potential health effects from all pollutants has not been completed.

A summary of the air quality report and findings follows:

- The air analysis for the Kanawha Valley Toxics Screening study was limited to four geographic areas or zones within the Kanawha Valley: Belle, Charleston/South Charleston, Institute, and Nitro. The valley terrain of the Kanawha Valley precluded modeling exposures throughout the 60-mile river stretch. We selected these four zones based on the geographic orientation of the valley within

TABLE 48

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

PRELIMINARY RISK SCREENING RESULTS
INTENDED FOR POLICY DEVELOPMENT

TOTAL UPPER-BOUND ESTIMATES OF LIFETIME CANCER
RISKS TO THE AVERAGE EXPOSED INDIVIDUAL
ACROSS POINT SOURCE POLLUTANTS

NITRO

POLLUTANT (Weight of Evidence)[1]	UPPER-BOUND RISKS TO THE AVERAGE EXPOSED INDIVIDUAL
Acrylonitrile (B1)	5.7×10^{-8}
Benzene (A)	1.2×10^{-9}
Carbon Tetrachloride (B2)	2.2×10^{-9}
Chloroform (B2)	3.4×10^{-9}
Methylene Chloride (B2)	3.0×10^{-10}
Vinyl Chloride (A)	1.9×10^{-10}
Ethylene Chloride (B2)	1.4×10^{-7}
Allyl Chloride (B2)	3.3×10^{-9}
Trichloroethylene (B2)	2.3×10^{-6}
Total	2.5×10^{-6}

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

[1] Weight-of Evidence rating derived by CAG, based on EPA's classification system: A = proven human carcinogen; B = probable human carcinogen (B1 indicates limited evidence from human studies, B2 indicates sufficient evidence from animal studies but inadequate evidence from human studies); C = possible human carcinogen; D = not classifiable, and E = no evidence of carcinogenicity.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

TABLE 49

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

PRELIMINARY RISK SCREENING RESULTS
INTENDED FOR POLICY DEVELOPMENT

CONTRIBUTION OF FACILITIES AND POINT SOURCE POLLUTANTS TO TOTAL UPPER-BOUND ESTIMATES OF RISK TO THE AVERAGE EXPOSED INDIVIDUAL BY RISK AND PERCENT OF TOTAL POINT SOURCE RISK

NITRO

POLLUTANT (Weight of Evidence)[1]	FACILITY		TOTAL RISK
	Fike	Monsanto	
Acrylonitrile (B1)	5.7×10^{-8} (2.3%)	-	5.7×10^{-8} (2.3%)
Benzene (A)	1.2×10^{-9} (<.1%)	-	1.2×10^{-9} (<.1%)
Carbon Tetrachloride(B2)	2.2×10^{-9} (.1%)	-	2.2×10^{-9} (.1%)
Chloroform (B2)	3.4×10^{-9} (.1%)	-	3.4×10^{-9} (.1%)
Methylene Chloride (B2)	3.0×10^{-10} (<.1%)	-	3.0×10^{-10} (<.1%)
Vinyl Chloride (A)	1.9×10^{-10} (<.1%)	-	1.9×10^{-10} (<.1%)
Ethylene Chloride (B2)	1.4×10^{-7} (5.4%)	-	1.4×10^{-7} (5.4%)
Allyl Chloride (B2)	3.3×10^{-9} (.1%)	-	3.3×10^{-9} (.1%)
Trichloroethylene (B2)	-	2.3×10^{-6} (91.9%)	2.3×10^{-6} (91.9%)
Total	2.1×10^{-7} (8.0%)	2.3×10^{-6} (91.9%)	2.5×10^{-6} (100%)

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

[1] Weight-of Evidence rating derived by CAG, based on EPA's classification system: A = proven human carcinogen; B = probable human carcinogen (B1 indicates limited evidence from human studies, B2 indicates sufficient evidence from animal studies but inadequate evidence from human studies); C = possible human carcinogen; D = not classifiable, and E = no evidence of carcinogenicity.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

TABLE 50

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

PRELIMINARY RISK SCREENING RESULTS
INTENDED FOR POLICY DEVELOPMENT

UPPER-BOUND ESTIMATES OF EXCESS
ANNUAL CANCER INCIDENCE ACROSS
POINT SOURCE POLLUTANTS

NITRO

POLLUTANT (Weight of Evidence)[1]	UPPER-BOUND ANNUAL CASES
Acrylonitrile (B1)	<.0001
Benzene (A)	<.0001
Carbon Tetrachloride (B2)	<.0001
Chloroform (B2)	<.0001
Methylene Chloride (B2)	<.0001
Vinyl Chloride (A)	<.0001
Ethylene Chloride (B2)	<.0001
Allyl Chloride (B2)	<.0001
Trichloroethylene (B2)	.0003
Total	.0003

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

1 Weight-of Evidence rating derived by CAG, based on EPA's classification system: A = proven human carcinogen; B = probable human carcinogen (B1 indicates limited evidence from human studies, B2 indicates sufficient evidence from animal studies but inadequate evidence from human studies); C = possible human carcinogen; D = not classifiable, and E = no evidence of carcinogenicity.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

TABLE 51

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

PRELIMINARY RISK SCREENING RESULTS
INTENDED FOR POLICY DEVELOPMENT

UPPER-BOUND ESTIMATES OF EXCESS
ANNUAL CANCER INCIDENCE ACROSS
FACILITIES

NITRO

Facility	UPPER-BOUND ANNUAL CASES
Artel	<.0001
Monsanto	.0003
Total	.0003

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

TABLE 52

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

PRELIMINARY RISK SCREENING RESULTS
INTENDED FOR POLICY DEVELOPMENT

UPPER-BOUND ESTIMATES OF LIFETIME
CANCER RISKS TO THE AVERAGE EXPOSED INDIVIDUAL
ACROSS COUNTY-WIDE AREA SOURCES

NITRO

AREA SOURCE	UPPER BOUNO LIFE-TIME CANCER RISK
Gas Marketing	3.4×10^{-7}
Solvent Use	1.8×10^{-6}
Heating	4.2×10^{-6}
Road Vehicles	9.8×10^{-6}
Waste Oil Burning	2.4×10^{-7}
Total	1.6×10^{-5}

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

the zone, the major facilities located within each zone, and the populations surrounding the facilities. The total population for all four zones is approximately 99,700.

- The list of pollutants considered for our analysis was provided by the West Virginia Air Pollution Control Commission's 1984 emission inventory and by EPA estimates of county-wide area sources. The 1984 emission inventory focused primarily on volatile organic compounds. The inventory surveyed all facilities within the four zones with the exception of small processing operations and blending facilities. The county-wide area source pollutants included both organic and inorganic pollutants. Our final selection of sources included seven point source facilities and five county-wide area source categories, and was based on available cancer health effects information for pollutants emitted by these sources.

- For our cancer health assessment of a select set of pollutants, we modeled pollutants that have a unit risk factor developed by EPA's Carcinogen Assessment Group. Through this selection process, 12 point source pollutants and an additional six county-wide area source pollutants were modeled. In addition to these 18 pollutants, we modeled propylene oxide and formaldehyde, two additional pollutants identified from the 1984 Emission Inventory by the West Virginia Department of Public Health for exposure analysis.

- The pollutants we have modeled do not necessarily represent the chemicals of most concern. The CAG has developed unit risk values for only a limited set of pollutants. Consequently, we have selected chemicals of known or suspected carcinogenicity for which we have adequate information to develop risk estimates. We do not know if we have modeled the pollutants posing the most risks.

- For those pollutants for which we did develop a preliminary conservative assessment of cancer effects, we examined two types of exposures: exposures in preselected neighborhoods surrounding the facilities and exposures to the average individual with each zone. The Model ISCLT provided predicted concentrations for these two exposures from point sources for the risk assessment. We also used LONGZ to incorporate turbulent intensity data collected in two of the study zones. LONGZ results are presented in Technical Appendix A. Results from our box-model analysis provided concentration estimates for our county-wide area sources.

- We were required to make several assumptions within our modeling analysis to allow us to assess exposures.

- We assumed that all residents in the study area live on the valley floor

  For the neighborhood exposures, all modeled receptors were on the valley floor and, therefore, this assumption did not affect this analysis. For exposures to the average individual, this assumption is not always true, since people do reside on the valley walls. Since the predominant releases are low level, we do not expect people on the valley walls to be exposed to higher concentrations than those on the valley floor.

  We did perform preliminary hillside receptor modeling in the Belle zone with the model LONGZ. We found that predicted concentrations were lower on the hillside than on the floor of the valley. Consequently, this assumption may lead to an over prediction of risk to the average individual.

- We assumed that there is no valley wall reflection

  For neighborhood exposures, it is unlikely that plume reflection off the valley walls will significantly affect concentrations at receptors surrounding the facilities. These neighborhoods are more affected by plumes emitted directly from the facilities, although plume reflection may affect neighborhoods further from the facilities and closer to the valley walls. For exposures to the average individual, exposure concentrations could be higher.

- For our Gaussian modeling, we assume that there is no interzone transport of pollutants

- It is unlikely that interzone transport of pollutants significantly affects concentrations within the neighborhoods surrounding the facilities. Again, these neighborhoods are much more likely affected by low level releases from the immediate facilities than facilities outside of the zone. For exposures to the average individual, interzonal transport may be an issue. For this case, we applied a box-model approach that allowed all pollutants to disperse evenly throughout the four contiguous valley zones. This analysis provides an upper-bound concentration value for these pollutants. We did not account for pollutant concentrations from sources outside of the valley.

- For exposures within the preselected neighborhoods surrounding the major facilities, we developed estimates of upper-bound, lifetime incremental cancer risk using conservative unit risk factors and assumptions of 70-year exposures from these modeled concentrations. We present a summary of the highest ranges predicted in neighborhoods in Table 53. According to our analysis, approximately 4,600

TABLE 53

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

PRELIMINARY RISK SCREENING RESULTS
INTENDED FOR POLICY DEVELOPMENT

COMPARISON ACROSS ZONES OF UPPER-BOUND
ESTIMATES OF LIFETIME, INCREMENTAL
CANCER RISK WITHIN NEIGHBORHOODS
SURROUNDING FACILITIES

ZONE	RANGE OF HIGHEST LIFETIME RISK WITHIN NEIGHBORHOODS	ESTIMATED NUMBER OF PEOPLE EXPOSED TO HIGHEST PREDICTED RISK (1980 CENSUS)	PRIMARY POLLUTANTS CONTRIBUTING TO RISK (Weight-of-Evidence)
Belle	$3x10^{-3}$	600	Chloroform (B2)
Charleston/South Charleston	1 to $5x10^{-3}$	2,700	Ethylene oxide (B1) Acrylonitrile (B1)
Institute	1 to $8x10^{-3}$	1,300	Ethylene oxide (B1) 1,3-butadiene (B2)
Nitro	1 to $6x10^{-6}$	1,453	Trichloroethylene (B2)

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

Weight-of Evidence rating derived by CAG, based on EPA's classification system: A = proven human carcinogen; B = probable human carcinogen (B1 indicates limited evidence from human studies, B2 indicates sufficient evidence from animal studies but inadequate evidence from human studies); C = possible human carcinogen; D = not classifiable, and E = no evidence of carcinogenicity.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

people may be exposed to an upper-bound, lifetime incremental risk on the order of 10^{-3}, or approximately one or more chances in a thousand of contracting cancer from a lifetime exposure to these pollutants.

- For exposures to the average individual, we developed estimates of incidence or predicted cancer cases per year using conservative unit risk factors and assumptions of 70-year exposures from these modeled concentrations. Specific conclusions for the average individual exposures follow.

- The total incidence from the pollutants across all zones is approximately seven-tenths of a cancer case per year, or more than one cancer case every two years within the study area if we assume no interzone transfer of pollutants. (See Table 54). Industrial point sources contribute almost 88 percent to the total incidence. To account for interzone transport the box model was used to develop conservative pollutant concentrations across all valley zones. Table 15 presents these estimated concentrations for the box model analysis. Table 55 presents upper-bound incidence from predicted concentrations for all studied pollutants, based on the box-model concentrations in Table 15 and the upper-bound potency factors. The predicted annual cancer incidence rate is 1.8 cases per year. Based on both modeling analyses, the predicted annual cancer incidence rate is between .7 cases (based on Gaussian model analysis) and 1.8 cases (based on box model analysis) per year.

- Table 56 presents incidence by point source pollutants across all four zones. Ethylene oxide, emitted from Institute, provides almost 56 percent of the total point source incidence rate in the study area. 1,3-Butadiene contributes almost 20 percent, acrylonitrile and chloroform each contribute approximately 10 percent. Carbon tetrachloride, methylene chloride, benzene, and vinylidene chloride contribute the remaining 5 percent to the study area incidence rate from point source pollutants.

- When we review contribution to incidence by specific facilities in Table 57, we find that almost 50 percent of the total incidence rate is contributed by Rhone Poulenc in the Institute zone and almost 30 percent is contributed by Union Carbide in South Charleston. County-wide area sources contributed more than 10 percent, while the remaining facilities, DuPont, Occidental, Union Carbide Technical center, Artel, and Monsanto together contributed more that 10 percent to the overall incidence rate.

SUMMARY TABLE 54

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

PRELIMINARY RISK SCREENING RESULTS

UPPER-BOUND ESTIMATES OF EXCESS ANNUAL CANCER INCIDENCE FOR POINT AND AREA SOURCES (AND PERCENT CONTRIBUTION TO TOTAL INCIDENCE)

BY ZONE

ZONE	POINT SOURCES	AREA SOURCES	TOTAL
Belle	.056 (7.9%)	.012 (1.7%)	.068 (9.6%)
Charleston	.211 (29.8%)	.059 (8.3%)	.270 (38.1%)
Institute	.354 (49.9%)	.015 (2.1%)	.369 (52.0%)
Nitro	.0003 (<.1%)	.002 (0.3%)	.002 (0.3%)
Total	.621 (87.6%)	.088 (12.4%)	.709 (100%)

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

TABLE 55

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

PRELIMINARY RISK SCREENING RESULTS
INTENDED FOR POLICY DEVELOPMENT

UPPER-BOUND ESTIMATES OF ANNUAL CANCER INCIDENCE
CALCULATED FROM PREDICTED BOX MODEL CONCENTRATIONS

POLLUTANT (Weight of Evidence)	INCIDENCE (Box Model)
Benzene (A)	0.041
Perchloroethylene (B2)	0.002
Methylene chloride (B2)	0.037
Trichloroethylene (B2)	0.002
1,3-Butadiene (B2)	0.438
Ethylene chloride (B2)	< 0.001
Ethylene bromide (B2)	0.001
Arsenic (A)	0.018
Benzo(a)pyrene (B2)	0.006
Cadmium (B1)	0.003
Beryllium (B2)	< 0.001
Carbon tetrachloride (B2)	0.021
chloroform (B2)	0.193
Ethylene oxide (B1)	0.839
Acrylonitrile (B1)	0.193
Allyl chloride (B2)	< 0.001
Vinylidene chloride (C)	0.001
Vinyl chloride (A)	< 0.001
Total	1.80

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

Weight-of Evidence rating derived by CAG, based on EPA's classification system: A = proven human carcinogen; B = probable human carcinogen (B1 indicates limited evidence from human studies, B2 indicates sufficient evidence from animal studies but inadequate evidence from human studies); C = possible human carcinogen; D = not classifiable, and E = no evidence of carcinogenicity.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

SUMMARY TABLE 56

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

PRELIMINARY RISK SCREENING RESULTS

UPPER-BOUND ESTIMATES OF EXCESS ANNUAL CANCER INCIDENCE FOR POINT SOURCE POLLUTANTS BY ZONE

POLLUTANT (Weight of Evidence)[1]	BELLE	CHARLESTON	INSTITUTE	NITRO	TOTAL	PERCENT CONTRIBUTION TO TOTAL POINT SOURCE INCIDENCE
Carbon Tetrachloride (B2)	.008			<.0001	.008	1.3%
Chloroform (B2)	.040		.018	<.0001	.058	9.3%
Methylene Chloride (B2)	.008	.001	.001	<.0001	.01	1.6%
Acrylonitrile (B1)		.046	.035	<.0001	.081	13.0%
Ethylene Oxide (B1)		.163	.185		.348	56.1%
Vinylidene Chloride (C)		.001			.001	.2%
Benzene (A)			<.0001	<.0001	<.0001	<.1%
1.3-Butadiene (B2)			.115		.115	18.5%
Vinyl Chloride (A)				<.0001	<.0001	<.1%
Ethylene Chloride (B)				<.0001	<.0001	<.1%
Allyl Chloride (B2)				<.0001	<.0001	<.1%
Trichloroethylene (B2)				.0003	.0003	<.1%
Total Incidence by Point Source Pollutants	.056	.211	.354	.0003	.6213	100%

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

[1] Weight-of Evidence rating derived by CAG, based on EPA's classification system: A = proven human carcinogen; B = probable human carcinogen (B1 indicates limited evidence from human studies, B2 indicates sufficient evidence from animal studies but inadequate evidence from human studies); C = possible human carcinogen; D = not classifiable, and E = no evidence of carcinogenicity.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

TABLE 57

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

PRELIMINARY RISK SCREENING RESULTS
INTENDED FOR POLICY DEVELOPMENT

UPPER-BOUND ESTIMATES OF EXCESS
CANCER INCIDENCES BY SOURCES
(AND CONTRIBUTED TO TOTAL RISK)

SOURCES	ZONES	UPPER-BOUND ANNUAL CASES	PERCENT CONTRIBUTION TO TOTAL INCIDENCE
Dupont	Belle	.001	.1%
Occidental	Belle	.055	7.8%
Union Carbide	South Charleston	.207	29.2%
Union Carbide-Tech Ctr	South Charleston	.004	.6%
Rhone Poulenc	Institute	.354	49.9%
Artel	Nitro	<.0001	< .1%
Monsanto	Nitro	.0003	< .1%
Total County-wide Area Sources		.088	12.4%
Total Incidence from Point and Area Source Pollutants		.709	100%

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

- Fugitive emissions are an important release type. Table 58 presents the percent contribution to incidence by fugitives and more nonfugitives releases for the three most important zones. In the Belle and Institute zone, fugitive emissions contribute significantly to the level of risk posed by point source pollutants emitted within the zones.

In this report we have presented a conservative assessment of the potential cancer risks which a small, select set of pollutants may pose within various populated areas of the Kanawha Valley. Again, we emphasize that our risk estimates are to be used for policy development purposes to set priorities in addressing the concerns and causes of the potential risks posed by toxic pollutants. <u>These estimates are not predictors of absolute risk posed by these pollutants within the Kanawha Valley</u>. The assumptions of the risk assessment do not allow one to make a definitive statement concerning the absolute risk posed by a particular pollutant or source. In this report we have investigated only 12 point source pollutants for their potential carcinogenic risks. We have not addressed noncancer health effects nor the effects that the remaining pollutants within the emissions inventory may pose, if any, to the population.

SUMMARY TABLE 58

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY AIR QUALITY REPORT

PRELIMINARY RISK SCREENING RESULTS

PERCENT CONTRIBUTION TO POINT SOURCE INCIDENCE BY FUGITIVE AND NON-FUGITIVE SOURCES WITHIN BELLE, CHARLESTON, AND INSTITUTE

ZONE	UPPER-BOUND ANNUAL CASES FROM POINT SOURCES	PERCENT CONTRIBUTION TO INCIDENCE WITHIN ZONE BY FUGITIVES	PERCENT CONTRIBUTION TO INCIDENCE WITHIN ZONE BY NON-FUGITIVES
Belle	.056	84.6%	15.3%
Charleston	.211	36.6%	63.2%
Institute	.354	70.2%	29.7%

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND POLLUTANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER: IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA, 1987

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KANAWHA VALLEY AIR QUALITY REPORT

Technical Appendices

(Copies available upon request)

Appendix	Title
A	Interpretation of LONGZ Model Output
A.1	- LONGZ Model Results (all sources combined)
A.2	- LONGZ Model Results (Point and Area Source breakdown)
A.3	- Population-Weighted Concentration by Pollutant (Main Grid) for LONGZ
B	Meteorological Monitoring Program
B.1	- Detailed Description of Meteorological Monitoring Program
B.2	- Rationale for the Selection of 20 m as the Preferred Monitoring Height for Wind Data
B.3	- Specifications for Meteorological Monitoring Systems
B.3.1	- Specifications for Climatronics F460 and R.M. Young Propeller Anemometer Wind Systems
B.4	- Description of Data Recovery Rates and Conformance to Data Quality Objective
B.5	- Monthly Wind Rose Data for all Four Zones
B.6	- Tables of Standard Deviation of Horizonta! and Vertical Wind Direction
C	Kanawha Valley Meteorological Monitoring/ Dispersion Modeling Protocol
D	County-wide Area Source Emission Estimates
E	Input Data Files for ISCLT Dispersion Modeling Analysis
F	Comparison of Box Model Methodology with Gaussian Modeling
G	Summary of Archived Data (SAROAD)

H	Results of Ambient Air Quality Monitoring During the Toxics Screening Study
H.1	- Results of EPA Region III Monitoring (December 9-12, 1985)
H.2	- Results of West Virginia Air Pollution Control Commission Monitoring (December 6-11, 1985)
H.3	- Results of EPA Office of Research and Development Monitoring (March 4, 1986 through April 12, 1986)
I	Preliminary Risk Estimates for Formaldehyde and Propylene Oxide
J	Fugitive Emissions Estimates for Primary Pollutants at Neighborhood Sites

Chapter Five

Drinking Water Analysis

KANAWHA VALLEY DRINKING WATER REPORT

I. PURPOSE AND INTRODUCTION

The Kanawha Valley Toxics Screening Study has four stated objectives that have provided overall direction for this technical report. In this drinking water analysis, we attempted to address these objectives. The four objectives are:

1. Using available data bases, identify many of the chemicals routinely released or present within various exposure pathways;

2. Develop a sense of the potential public health concerns these toxic pollutants may pose in various exposure pathways, based on health effects and exposure information;

3. For a select number of chemicals, provide an initial conservative assessment of potential cancer risk and potential noncancer health risk for predicted for observed concentrations within exposure pathways; and

4. Identify data and information gaps and outline needs and options for future study directions to enable a more detailed investigation of health issues where warranted.

To meet the first objective, we reviewed available monitoring data from community water supply systems to determine pollutants present within these systems. We relied solely on monitoring information required by federal regulations, and only for public water supply systems. Due to this limited set of pollutants which are monitored by the State, we may have underestimated health risks from drinking water since we did not account for all the potential pollutants. For a small set of pollutants detected within the water supply systems, trihalomethanes, we developed a conservative assessment of potential cancer risk. For all monitored pollutants for which we have health effects information, we assessed the potential for noncancer health effects. Finally, we outlined several datagaps which limit our ability to draw additional conclusions of possible health effects posed by pollutants in the drinking water systems within the Kanawha Valley.

This report is divided into nine sections. The first two sections outline the general methodology and the health effects analyses used within the report. Section III and IV provide descriptions of the general study area and the monitoring requirements for the drinking water systems. Section V outlines our selection of pollutants for the exposure and health assessment. In Section VI we develop exposure concentrations for the selected pollutants. Section VII presents health effects information for the pollutants studied. In Section VIII we present the results of our risk calculations for a small set of pollutants within the drinking water systems. Section IX presents our summary and conclusions.

II. DESCRIPTION OF THE GENERAL METHODOLOGY

In this section we briefly outline the methods used to estimate exposures to a limited set of toxic contaminants in the public water supply systems and describe their potential health effects from these exposures. We combine estimates of human exposure levels to these chemicals with information on contaminant toxicity to estimate potential excess lifetime average individual risks and excess cancer incidence (i.e., risk to the population). We also estimate the number of people exposed to the potential increased risk of noncancer health effects.

We have reviewed data from public water supply systems within the Kanawha Valley study area. These systems serve the majority of the population within the study area. The State does not collect water quality information for toxics for the other two systems: non-community water supply systems and private wells.

For the public water supply systems, we reviewed pollutants regulated by the Safe Drinking Water Act. The Safe Drinking Water Act requires public water supply systems to monitor regularly for designated drinking water contaminants. These pollutants cannot exceed Maximum Contaminant Levels (MCLs) determined by EPA.

Although we only developed potential exposure concentrations for MCL pollutants, we did review available priority pollutant scans of the major public water system (the West Virginia Water

Company) which serves more than 78% of the public water supply system population to determine the potential for other pollutant exposures. These priority pollutant scans are in Technical Appendix A. According to Technical Appendix A, the priority polluant scans did not detect priority pollutants except for several trihalomethanes during the monitoring.

From monitoring data, we analyzed possible exposure of the population to disinfection products, inorganics, and pesticides in the drinking water. These are the classes of MCL pollutants monitored by the West Virginia Department of Health.

For disinfection products (trihalomethanes) formed from the chlorination of drinking water, we developed average concentration values based on monitoring data over a three-year period. For these average concentrations, we assumed standard EPA exposure assumptions (a 70-kg adult consumes two liters of water per day over a 70-year period) to develop exposure concentrations. We then multiplied upper-bound cancer unit risk values, developed by EPA's Carcinogenic Assessment Group (CAG), by the exposure concentrations to obtain lifetime, incremental individual cancer risk for trihalomethanes.

Due to the limited monitoring information for metals and pesticides, we did not develop average system concentrations for these pollutants, and did not assess potential lifetime excess average individual cancer risk for inorganics nor pesticides. Many observations for these pollutants were below the detection limit.

For noncancer health effects associated with exposure to trihalomethanes, we compared our estimated average concentrations to EPA developed Reference Dose (RfD) criteria to determine if such criteria are exceeded. For metals and pesticides, we compared the RfDs to the highest monitored value. Again, many of these values are below the detection limit.

Health Assessments

Cancer Assessment: This study uses a risk assessment screening methodology to evaluate and compare, in a very limited fashion, the potential health risks from exposure to a limited set of toxic pollutants. Risk to an individual is defined as the increased probability that an individual exposed to one or more chemicals will experience a particular adverse health effect during his or her lifetime. Several measures of carcinogenic risk are used in these analyses, including estimated risk to an individual and projected risk to the entire population (i.e., incidence).

This risk screening methodology involves both a qualitative and quantitative assessment of the potential carcinogenicity of the selected pollutants. As a screening study, this

analysis employs both types of assessments. The Carcinogen Assessment Group (CAG) reviews the evidence of carcinogenicity for selected pollutants, and classifies pollutants as human carcinogens (Group A), probable human carcinogens (Group B), possible human carcinogens (Group C), not classified as carcinogens due to inadequate evidence (Group D), and not carcinogenic to humans (Group E). These classifications accompany all quantitative risk estimates within this study.

For those chemicals in groups A, B, and C, CAG provides quantitative, upper-bound estimates of carcinogenic unit risk factors. A unit risk factor allows the calculation of the estimated individual risk posed by exposure to a chemical given certain exposure assumptions. To calculate individual risk, a chemical's unit risk factor is multiplied by the estimated concentration of the pollutant.

Individual Lifetime Risk = Unit Risk Factor $(ug/l)^{-1}$ x Concentration (ug/l)

The concentration is simply the concentration of the chemical found in the drinking water. The unit risk factor assumes that an average person weighs 70 kg and drinks 2 liters of water each day (2 l/day).

SAMPLE CALCULATION

POSSIBLE RISK OF CANCER ASSOCIATED WITH INGESTION OF CHLOROFORM IN DRINKING WATER

Estimated Average Individual Lifetime Risk	=	Unit Risk Factor x Concentration
Cancer Unit Risk Factor for the Ingestion of Chloroform	=	2.3×10^{-6} $(ug/l)^{-1}$
Concentration	=	For this example assume 1 ug/l of chloroform in the drinking water.
Esimated Average Individual lifetime Risk	=	2.3×10^{-6} $(ug/l)^{-1}$ x 1 ug/l
	=	2.3×10^{-6}

This estimated average individual risk, based on upper-bound unit risk factors, indicates that conservatively, there is approximately a two in a million increased chance of developing cancer over a 70-year lifetime, constant exposure to this concentration of chloroform. The actual risk could be considerably lower.

For the disinfection products such as trihalomethanes, we present risks to the average individual based on average concentrations monitored by the State. Incidence, another measure of risk, is the probable number of cancer cases expected over a 70-year period when a population is exposed to the average individual exposure concentration. Incidence is calculated by multiplying individual risk by the population number and can be annualized by dividing by 70.

The overall uncertainty in the risk assessment assumptions is great enough that results should be considered rough indicators of the probable magnitude of the effects, not as precise, site-specific prediction of effects. Furthermore, EPA policy dictates that the most conservative approach be used in developing unit risk factors. Consequently, EPA develops estimates that are generally unlikely to underestimate the true potency of the chemical. Indeed, in many cases the lower-bound estimate for a chemical may be zero. It is important to remember the conservative nature of our risk estimates and the limitations of these values throughout the study.

Noncancer Health Effects: No currently accepted techniques exist for estimating the probability or incidence of noncancer effects. Therefore, in evaluating the potential noncancer health risks within this study, we rely on benchmark values or Reference Doses (RfDs), expressed in units of mg/kg/day, which represent dose levels below which adverse health effects are not likely to occur in most people. Unlike cancer effects, noncancer health effects are assumed to be threshold events. This threshold assumption means that noncancer health effects are assumed more likely to occur above these exposure concentrations. Exposures that are less than the RfD are not likely associated with noncancer health effects and therefore are less likely to be of regulatory concern.

In our analyses, we modify the RfDs to determine safe ingestion exposure concentrations for a pollutant through ingestion of drinking water. (In our analysis, we assumed the only exposure route for the studied pollutant is drinking water ingestion. In fact, all exposure routes must be considered when reviewing RfD levels.) To determine safe exposure concentrations, the RfD is converted to an exposure concentration in units of ug/liter. This exposure concentration implies several assumptions. Again, we assume that the average individual weighs 70 kg and drinks 2 liters of water a day, and the average individual is exposed to these concentrations for 70 years. Once developed, these criteria are then compared to ambient pollutant concentrations. When ambient pollutant concentrations exceed the RfD-based criteria, there is an increased probability of a noncancer effect occuring within the exposed population. Currently we cannot quantify the increased probability, but only note that there is an increased probability when the criteria are exceeded.

III. DESCRIPTION OF STUDY AREA

The Kanawha Valley study area contains three types of public water supply systems: private wells, non-community water supply systems, and community water supply systems. The community water supply systems are the predominant water supply with 14 such systems serving households within the study areas. TABLE 1 lists the community water supply systems, the population served by each system, and their water sources. Map 1 outlines the boundaries of these systems. (Map boundaries are numbered in sequence according to TABLE 1.)

TABLE 1

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY DRINKING WATER REPORT

COMMUNITY WATER SYSTEM, POPULATIONS, SOURCES
WITHIN STUDY AREA

MAP KEY	SYSTEM	POPULATION SERVED	WATER SOURCE
1.	Kanawha Falls Public Service District	3,885	Kanawha River
2.	Amstrong Public Service District	2,125	Armstrong Creek
3.	Smithers Public Water Supply	2,500	Mine Impoundments/Wells
4.	West Virginia Water Company (Montgomery District)	4,907	Kanawha River
5.	Pratt Crown Hill Public Water Supply	2,300	Kanawha River
6.	Cedar Grove Water Company	3,600	Kanawha River
7.	West Virginia Water Company (Kanawha Valley District)	190,000	Elk River
8.	Washington Public Service District	1,500	Coal River
9.	St. Albans Public Water Supply	16,200	Coal River
10.	Sissonville Public Water System	4,000	Pocatalico River
11.	North Putnam Water Supply	2,600	West Virginia Water Company (Kanawha Valley District)
12.	Kanawha Orchard Public Service District	525	South Putnam Public Service District
13.	South Putnman Public Service District	4,900	Surface Impoundments
14.	Winfield Public Water Supply	1,500	Surface Impoundments
	Total:	240,542	

Source: Department of Health, West Virginia, 1987

By comparison, the other two systems, private wells, and non-community water suppy systems, serve a much smaller population. While it is difficult to estimate the exact proportion of these users to the number of persons on public water systems, estimates from the Charleston Planning and Development Commission suggest that there may be approximately 22,000 private wells in Kanawha County (Personal Communication, McQueen, 1987). It would be difficult to assess the quality of the water in these supplies since private wells are not required to monitor for water quality in Kanawha Valley and hence very little monitoring data exists.

Non-community water supply systems serve transient or intermittent users and can include facilities such as campgrounds, parks, schools, gas stations, and motels which have their own water supply systems. Such systems monitor for coliform bacteria, turbidity, and nitrate.

For this analysis we examined only the public water supply systems. Again, we are limited to available monitoring data for selected toxics within the public water supply systems. Although the public water supply systems serve most of the population within the study area, individual wells and non-community water systems with wells may have toxic exposure concentrations through contamination of the groundwater supply from hazardous waste sites within the study area. Contamination of wells has been detected within the valley; Superfund Preliminary Assessments have detected well contamination from Heizer Creek Dump and South Charleston Municipal Landfill (Draft Site Inspection Report, NUS Corp., 1984). The lack of exposure information for private wells serving households and non-community water systems is a limitation to this drinking water analysis. Although the number of residents obtaining drinking water from private wells is relatively small, private wells and non-community water supply systems could be a source of toxic exposure to individuals.

IV. REGULATORY OVERVIEW OF PUBLIC COMMUNITY WATER SYSTEMS

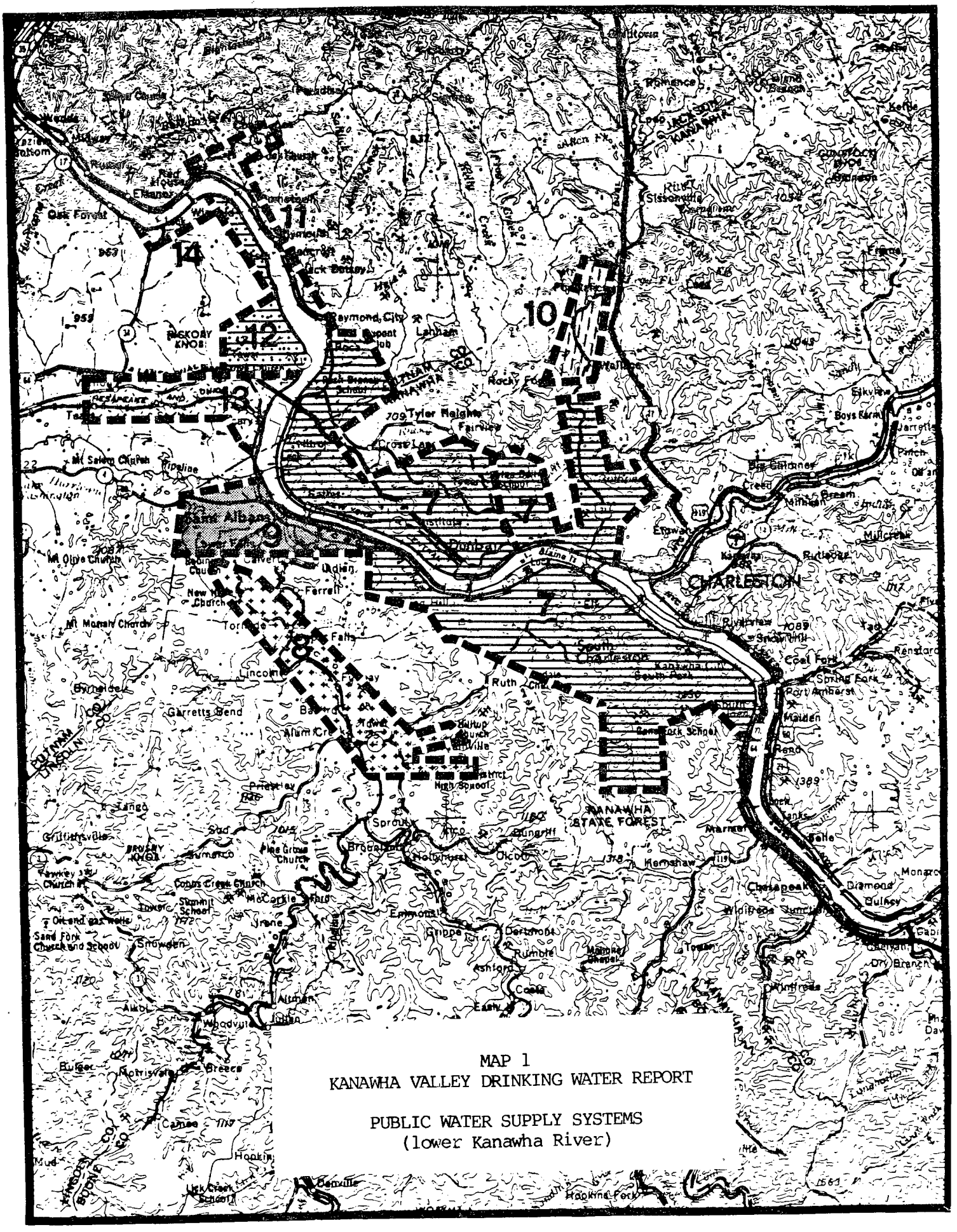
The National Interim Primary Drinking Water Regulations, established under the Safe Drinking Water Act, set Maximum Contaminant Levels (MCLs) and monitoring requirements for community water supplies. These MCLs include inorganic chemicals, three organic chemical groups (chlorinated hydrocarbon pesticides, chlorophenoxy pesticides, and trihalomethanes) radionuclides, microbes, and turbidity.

The State is responsible for monitoring MCL pollutants. Inorganics are monitored once a year. Additional monitoring occurs if contamination is believed to have occurred. Pesticides are also monitored once a year during the spring or fall season when contamination, usually from run-off, may occur.

MAP 1
KANAWHA VALLEY DRINKING WATER REPORT

PUBLIC WATER SUPPLY SYSTEMS
(upper Kanawha River)

CHARLESTON
South Charleston
Kanawha City
South Park
South Marmet
KANAWHA STATE FOREST
Coalburg
Boomer
Cedar Grove
Montgomery
Smithers
Chesapeake
Gauley Bridge
Kanawha Falls
Boomsboro
COTTON HILL
Nitro
Dunbar
Institute
Alum Creek
Sissonville
Pinnacle Creek
Elkview
Pratt
Clendenin
Dixie
Belle
Marmet
Ward
Mount Carbon
Eagle
Bonwood
Glasgow
Cross Lanes
Rand
Kenna
Ripley
Spencer
Pond Fork
Pond Gap
Blakeley
Hilton
Bentree
Sissonville
Elkhorn
Poca
Dunbar
Institute
Alum Creek
Nitro
Cross Lanes
Rand
Kenna
Ripley
Spencer
Pond Fork
Pond Gap
Blakeley
Hilton
Bentree
Dixie
Belle
Marmet
Ward
Mount Carbon
Eagle
Bonwood
Glasgow
Gauley Bridge
Kanawha Falls
Boomsboro
COTTON HILL
Nitro
Dunbar
Institute
Alum Creek
Sissonville
Elkhorn
Poca
Clendenin


14
11
12
13
10
9
8
7
6
Raymond City
Lanham
Tyler Heights
Dunbar
South Charleston
CHARLESTON
Institute
Ruth
Alum Creek
Sissonville
Elkview
Clendenin
Marmet
Chesapeake
Belle
Nitro
Cross Lanes
Hurricane
Teays
Winfield
Poca
Scott Depot
Hometown
Ashford
Rumble
Cross Roads
Dry Branch
Sandy Creek
Big Coal
Kanawha City
Spring Fork
Coal Fork
Rand
Malden
Dickinson
Ward
Sharon
Elkhorn
Pratt
Alum Creek
Sissonville
Ripley
Gallipolis
Point Pleasant
Mason
Leon
Winfield
Buffalo

Community water systems which serve a population of 10,000 or more individuals and which use chlorine to disinfect drinking water must also monitor for trihalomethanes. Systems must monitor at least four times a year. The MCL level pertains to the total trihalomethane concentration, the sum of the concentrations of chloroform, bromoform, bromodichloromethane, and chlorodibromomethane.

V. POLLUTANT SELECTION

We analyzed MCL pollutants for exposure concentrations determined from monitoring information provided by the State. These pollutants and their sources are reviewed below.

Disinfection Agents and By-products

Disinfection is a process in which water is treated with chemical agents, most commonly chlorine, to kill microbial pathogens (disease causing agents). Without disinfection, people could be exposed to numerous infectious agents through their drinking water and periodic outbreaks of diseases such as cholera or typhoid could result. Disinfection techniques commonly maintain a residual disinfecting agent (chlorine if the disinfection process is chlorination) in the treated water, to continue disinfecting water until it is used. Unfortunately, potentially harmful by-products such as trihalomethanes result from the reaction of chlorine with organic matter (such as plant and animal matter) in the water.

We have analyzed the potential effects of chlorination. Organic chemicals of concern created through this process include the trihalomethanes:

- chloroform;
- bromoform;
- chlorodibromomethane;
- dichlorobromomethane.

Inorganics

A number of inorganic substances, primarily metals and other minerals, are found in drinking water. Most of these substances are probably from natural background sources (i.e., they are present in soils and are picked up by water traveling through those soils). Some of these chemicals may be the result of man-made contamination, but, for most substances, this is difficult to determine and specific incidents that cause the contamination cannot be identified.

Metals and minerals for which we have reviewed available monitoring data include:

- arsenic;
- barium;
- cadmium;
- chromium;
- lead;
- mercury;
- selenium;
- silver; and
- zinc;

Pesticides

Pesticides could contaminate imported surface water primarily through runoff from agricultural areas through which the water travels; such contamination would not necessarily have to occur in or near Kanawha Valley to affect local water. Pesticides from local sources could cause ground-water contamination. The State monitors for the following pesticides:

- Endrin;
- Lindane;
- Methoxychlor;
- Toxaphene;
- 2,4-D;
- Silvex (2,4,5-TP).

VI. EXPOSURE

Disinfection Agents and By-products

TABLE 2 presents average concentrations for monitored trihalomethanes within three community water supply systems: Saint Albans, West Virginia Water Company (Kanawha Valley District), and the West Virginia Water Company (Montgomery District) Federal regulations require these three systems to monitor for trihalomethanes on a regular basis. Although the West Virginia Water Company (Montgomery District) serves less than 10,000, the system monitors for trihalomethanes as part of the larger West Virginia Water Company. These monitoring data are limited and subject to significant uncertainties introduced by seasonal and sampling location. Seasonal variations are due mainly to the variations in organic matter in the raw water and the effects of temperature on the formation of trihalomethanes. As the distance from the chlorination point increases, the reaction time and, therefore trihalomethane formation, also increases giving rise to the sampling point variations. EPA's Office of Water in their water quality programs does not attempt to quantify this variation, but rather accounts for the

TABLE 2

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY DRINKING WATER REPORT
PRELIMINARY RISK SCREENINNNG RESULTS

ESTIMATES OF AVERAGE LEVELS OF INDIVIDUAL TRIHALOMETHANES
IN COMMUNITY WATER SUPPLY SYSTEMS BASED ON SIXTY OBSERVATIONS

		UG/L				
SOURCE	POPULATION	CHLOROFORM	DICHLOROBRO-MOMETHANE	CHLORODIBRO-MOMETHANE	BROMOFORM	THM TOTAL
Saint Albans[1]	16,200	29	12	1	<1	42
West Virginia Water Company[2] (Kanawha Valley District)	192,600	72	4	1	<1	77
West Virginia Water Company (Montgomery District)[1]	4,907	50	4	<1	<1	54
Small Systems[3]	26,835					42-77

[1]Estimates for Saint Albans and West Virginia Water Company (Montgomery District) are based on data averaged from 1983 to October 1, 1986.

[2]Estimates for the West Virginia Water Company are averaged with data from 1984-October 1, 1986. The West-Virginia Water Company includes North Putnam Water Public Water Supply System.

[3]We applied a range of the lowest and highest observed THM concentration within the study area to the smaller systems. The lowest observed average THM concentration is 42 ug/l (Saint Albans), the highest concentration is 77 ug/l (West Virginia Water Company, Kanawha Valley District).

Source: Department of Health, West Virginia, 1987

the variation by averaging quarterly monitoring samples from four different points within the delivery system.

Other small systems within the Kanawha Valley do not monitor for trihalomethanes although such systems chlorinate the supply systems (personal communication, Hodges, 1986). To estimate trihalomethane totals for these systems, we assume a range of trihalomethane concentrations for these systems. For these smaller systems we used the lowest observed concentration of trihalomethanes, which was 42 ug/l (Saint Albans Water District) and the highest observed concentration of trihalomethanes which was 77 ug/l (West Virginia Water Company, Kanawha Valley District). Based on these observations, we assigned a range of 42 to 77 ug/l for the smaller systems.

It is important to note that these systems are beneath the MCL requirement for total trihalomethanes (100 ug/l), in some cases by more than half.

Inorganics and Pesticides

TABLE 3 presents the highest observed concentrations for metals, minerals, and pesticides regulated by the Primary Drinking Water Regulations. Inorganics and pesticide concentrations come from a more limited data set than the trihalomethanes; Federal regulations require only a yearly measurement for these pollutants. To obtain a larger data set, we reviewed monitoring data from 1981 to 1985.

VII. TOXICOLOGY EVIDENCE

Cancer Effects

TABLE 4 and TABLE 5 summarize the available evidence on the chronic toxicity of those substances for which we reviewed monitored concentrations. TABLE 4 presents the estimates of the carcinogenic potential and unit risk factors for disinfection agents and by-products, and inorganics and pesticides. Evidence of the carcinogenicity of trihalomethanes is strongest for chloroform. EPA classifies it as a probable carcinogen (B2), and has calculated a unit risk factor for it. Experiments indicate that chlorodibromomethane and dichlorobromomethane may also be carcinogens (NTP, 1984), and many toxicologists suspect that bromoform may also be carcinogenic. Since EPA's Carcinogen Assessment Group (CAG) has not developed a potency evaluation for these chemicals, we analyzed risks from total trihalomethanes (THMs) as if all THMs are as potent as chloroform.

TABLE 3

KANAWHA VALLEY TOXICS SCREENING STUDY
KANAWHA VALLEY DRINKING WATER REPORT

WATER QUALITY DATA

HIGHEST OBSERVED CONCENTRATION OF PESTICIDES IN TREATED WATER (1981-1985)[1]
(µg/l)

	Maximum Contaminant Level	Armstrong	Winfield	S. Putnam	Sissonville	Washington PSD	Kanawha Falls	Pratt	Cedar Grove	WV Water Co.	WV/Montgomery	St Albans	Smither:
rsenic	50	<1	<1	<1	10	<1	5	10	10	<1	<1	10	<1
arium	1000	9	10	100	90	7	<1	40	40	<1	<1	40	<1
admium	10	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	0.2	<0.1
hromium	50	<1	<1	<1	<1	<1	<1	<1	<1	2	<1	<1	<1
ead	50	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	1	<1
ercury	2	<.2	<.2	<.2	<.2	<.2	<.2	<.2	<.2	0.7	<.2	<.2	<.2
elenium	10	<2	<2	<2	<2	<2	6	<2	<2	<2	<2	<2	5
ilver	50	<.2	<.2	<.2	<.2	<.2	<.2	<.2	<.2	<.2	<.2	<.2	<.2
inc		NA	NA	NA	NA	NA	<5	NA	NA	<5	10	<5	NA
ndrin	0.2	<.01	<.01	<.01	<.01	<.01	<.01	<.01	<.01	<.01	<.01	<.01	NA
indane	4	<.005	<.005	<.005	<.005	<.005	<.005	<.004	<.004	<.004	<.004	<.005	NA
ethoxychlor	100	<.03	<.03	<.03	<.03	<.03	<.03	<.03	<.03	<.03	<.03	<.03	NA
oxaphene	5	<.2	<.2	<.2	<.2	<.2	<.2	<.2	<.2	<.2	<.2	<.2	NA
ilvex	10	<.04	<.04	<.04	<.04	<.04	<.04	<.04	<.04	<.04	<.04	<.04	NA
,4 D	100	<.2	<.2	<.2	<.2	<.2	<2	<2	<2	<2	<2	<2	NA

[1]The "LESS THAN" Sign (<) indicates a concentration below the detection limit.

Source: Department of Public Health, West Virginia, 1987.

TABLE 4

KANAWAH VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY DRINKING WATER REPORT
PRELIMINARY RISK SCREENING RESULTS

UPPER-BOUND CANCER UNIT RISK VALUES:
POLLUTANTS IN DRINKING WATER

POLLUTANT	UNIT RISK FACTOR INGESTION $(ug/l)^{-1}$	WEIGHT-OF-EVIDENCE[1]
Chloroform (total trihalomethane	2.3×10^{-6}	B2
Arsenic	4.5×10^{-4}	A
Lindane	3.1×10^{-5}	B2-C
Toxaphene	3.2×10^{-5}	B2

[1] Weight-of-Evidence rating derived by CAG, based on EPA's classification system: A = proven human carcinogen; B = probable human carcinogen (B1 indicates limited evidence from human studies, B2 indicates sufficient evidence from anumal studies but inadequate evidence from human studies); C = possible human carcinogen; D = not classifiable, and E = no evidence of carcinogenicity.

Source: Carcinogen Assessment Group, 1986.

Among the inorganics and pesticides, arsenic, lindane, and toxaphene have been found by EPA's CAG to be potentially carcinogenic. (EPA considers cadmium and hexavalent chromium carcinogenic via inhalation of air, but not via ingestion from drinking water). The evidence for arsenic's carcinogenicity by ingestion is epidemiologic evidence correlating high drinking water arsenic levels with a form of skin cancer which is rarely fatal. However, the carcinogenicity of arsenic ingested through drinking water is the subject of some dispute. Some scientists believe that arsenic may not be harmful, and may even be beneficial at low levels. EPA's Office of Drinking Water has concluded that arsenic is not harmful at low levels, and proposed a Maximum Contaminate Level (MCL) of 50 micrograms per liter.

Noncancer Effects

The estimated toxicity of water disinfection agents and by-products for effects other than cancer is summarized in TABLE 5. We present benchmark criteria based on Reference Doses, below which exposure is not considered likely to develop adverse noncancer health effects within the general population over a lifetime.

Strong scientific evidence exists that airborne lead can have adverse health effects, especially for children. Waterborne lead has also been shown to have adverse health effects. The health effects of lead are documented in Volume IV, 1986 "Air Quality Criteria for Lead". Evaluating effects from chronic lead exposure is a complex issue involving evaluation of the total body burden from air, water, and dust exposure and is the subject of current EPA research. No EPA reference dose has yet been estimated. Because of the issue's complexity, the ongoing Federal initiatives, and the lack of an estimated no-effect threshold, we have not estimated health effects from lead exposure. All lead concentrations are below the detection limit in our dataset.

Cadmium, mercury, silver, hexavalent chromium, and selenium may pose risks of noncancer effects at low levels. Other substances, such as barium and zinc, are quite a bit less toxic. Zinc, is considered beneficial in drinking water at some concentrations but harmful at higher concentrations.

VIII. RISK ESTIMATES

Disinfection By-products

We have calculated individual lifetime risks, based on upper-bound unit risk factors and aggregate incidence for trihalomethanes exposure by assuming that all trihalomethanes have the same carcinogenic potency as chloroform. Structural similarities and recent studies (NTP, 1984) suggest that all four may eventually be

TABLE 5

KANAWHA VALLEY DRINKING WATER REPORT

NONCANCER HEALTH EFFECTS
POLLUTANTS IN DRINKING WATER

Pollutant	Health Effect	Reference Dose[1] (µg/l)	Study RfD Based On
Chloroform	liver cysts	350.0	Heywood et al. (1979)
Bromoform	hepatic lesions	210.0	Chu et al. (1982)
Bromodichloromethane	hepatic lesions	17.5	Chu et al. (1982)
Dibromochloromethane	hepatic lesions	21.0	Chu et al. (1982)
Barium	increased blood pressure	1,750.0	Perry et al. (1983)
Chromium - III	none observed	35,000.0	Ivankovic and Preussmann (1975)
Chromium - VI	none observed	175.0	MacKenzie et al. (1958)
Mercury (inorganic)	renal and kidney damage	70.0	Fitzhugh et al. (1950)
Methyl Mercury	CNS effects	1.0	Clarkson et al. (1973)
Silver	argyria	105.0	Gaul and Staud (1935) Blumberg and Carey (1934) East et al. (1980)
Fluoride	dental fluorosis	2,100.0	Underwood (1977)
2,4-D	liver effects	350.0	Hazelton Labs (1983)

[1]EPA/RfD indicates a benchmark value derived from no-effect threshold value. Exposures greater than the RfD are assumed to be associated with an increase in noncancer health effects. EPA reviews and verifies these thresholds internally through the Reference Dose Workgroup.

found carcinogenic. EPA has used this approach in the past in developing drinking water regulations. TABLE 6 presents our estimates of average individual risks from trihalomethanes, and TABLE 7 presents our estimates of increased incidence for each source of community water supplies.

We estimated that the upper-bound increased lifetime risk of cancer for average exposed individual drinking water is approximately one to two chances in ten thousand. This level is comparable to what we have seen in several other cities. For the entire study area, we project an upper-bound increased incidence of more than one-half cases per year for a 70-year exposure of this concentration level of trihalomethanes.

In order to compare estimates of increased incidence for individuals drinking public water supplies in the valley directly to the increased incidence caused by the inhalation of selected point source air emissions, we calculated the water related incidence within the smaller study area of the air analysis by multiplying the average individual risk involved with drinking water from the West Virginia Supply Company ($2x10^{-4}$) by the populations in each of the four separate air zones defined in the "Kanawha Valley Air Quality Report" (Belle, Institute, Charleston, and Nitro) and divided by 70. The total population of these four zones is 99,660. We estimate that 93% of the population in the four air zones is served by the West Virginia Water Company (100% in Belle and Charleston, 85% in Institute, and 60% in Nitro). Nitro and Institute are also served by South Putnam PSD, Kanawha Orchard PSD, and Saint Albans Public Water Supply. By these assumptions the incidence related to trihalomethane concentrations in these four zones is approximately .3 cases per year.

TABLE 8 compares the highest average levels of disinfection-related contaminants found in drinking water with the estimated reference dose criteria for chronic health effects other than cancer. Unlike cancer, exposure to a contaminant at a dose below the estimated RfD are likely not associated with noncancer health effects. All trihalomethane levels appear to be below estimated thresholds for noncancer effects.

Measured trihalomethane exposures are comparable with those in other areas where chlorinated surface water is supplied. TABLE 9 presents information on chloroform levels in some other cities.

Inorganics and Pesticides

CAG has developed potency values for arsenic, lindane, and toxaphene. Arsenic is rated a human carcinogen (Group A), while toxaphene is considered a probable carcinogen (Group B2). Lindane is both B2 and C according to the CAG although the Office of Water within EPA considers lindane in category C. All measured concentrations for lindane and toxaphene are below the detection limit and no risk calculations are provided for these pollutants.

TABLE 6

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY DRINKING WATER REPORT
PRELIMINARY RISK SCREENING RESULTS

UPPER-BOUND ESTIMATES OF
LIFETIME CANCER RISKS TO THE AVERAGE EXPOSED INDIVIDUAL

INTENDED FOR POLICY DEVELOPMENT

SYSTEM	ESTIMATED RISK	WEIGHT OF EVIDENCE
Saint Albans	1×10^{-4}	B2[2]
West Virginia Water Company (Kanawha Valley District)	2×10^{-4}	B2
West Virginia Water Company (Montgomery District)	1×10^{-4}	B2
Smaller Systems	$1 \times 10^{-4} - 2 \times 10^{-4}$	B2

[1]The unit risk factors used in this analysis are based on conservative assumptions that generally produce upper-bound estimates. Because of limitations in data and methods in several areas of the analysis, such as exposure calculations and pollutant selection, risk estimates were calculated as aids to policy development, not as predictions of actual cancer risks in the Kanawha Valley. Actual risks may be significantly lower; in fact, they could be zero. The proper function of the estimates is to help local officials select and evaluate issues and set priorities for the topics examined.

[2]EPA considered chloroform a Class B2, or probable human carcinogen. EPA has not classified the other THMs for carcinogencity. This analysis assumes similar unit risk factors for other THMs as chloroform.

Source: Regulatory Integration Division, 1987
EPA, Office of Policy Analysis

TABLE 7

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY DRINKING WATER REPORT
PRELIMINARY RISK SCREENING RESULTS

UPPER-BOUND ESTIMATES
OF ANNUAL EXCESS CANCER
INCIDENCE: TRIHALOMETHANES
IN DRINKING WATER

INTENDED FOR POLICY DEVELOPMENT

SYSTEM	ESTIMATED ANNUAL EXCESS[1] INCIDENCE (cases/year)	WEIGHT OF EVIDENCE
Saint Albans	.02	B2[2]
West Virginia Water Company	.55	B2
West Virginia Water Company (Montgomery District)	.01	B2
Smaller Systems[3]	.04 - .08	B2
TOTAL	.62 - .66	B2

Source: EPA, Office of Policy Analysis, Regulatory Integration Divison, November 1986.

[1]The unit risk factors used in this analysis are based on conservative assumptions that generally produce upper-bound estimates. Because of limitations in data and methods in several areas of the analysis, such as exposure calculations and pollutant selection, risk estimates were calculated as aids to policy development, not as predictions of actual cancer risks in the Kanawha Valley. Actual risks may be significantly lower; in fact, they could be zero. The proper function of the estimates is to help local officials select and evaluate issues and set priorities for the topics examined.

[2]EPA considers chloroform a Class B2, or probable, human carcinogen. EPA has not classified the other THM for carcinogenicity.

[3]Data on levels of THMs is not available. This analysis assumes that the THM concentration for smaller systems could be from 42 to 77 ug/l.

TABLE 8

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY DRINKING WATER REPORT
PRELIMINARY RISK SCREENING RESULTS

COMPARISON OF TRIHALOMETHANE REFERENCE DOSE CRITERIA
TO HIGHEST ESTIMATED AVERAGE CONCENTRATIONS

TRIHALOMETHANE	HIGHEST CONCENTRATION (ug/l)	REFERENCE DOSE CRITERIA (ug/l)
Chloroform	72 (West Virginia Water Co.)	350
Bromoform	<1 (all monitored systems)	210
Dichlorobromomethane	12 (Saint Albans)	17.5
Chlorodibromomethane	1 (Saint Albans, West Virginia Water Co.)	21

Source: Regulatory Integration Division, Office of Policy Analysis, 1987

TABLE 9

KANAWHA VALLEY TOXICS SCREENING STUDY

KANAWHA VALLEY DRINKING WATER REPORT

AVERAGE CHLOROFORM LEVELS IN DRINKING WATER
FOR SELECTED CITIES

(population >500,000) [1]

City	Average ug/l	Population 1980
Boston, MA	3.7	562,994
Chicago, IL	14.5	3,005,072
Cleveland, OH	15.0	573,822
Columbus, OH	171.0	564,871
Dallas, TX	18.0	904,078
Detroit, MI	10.5	1,203,339
Houston, TX	123.0	1,595,138
Los Angeles, CA	32.0	2,966,850
Memphis, TN	2.5	646,335
Milwaukee, WI	8.9	636,212
New York, NY	22.0	7,071,639
Philadelphia, PA[2]	47.7	1,656,300
San Antonio, TX	0.1	785,880
San Diego, CA	43.5	875,538
San Francisco, CA	58.5	678,974
Washington, DC	47.0	638,333

[1]These cities were selected from a sample of 137 cities studied in the source document.

[2]Reported chloroform concentrations are an average of the levels at Baxter, Queen Lane, Belmont plants. Populations data as of July 1983. See U.S. EPA, Final Draft Report of the Philadelphia Integrated Environmental Management Project, June 1986.

Source: U.S. EPA, Office of Standards and Regulations, Chemical Control Options Analysis for Chloroform, (Contract Nos. 68-02-3168, 68-01-6775, and 68-01-6715), December 1984.

We did review the detection limit for these chemicals to determine what upper-bound individual risk would exist at these levels. For arsenic the detection limit within these systems is 1 ug/l. When these detection limits are assumed exposure concentrations, this limit will allow us to detect upper-bound individual lifetime risks greater than 10^{-4}. Concentrations of arsenic have been detected higher than 1 ug/l. For toxaphene, the detection limit is .2 ug/l within these systems. At these concentrations, these laboratory methods will detect risks greater than 10^{-6}. The detection limit for lindane is .005 ug/l. If we assume that this is the exposure concentration, then the upper-bound risk would be 2×10^{-7}.

The highest observed concentrations for selected metals and pesticides in TABLE 5 are below their respective verified Reference Dose for noncancer health effects.

IX. SUMMARY AND CONCLUSIONS

We examined data on monitored toxic pollutant concentrations within the 14 public water supply systems regulated under the Safe Drinking Water Act within the Kanawha Valley. The pollutants reviewed were trihalomethanes, inorganics and pesticides. We did not review potential exposure of toxics within private wells and non-community water systems. The State is not required to monitor regularly within these systems.

The larger systems, Saint Albans, and the West Virginia Water Company, Kanawha Valley and Montgomery Systems, serve a population of approximately 211,000 people. For these systems, we obtained monitoring data for trihalomethanes, and developed an initial conservative assessment of potential cancer risks, as well as an assessment of noncancer health effects for this pollutant category. We extended this analysis to the remaining systems assuming similar trihalomethane concentrations within these systems. Limited monitoring data precluded developing a more detailed health effects analysis of the other pollutants.

The specific findings and conclusions follow.

- Fourteen public water supply systems serve a population of approximately 240,000 people within the Kanawha Valley. For most of these systems monitoring data exists for 19 pollutants. Although we had a limited set of priority pollutant scans for other pollutants, we were confined in our exposure and health effects analysis to these 19 pollutants (See TABLE 2 and TABLE 3).

- Our analysis of trihalomethane data for the three major public water supply systems indicates that an average individual could be exposed to an upper-bound, incremental risk of 1 to 2 x 10^{-4} (a one to two out of ten thousand) probability of contracting cancer over a 70-year exposure to these concentrations of trihalomethanes. (We assume that all trihalomethanes have the same potency as chloroform, a B2 carcinogen.) We assume that the remaining systems have the same levels of trihalomethanes, and consequently risk, since many of these systems draw water from surface water supplies and chlorinate their drinking water also.

- When we calculate incidence for the total population served by the fourteen public water supply systems, we estimate approximately six tenths of a cancer case per year, assuming a lifetime exposure to these concentrations of trihalomethanes. To facilitate comparison of pollutant incidence within the analysis of the Kanawha Valley Toxics Screening Study, we calculated incidence posed by trihalomethanes in drinking water within the air zones defined within the air quality analysis: Belle, Charleston, Institute, and Nitro. These systems are served primarily by the West Virginia Water Company. The incidence from chloroform in drinking water within these zones is approximately one third of a case per year, (or one case every three years) using upper-bound potency values, and assuming a 70-year exposure from this concentration of trihalomethanes.

- We compared reference dose levels for trihalomethanes to our observed average concentration level for these pollutants. RfD levels were not exceeded for any of the trihalomethanes. We also compared reference dose levels for the highest observed concentration for MCL inorganics and pesticides. For this limited data base, we did not see any observed concentrations exceeding references dose levels.

Datagaps

We obtained limited monitoring information for many of the smaller systems and no information on private wells within the study area. Our ability to determine potential exposure for these drinking water systems is extremely limited. In addition, our health effects information is limited. For health assessments, our knowledge on health effects is continually evolving. As we learn more on the potential health effects of many of these pollutants, we should be able to determine, given the availability of pollutant concentrations, which pollutant concentrations may prove harmful.

Additional monitoring would be useful in the following areas:

- Pollutant scans of potential contaminants for public water systems in addition to those scans currently conducted by the West Virginia Water Supply Systems, may determine if the population is exposed to additional pollutants not routinely monitored under the Safe Drinking Water Act.

- There are estimated 22,000 private well systems within Kanawha County, one of three counties in the study areas. Monitoring these supplies would determine if these people are exposed to toxics entering groundwater systems.

REFERENCES

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DRINKING WATER REPORT

Appendix A: Priority Pollutant Scan of West Virginia Water Supply Company - July 1, 1985 (available upon request)

Chapter Six

Surface Water Analysis

KANAWHA VALLEY SURFACE WATER REPORT

I. INTRODUCTION AND PURPOSE

The Kanawha Valley Toxics Study has four stated objectives which provided overall direction to these technical reports.

1. Using available data bases identify many of the chemicals routinely released or present within various exposure pathways.

2. Develop a sense of potential public health concerns these toxic pollutants may pose in various pathways based on health effects and exposure information.

3. For a select number of pollutants, provide an initial, conservative assessment of potential cancer risk and other potential non-cancer health risk from predicted or observed concentrations within exposure pathways.

4. Identify data and information gaps and outline needs and options for future study direction to enable a more detailed investigation of health issues where warranted.

To attempt to meet these objectives we relied primarily on available data. We reviewed information on pollutants detected in industrial discharges, non-point source run-off, the water column and fish tissue. To assess potential health effects of these pollutants, we reviewed fish consumption as one exposure pathway within the study area. Information on fish tissue concentrations and water column concentrations provided limited data for this preliminary assessment of potential health concerns. Where possible, given limited tissue concentration data for select chemicals, we estimated potential health effects if fish were consumed, although actual ingestion rates are not known for the study area. Finally, we outlined the many datagaps which prevent a full assessment of health effects of toxic pollutants.

Within our analysis, we have focused on fish consumption to assess the potential public health concerns toxics may pose. Although we focused on this one exposure pathway we recognized that other routes of exposure to toxic chemicals within the Kanawha River may be important. We did not investigate waterfowl harvesting and consumption, for instance, although this area represents an important duck and goose hunting area for the State. Swimming and boating do occur, and therefore dermal contact is likely. Although the State believes that water contact through recreational uses of the river maybe a significant route of exposure to toxic chemicals for a sizeable portion of the population, this issue is not considered in this analysis. We could not estimate potential risk from these activities due to the lack of information on exposure.

Ingestion of water from the Kanawha River is another potential route of exposure. Four of the 14 public water supply systems identified in the drinking water analysis draw water from the Kanawha River. The sources for these systems, however, are upriver from the industrial facilities within the study area and are not considered contaminated from toxic chemicals discharged by industry into the Kanawha River (Personal Communication, Hodges, 1986.) The river below Belle is not designated as a drinking water source. Since this is an unlikely major route of exposure to toxics, we did not consider the potential health effects from ingestion of drinking water from the Kanawha River.

The consumption of contaminated fish tissue is also a route of exposure for which exposure levels are very difficult to determine. Nevertheless, exposure levels are considered significant because fishing has been observed along the Kanawha River within the study area. We did not estimate exposure or consumption rates within the study area in order to calculate risk. Instead, we estimated consumption rates associated with various levels of carcinogenic risk, and consumption rates which exceed Reference Dose levels for noncancer health effects.

Although we believed that fish consumption may be the most significant route of exposure of toxics from the Kanawha River, consumption of fish from the lower Kanawha River has probably been discouraged by a recent health advisory on fish consumption. This health advisory applies to the Kanawha River below the Coal River, downriver from Charleston, and extends to the Ohio River. The State issued the advisory due to the detection of elevated concentrations of dioxin within fish tissue. The advisory probably has also discouraged fishing further upstream.

The West Virginia Department of Natural Resources conducted a creel study, a study of the number and type of fish caught, at Marmet and Winfield dams during 1986. The final report for this project is expected to be completed in 1987 (Personal Communication, Kain, 1986). While it is recognized that direct inferences can only be made for the specific area studied, the results of this data gathering effort will allow more accurate estimates of consumption patterns on the Kanawha River as a whole.

II. DESCRIPTION OF THE GENERAL METHODOLOGY

We have divided this analysis into three categories of pollutants: (a) Pesticides, Dioxin, and Polychlorinated Biphenyls (PCBs); (b) Metals; and (c) Industrial Discharged Organics. These categories are reviewed separately due to the different analyses applied to the available data for each category of pollutants. In many cases, we have extremely limited data from a few segments of the Kanawha River. Consequently, our ability to draw conclusions for the entire river within the study is limited.

We did not review biological oxygen demand, total suspended solids, oil and grease, coliform bacteria, pH, ammonia, phosphate, and physical and biological factors such as temperature, turbidity and bacterial concentration. While these conventional and non-conventional pollutants have both potential health effects, through bacterial contamination, and ecological impacts, we limited the scope of the analysis to the effects of a limited set of toxics on human health and to provide a general sense of the ecological impacts of some toxic pollutants.

For each pollutant category, we attempted to identify the pollutants which exist within the water column or fish tissue which may pose potential health effects if consumed through fish consumption. We then describe the potential sources, both point and non-point, for these pollutants.

For the review of potential health effects we reviewed fish tissue data if available, and estimated various levels of consumption needed to exceed a designated cancer risk level or Reference Dose (RfD) level for noncancer health effects. Where fish fillet data were not available, we compared observed ambient water concentrations to EPA derived water quality criteria for the protection of human health. These values, based on pollutant specific bioconcentration factors and a national average individual consumption rate, attempt to provide water quality concentrations at which a pollutant may bioaccumulate in fish to various tissue concentration levels, and consequently to various risk levels. These water quality criteria are set at several water concentrations which represent these various levels of carcinogenic risk.

Finally, in order to determine if aquatic organisms are potentially at risk from high concentrations of toxic chemicals in the water column, we compared water concentrations with an additional set of water quality criteria which establish ambient concentrations above which aquatic organisms may be harmed. These are the water quality criteria for the protection of aquatic life.

III. HUMAN AND AQUATIC HEALTH ASSESSMENTS

Cancer Assessment

This study employed a risk assessment screening methodology to evaluate and compare, in a very limited fashion, the potential health risks from exposures to a limited set of toxic pollutants. Risk to an individual is defined as the increased probability that an individual exposed to one or more chemicals will experience a particular adverse health effect during his or her lifetime. For the surface water report, we present our analyses in terms of the estimated incremental lifetime risk to an individual given various consumption rates and concentrations of toxic chemicals in water. We did not calculate incidence, the projected risk to the entire population, due to the lack of actual exposure information on fish consumption.

The risk screening methodology involved both a qualitative and quantitative assessment of the potential carcinogenicity of the selected pollutants. As a screening study, this analysis

employed both types of assessments. EPA's Carcinogen Assessment Group (CAG) reviews the evidence of carcinogenicity for selected pollutants, and classifies pollutants as human carcinogens (Group A), probable human carcinogens (Group B), possible human carcinogens (Group C), not classified as carcinogens due to inadequate evidence (Group D) and not carcinogenic to humans (Group E). These classifications accompany all quantitative risk estimates within this study.

For those chemicals in Groups A, B, and C, CAG provides quantitative, upper-bound estimates of carcinogenic unit risk factors. The traditional approach to carcinogen risk assessment at EPA has been to take the most conservative approach in developing potency estimates. In doing so, EPA develops estimates that are likely to overestimate the true potency of a chemical. We feel that such an approach is appropriate, particularly for public health protection and setting priorities, in order not to underestimate potential human health impacts.

A unit risk factor allows the calculation of the estimated individual risk posed by exposure to a chemical given certain exposure assumptions. To calculate individual risk from the consumption of fish tissue, a pollutant's unit risk factor is multiplied by the fish tissue concentration of the pollutant and the consumption rate of fish by an individual.

Individual Lifetime Risk	=	Unit Risk Factor $(mg/kg\text{-}day)^{-1}$	x	Concentration (mg/kg)	x	Consumption Rate (kg/day)	x	Body Weight Assumption (kg)

Concentration refers to the concentration of the pollutant found in the fish tissue. The average person is assumed to weigh 70 Kg.

Lacking consumption rates for fish by recreational fisherman within the study area, we could not calculate the individual lifetime risks for those categories of pollutants for which we have fish tissue data. To provide some estimate of potential health effects from fish consumption, we specified several levels of risk and calculated a consumption rate associated with the specified risk level.

SAMPLE CALCULATION

CONSUMPTION RATES ASSOCIATED WITH SPECIFIED LEVELS OF RISK FROM CHLORDANE BIOACCUMULATED WITHIN FISH TISSUE

Average Individual Lifetime Risk	=	Unit Risk Factor	X	Concentration	X	Consumption Rate	X	Average Body Weight Assumption

- To solve for consumption rate we can rearrange the equation as follows:

$$\text{Consumption Rate} = \frac{\text{Average Individual Lifetime Risk}}{\text{Unit Risk Factor X Concentration X Body Weight Assumption}}$$

- Assume the following data exists for:

Individual Lifetime Risk	=	For this example, set average individual risk at 1 x 10-5
Unit Risk Factor for Ingestion of Chlordane	=	1.3 (mg/kg-day)-1
Concentration	=	For this example, assume 0.003 mg/kg of chlordane in fish tissue.
Average Body Weight Assumption	=	70 kg

$$\text{Consumption Rate} = \frac{1 \times 10^{-5}}{1.29\ (\text{mg/kg-d})^{-1} \times 0.003\ \text{mg/kg} \times 1/70\ \text{kg}}$$

$$= 0.18\ \text{kg/day}$$

We can convert this consumption rate easily to pounds per week by multiplying this derived consumption rate by 2.2 lbs/kg and 7 days/week. For this example, 0.18 kg/day equals 2.8 lbs/week.

This calculated consumption rate, based on upper bound unit risk factors, indicates that a consumption rate of 2.8 pounds per week for seventy years potentially creates a one in 100,000 increased chance of developing cancer over a lifetime.

We can modify this equation to calculate the consumption rate for a specified level of risk when the cancer health effects from more than one chemical are considered. It should be noted here that we are assuming that cancer risks are additive. Analyzing the effects of a chemical in combination with other chemicals is an extremely complex task, and such analysis has not been done for most pollutants. In the absence of this information, the additivity assumption seems most appropriate.

For two pollutants X, and Y, the equation becomes:

Consumption Rate =

$$70 \times \left[\frac{\text{Individual Lifetime Risk}}{\left(\text{Unit Risk Factor for Pollutant X} \times \text{Concentration of Pollutant X}\right) + \left(\text{Unit Risk Factor for Pollutant Y} \times \text{Concentration of Pollutant Y}\right)} \right]$$

For the industrial organic chemicals however, we did not have fish tissue data to calculate consumption rates. For those organic chemicals, we compared observed water concentrations to EPA derived water quality criteria to protect human health from ingestion of contaminated fish tissue. For suspected or proven carcinogens, EPA provides a set of water concentrations associated with a range of incremental cancer risk resulting from consumption of fish tissue. It is assumed that toxics at various water concentrations will bioconcentrate within fish tissue and can expose consuming individuals to a health risk. For this report, we compared observed water concentration to estimated water concentrations associated with 1×10^{-7}, 1×10^{-6} and 1×10^{-5} average individual incremental cancer risks. These estimated water concentrations are calculated based on lifetime ingestion of fish and assume that a 70-Kilogram adult consumes 6.5 grams of fish daily for a seventy year lifetime.

Noncancer Health Effects

No currently accepted techniques exist for estimating the probability of incidence of noncancer effects. Therefore, in evaluating the potential noncancer health risks within this study, we relied on benchmark values or Reference Dose (RfDs), expressed in units of mg/kg/day, which represent doses levels below which adverse health effects are not likely to occur in most people. Unlike cancer effects, noncancer health effects are assumed to be threshold events. This threshold assumption means that noncancer health effects are assumed more likely to occur above these exposure concentrations. Exposures that are less than the RfD are not likely associated with noncancer health effects and therefore less likely to be of regulatory concern.

To calculate an RfD, EPA scientists collect the available animal and human data and note the various dose levels (in milligrams per day) at which different health effects are seen. The scientists then compare the dose with effect information and identify the NOEL (the No Observed Effect Level). The NOEL represents the highest dose tested that did not produce observable results. The scientists also try to define the LOEL (Lowest Observed Effect Level), which is the lowest dose tested at which some type of effect occurs. They also try to define the FEL (Frank Effect Level), which is the dose that involves more serious health problems.

There will be many NOELs and LOELs for each chemical. These levels depend on the doses selected and the health effects tested for by the researchers. Research is very expensive and each experiment cannot be exhaustive. Since EPA is dependent on available research, there is some uncertainty in the definition of NOELs and LOELs.

To calculate the RfD, EPA scientists select the most reliable NOEL and divide it by safety factors. The selection of appropriate safety factors is based on the nature of the study from which the NOEL was derived. Most safety factors are multiples of ten, with each one representing an extra degree of uncertainty to account for extrapolation from animal data to the average human, from the average human to the most sensitive subgroup, from subacute effects to chronic effects, and the LOEL to the NOEL. In general, the safety factor is larger than the expected differences in the exposure levels producing these effects, and is therefore a conservative estimate of the actual threshold.

For our analysis, we calculated the number of pounds per week which, if consumed, would exceed the Reference Dose for the pollutant. We only reviewed noncancer health effects of pollutants for which EPA has established an RfD.

Water Quality Criteria Exceedances

We derive a sense of the potential for harmful chronic ecological effects from pollutants by simply comparing ambient water concentrations to EPA derived ambient water quality criteria for the protection of aquatic life. These criteria are derived from aquatic toxicology data for chronic effects on both plants and animals. EPA derives, in most cases, the highest four-day average concentration which, if exceeded, may result in the loss of sensitive species or the impairment of reproductive functions. In many cases, these four day concentrations are made a function of such water quality characteristics as pH or hardness if a correlation between toxicity and the characteristic can be established. For this analysis, we compare the percentage of observations that exceed the water quality chronic criteria for the protection of aquatic life. A high percentage of water quality criteria exceedances over an extended period of time would indicate that chronic toxicological impacts are likely and sensitive aquatic species may be eliminated.

IV. DESCRIPTION OF STUDY AREA

The Kanawha River begins at the confluence of the New and Gauley Rivers and flows northwestward toward the Ohio River. The Kanawha River within the study is bounded upstream by Alloy and downstream by Winfield Dam. The distance between Alloy and Winfield is approximately 60 miles.

The Kanawha River drains an area characterized by rugged hills and mountains with deep valleys cut by rivers and streams. Three major tributaries, the Coal, the Pocatalico and the Elk Rivers, drain into the river within the study area. Three dams have been built within the study area, and help maintain the Kanawha River at a minimum width of 300 feet and a depth of nine feet.

Land use adjacent to the river is primarily urban and industrial with other land uses including mining, recreation, transportation and utility right-of-ways.

For this analysis, we identified 22 major industries which discharge into the Kanawha River. The Kanawha Valley, similar to other metropolitan areas, is likely to have non-point source run-off characteritic of residential and commercial areas. The large concentration of industrial facilities may also provide non-point source loadings from run-off from industrial lots. Hazardous waste sites and sediments may also contribute pollutant loadings deposited from past industrial activity.

V. PESTICIDES, DIOXIN, AND POLYCHLORINATED BIPHENYLS (PCBs)

Pesticides, dioxin, and PCBs are the first of three categories of pollutants reviewed within the surface water analysis of the Kanawha Valley Toxics Study. In this section we briefly outline the methods used to estimate the potential health effects of consumption of a limited set of pesticides, PCBs, and dioxin bioconcentrated within edible fish tissue.

It is important to reemphasize that we do not currently know recreational fish consumption patterns within the study area. Indeed, consumption may be zero. The West Virginia Department of Health has issued a health advisory for the lower Kanawha River due to the recently detected elevated concentrations of dioxin within fish in the Nitro area. Since exposure is not known, and may be zero, we have estimated the potential health effects of several hypothetical consumption or exposure rates. Despite the advisory, fish consumption from the Kanawha River may be occurring.

To estimate potential exposure concentrations from contaminated fish tissue, we calculated potential fish tissue concentrations for a small segment of the river. We reviewed the only available data for fillet concentrations of a limited set of pesticides, PCBs, and dioxin sampled in fish tissue in the Nitro area

on the lower Kanawha River. We calculated mean concentrations for selected pollutants from this limited dataset. Although these observed concentrations may be adequate to determine fillet fish tissue concentrations within the Nitro area, the limited sampling territory precludes drawing inferences for the entire Kanawha River within the study area. In fact, a 1986 survey by WV DNR and Region III EPA suggests that dioxin levels at different sites on the Kanawha River may vary with location (Personal Communicaton, Smith 1986).

We reviewed toxicity data for our selected pollutants. We calculated weekly consumption rates needed to exceed a defined level of risk. In addition, we examined noncancer health effects by calculating weekly consumption rates needed to exceed available Reference Dose (RfD) levels.

Potential Pollutants

The Department of Natural Resources (DNR) has monitored for pesticides and PCBs in whole fish tissue over the years. Recently, DNR has monitored for dioxin in the Nitro area. Pesticide run-off from agricultural and residential areas and accidental PCB spills from industry over the years have led to probable contamination of fish tissue. The source of the dioxin has not yet been determined. Since these pollutants readily bioaccumulate within fish tissue, West Virginia has sampled fish throughout the Kanawha River over the past years to determine if elevated concentrations of these pollutants occur.

The Department of Natural Resources has sampled for the following pesticides and organics within whole fish tissue over the years (DNR, 1986):

Aldrin	Trans - Nonachlor
Dieldrin	Cis - Nonachlor
Endrin	Endosulfan I
Hexachlorobenzene	Endosulfan II
Lindane	Heptachlor
Methoxychlor	Heptachlor Epoxide
Toxaphene	O,P'- DDD
Alpha-BHC	P,P'- DDD
Beta-BHC	O,P'- DDE
Delta-BHC	P,P'- DDE
Arochlor 1254	O,P'- DDT
Arochlor 1260	P,P'- DDT
Trans-Chlordane	
Cis-Chlordane	

Toxaphene is the only pollutant from this list which has not been detected within whole fish tissue.

Observed Fish Tissue Concentrations

In September 1985, the West Virginia DNR sampled fish fillet tissue at Nitro, West Virginia. Nitro is the last of the industrial areas on the Kanawha River within the study area. DNR sampled for seven fish species including, but not limited to, those commonly caught and harvested in the area. The fish were collected as a follow-up to the National Dioxin Study and were intended to help determine the degree of dioxin contamination in various species. These species were Smallmouth Buffalo, Channel Catfish, Sauger, Smallmouth Bass, Spotted Bass, Largemouth Bass, and Freshwater Drum. It should be noted that we limited our analysis to fillet rather than whole fish samples due to the difficulties and uncertainties associated with estimating concentrations of pollutants in fillets from whole fish samples.

Fish between 10 and 14 inches, which approximate the average size caught by fishermen, were targeted for analyses. DNR analyzed composite fillet samples containing between three and five fish of an individual species for chlordane, PCB, dioxin, and dieldrin. TABLE 1 presents a mean fillet concentration, calculated from reported composite concentrations, for each pollutant by fish species.

Mean concentrations of pollutants are the highest for the Smallmouth Buffalo, Channel Catfish, Freshwater Drum, and Sauger The three bass species have lower, often nondetected concentrations for these pollutants. Differences in migratory habits, positions within the food chain, and lipid concentrations which accumulate these pollutants within fish tissue may account for these differences.

Potential Human Health Risks

Contamination by pesticides, PCBs, and dioxin of fish tissue may present a health hazard if the bioaccumulated toxic chemical is present in large enough concentrations and consumed by individuals. Concentrations of pesticides, PCBs, and dioxin have been detected in fish tissue of several species. Such concentrations may present a health concern for the consuming public.

Due to the lack of exposure information for fish consumption, we instead calculated weekly consumption rates which could expose a consuming individual to a designated level of carcinogenic risk. Also, we calculated a weekly consumption rate which will provide toxic pollutant concentrations exceeding noncancer health effect levels which are considered safe for pollutants with established

TABLE 1

KANAWHA VALLEY SURFACE WATER REPORT

SEPTEMBER, 1985 OBSERVED PESTICIDE, DIOXIN, AND PCB CONCENTRATIONS[1] IN FISH FILLET AT NITRO, WEST VIRGINIA

(mg/kg)

Fish Species	Number of Composite Samples	Chlordane[2]	PCBs[3]	Dieldrin	Dioxin[4]
Smallmouth Buffalo	4	0.199	1.127	0.015	0.000032 †
Channel Catfish	2	0.104	0.655	0.005	0.000023
Sauger	2	0.032	0.264	ND	0.000006
Smallmouth Bass	1	ND	ND	ND	0.000007
Spotted Bass	1	ND	ND	ND	0.000013
Largemouth Bass	3	0.003	0.067	ND	0.000004 †
Freshwater Drum	2	0.03	0.13	0.005	0.000008

[1]Concentrations shown in this table represent the mean of composite samples. Each composite contains between 3 and 6 fish specimens. Specimens collected by DNR, West Virginia.

[2]Chlordane concentrations are the sum of Cis-and Trans-chlordane and Cis-nonachlor concentrations in fillets.

[3]PCB concentration is the concentration of Arochlor 1260.

[4]Dioxin values were measured in picograms/gram and have been converted to mg/kg for this table.

†These mean values were calculated from 5 composite samples.

Reference Doses. In both cases, we set a specified level of cancer risk or a Reference Dose (RfD) level for noncancer health effects and calculated the weekly consumption rate needed to exceed these levels.

For carcinogenic risk, we calculated a series of consumption rates which correspond to exposure levels associated with estimated lifetime average individual risks of 1×10^{-5}, 1×10^{-4}, and 1×10^{-3}. These risk levels were chosen for policy development purposes only.

Chronic Toxicity Data: TABLE 2 presents the carcinogenic unit risk factors and strength of evidence classifications for PCBs, chlordane, dioxin, and dieldrin. The unit risk factors were developed by EPA's Carcinogen Assessment Group, CAG, and represent plausible upper-bound values.

For noncancer health effects, only chlordane has a Reference Dose to date. The RfD is 0.00005 mg/kg-day. RfDs for PCBs, dieldrin and dioxin have not yet been established by EPA's Reference Dose Workgroup. RfDs are benchmark concentrations to determine the potential for noncancer health effects. Exposures below the RfD are not likely to be associated with noncancer health effects.

Exposure Concentrations

For fish tissue exposure concentrations, we assumed concentrations observed within the 1985 fish fillet data from the Nitro area presented in TABLE 1 . As we have noted, these samples are from a limited geographic area with in the study area and are from a single point in time. They may not be representative of the entire study reach or of contaminant levels (i.e., exposure levels) over time. DNR has detected pesticides and PCBs pollutants in whole fish tissue throughout the Kanawha River in the study area.

Consumption Rates

We calculated individual consumption rates for various fish species which would exceed defined levels of lifetime incremental risk. These consumption rates are based on the upper-bound potency values in TABLE 2 and the assumed exposure concentrations from TABLE 1. TABLE 3 presents the results of this analysis.

According to TABLE 3, consumption rates could range from one tenth of a pound to one pound per week, depending on fish species, for an individual to be exposed to an upper-bound lifetime risk of 1×10^{-3} (a one out of a thousand chance). Consumption rates are lower for lower risk levels.

Dioxin and PCBs contribute more than 90 percent of the risk for each species. For Smallmouth Bass and Spotted Bass, dioxin was the only pollutant detected. At the observed concentration of dioxin for these two species, an individual would need to consume one half pound of Spotted Bass or one pound of Smallmouth Bass to be exposed to an upper bound risk of one in a thousand.

When interpreting TABLE 3, the reader should keep in mind that the traditional approach to carcinogen risk assessment at EPA has been to take the most conservative approach in developing potency estimates. In doing so, EPA develops estimates that are highly

TABLE 2

KANAWHA VALLEY SURFACE WATER REPORT
PRELIMINARY RISK SCREENING RESULTS

UPPER-BOUND CANCER UNIT RISK VALUES:
POLLUTANTS IN FISH TISSUE

Pollutant	Unit Risk Factor for Inhalation $(mg/kg\text{-}day)^{-1}$	Grouping Based[1] on EPA Criteria
PCB	4.34	B2
Chlordane	1.33	B2
Dieldrin	20.02	B2
Dioxin	1.60E+05	B2

[1] Weight-of-Evidence rating derived by CAG, based on EPA's classification system: A = proven human carcinogen; B = probable human carcinogen (B1 indicates limited evidence from human studies, B2 indicates sufficient evidence from animal studies but inadequate evidence from human studies); C = possible human carcinogen; D = not classifiable; and E = no evidence of carcinogenicity.

Source: Carcinogen Assessment Group, EPA, 1986.

TABLE 3

KANAWHA VALLEY SURFACE WATER REPORT
PRELIMINARY RISK SCREENING RESULTS

WEEKLY RATES OF FISH CONSUMPTION
ASSOCIATED WITH VARIOUS RISK LEVELS

INTENDED FOR POLICY DEVELOPMENT
UPPER-BOUND ESTIMATES

	CONSUMPTION RATES PRODUCING SPECIFIED RISK LEVEL1 (lbs/week)			% CONTRIBUTION OF POLLUTANTS TO RISK			
FISH SPECIES 2	10^{-3}*	10^{-4}*	10^{-5}*	PCB°	CHLORDANE°	DIELDRIN°	DIOXIN°
Small Mouth Buffalo	0.103	0.0103	0.00103	47%	2%	3%	48%
Channel Catfish	0.160	0.0160	0.00160	42%	2%	3%	53%
Sauger	0.496	0.0496	0.00496	53%	2%	0%	46%
Large Mouth Bass	1.025	0.1025	0.00103	41%	1%	0%	57%
Freshwater Drum	0.509	0.0509	0.00509	27%	1%	9%	63%
Smallmouth Bass	0.960	0.0960	0.00960	0%	0%	0%	100%
Spotted Bass	0.520	0.0520	0.00520	0%	0%	0%	100%

THE UNIT RISK FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS, CONSUMPTION RATES ASSOCIATED WITH VARIOUS RISK LEVELS WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS ACTUAL CANCER-CAUSING CONSUMPTION RATES IN THE KANAWHA VALLEY. ACTUAL RISKS FROM THESE RATES MAY BE SIGNIFICANTLY LOWER; IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

Fish samples obtained by the West Virginia Department of Natural Resources in September, 1985 at Nitro, West Virginia.

*Risk levels are arbitrarily chosen for demonstration purposes only.

°% Concentrations were developed from the concentration values in Table 1.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA.

likely to overestimate the true potency of a chemical. We feel that such an approach is appropriate, particularly for public health protection and setting priorities, in order not to underestimate potential human health impacts. We also calculated the weekly consumption rate needed to exceed the chlordane RfD. Of the pollutants studied in this section of the analysis, there is a Reference Dose only for chlordane. According to TABLE 4, the weekly consumption rates for Smallmouth Buffalo, Channel Catfish, Sauger, and Freshwater Drum would range from one third of a pound to almost 2 pounds. Since chlordane was detected in limited amounts, or not at all in the bass species, calculated consumption rates are much higher for these species (18 lbs/week for Large Mouth Bass).

It is stressed that these results only apply to a limited segment of the river and are based on a one day sampling event. We cannot say that these findings apply to the whole Kanawha River within the study area.

VI. METALS AND INORGANICS

Metals and inorganics are the second of three categories of pollutants reviewed within the surface water analysis of the Kanawha Valley Toxics Study. In this section we briefly outline the methods used to develop a sense of the potential health and ecological effects posed by metals and inorganics.

Like the pesticides analysis, it is important to emphasize that we do not currently know recreational fish consumption patterns within the study area. Again, consumption may be zero. Since exposure is not known, we have estimated the potential health effects of several hypothetical consumption rates for cancer and noncancer health effects.

We have calculated edible fish tissue mean concentrations for a limited set of metals within fillet tissue at the Nitro area. This is the same dataset we used for the pesticide, PCB and dioxin analysis. Although these observed concentrations may be adequate to determine fillet fish tissue concentrations within this area, the limited sampling territory precludes drawing inferences for the entire study area.

To determine potential health effects for these bioconcentrated metals we reviewed health effects information for a small set of inorganics. From these data we estimated weekly consumption rates which may pose potential chronic health effects.

For potential harmful effects on aquatic organisms we simply compared observed ambient water concentrations to EPA chronic criteria values for the protection of aquatic life to determine if such criteria were exceeded during the sampling period.

Pollutant Sources

Metals and inorganics are present in point source industrial discharges, urban non-point source run-off, and possibly in hazardous waste site run-off and contaminated groundwater discharge to the surface water.

TABLE 4

KANAWHA VALLEY SURFACE WATER REPORT

INTENDED FOR POLICY DEVELOPMENT

LEVELS OF WEEKLY FISH CONSUMPTION
THAT COULD EXCEED THE REFERENCE DOSE
FOR NONCANCER HEALTH EFFECTS FOR CHLORDANE[1]

Fish Species	Consumption (lbs/week)
Small Mouth Buffalo	0.3
Channel Catfish	0.6
Sauger	2.0
Freshwater Drum	1.8
Small Mouth Bass	Not Detected
Spotted Bass	Not Detected
Large Mouth Bass	18.0

[1]The RfD for Chlordane is 0.00005 (mg/kg-day), based on Yonemura et al. (1983) showing liver necrosis effects.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA.

To determine industrial point source contribution to the Kanawha River within the study area, we reviewed NPDES permit applications, discharge monitoring reports, and priority pollutant scans of the major industrial facilities for 1983, 1984, and 1985. The list of metals detected in the discharge of the facilities is presented in TABLE 5.

Non-point source run-off is an additional source of metals and inorganics to the river. Unfortunately, we did not have site-specific metal loadings from non-point source run-off. Consequently, we developed non-point source estimates from the National Urban Storm Water Run-off Program (NURP, 1982). This program was conducted from 1980 to 1982, with run-off samples collected from predominately residential and commercial areas throughout the country. Although these estimates are not site specific, and may be off by an order of magnitude, they may be sufficient to determine the magnitude of non-point source contributions for this screening exercise. Technical Appendix A describes our technique used to estimate non-point source loading.

Appendix A also outlines our technique for estimating point source loadings. Our point source loadings are based on 1984 information. We collected discharge information for facilities from priority pollutant scans, NPDES permits, and DMRs. For each pollutant we totaled the loadings based on flow and concentration information obtained from the database.

We were unable to quantify run-off from industrial lots nor hazardous waste sites. Our analysis of generic scenerios of hazardous waste site contamination of groundwater discharge (See Kanawha Valley Toxic Screening Study "Hazardous Waste Site Analysis") indicates that loadings from groundwater are probably minimal.

TABLE 6 presents loading estimates for selected metals from non-point and point sources. For arsenic and nickel, there appear to be equal loading rates to the river from both point and non-point sources. Non-point source contributions for total chromium, copper, lead, antimony, cyanide, and zinc appear to outweigh the contribution from point sources. Cadmium is the only metal where point source loadings are larger than a non-point source loading.

Ambient Water Concentrations

For water quality analysis, we divided the river into five segments. These segments correspond to the Valley Zones we established for the ambient air quality analysis and correspond to industrial clusters in the Kanawha Valley. Segment 1 is the river stretch from Alloy to Charleston (90-57 KRM). DNR maintains a river monitoring station at Kanawha River Mile 74. We used data from this station to develop the average water concentration for this segment. Segment 2 is the Charleston area between River Mile 57 and 50. We obtained ambient water monitoring data from a sampling station at Kanawha River Mile 52 to represent this segment. The Institute area is Segment 3 stretching from Kanawha River Mile 50

TABLE 5

KANAWHA VALLEY SURFACE WATER REPORT

MAJOR FACILITIES DISCHARGING METALS

MAP KEY	FACILITY	ALUMINUM	ANTIMONY	ARSENIC	BARIUM	CADMIUM	CHROMIUM[1]	COPPER	LEAD	MANGANESE	MERCURY	NICKEL	SELENIUM	ZINC
1	Allied Chemicals											✓		✓
2	Appalachian Power (Amos Plant)		✓	✓		✓	✓	✓	✓	✓	✓	✓	✓	✓
3	Appalachian Power (Kanawha River Plant)		✓	✓				✓	✓		✓	✓		✓
4	Autex Fibers (plant closed)													
5	Chemical Leaman Tank Lines						✓							
6	Costal Tank Lines						✓							
7	Occidental		✓			✓		✓	✓		✓	✓		✓
8	Dupont		✓	✓				✓				✓		✓
9	Elkem Metals		✓	✓			✓	✓	✓		✓	✓		✓
10	FMC, Nitro			✓			✓							
11	FMC, South Charleston			✓		✓	✓	✓	✓			✓		✓
12	CST/Fike Chemicals (Artel)	✓		✓	✓	✓	✓	✓	✓	✓	✓	✓		
13	Kincaid Enterprises													
14	Mason Dixon Tank Lines													
15	Monsanto		✓	✓		✓	✓	✓	✓		✓			
16	S. Charleston Sewage Treatment Plant							✓						
17	Rhone Poulenc		✓	✓		✓			✓					✓
18	Union Carbide, Technical Center	✓			✓									
19	Union Carbide, S. Charleston		✓					✓	✓		✓	✓	✓	✓
20	Hatfield-Henson			✓				✓	✓			✓		✓
	P.B. and S. Chemical Company							✓						
	Smith Fastner Co., Inc.					✓		✓	✓					✓

[1]Chromium present in the waste stream is mainly Chromium-VI.

SOURCE: Discharge Monitoring Reports, Permit Applications and priority pollutant scans.

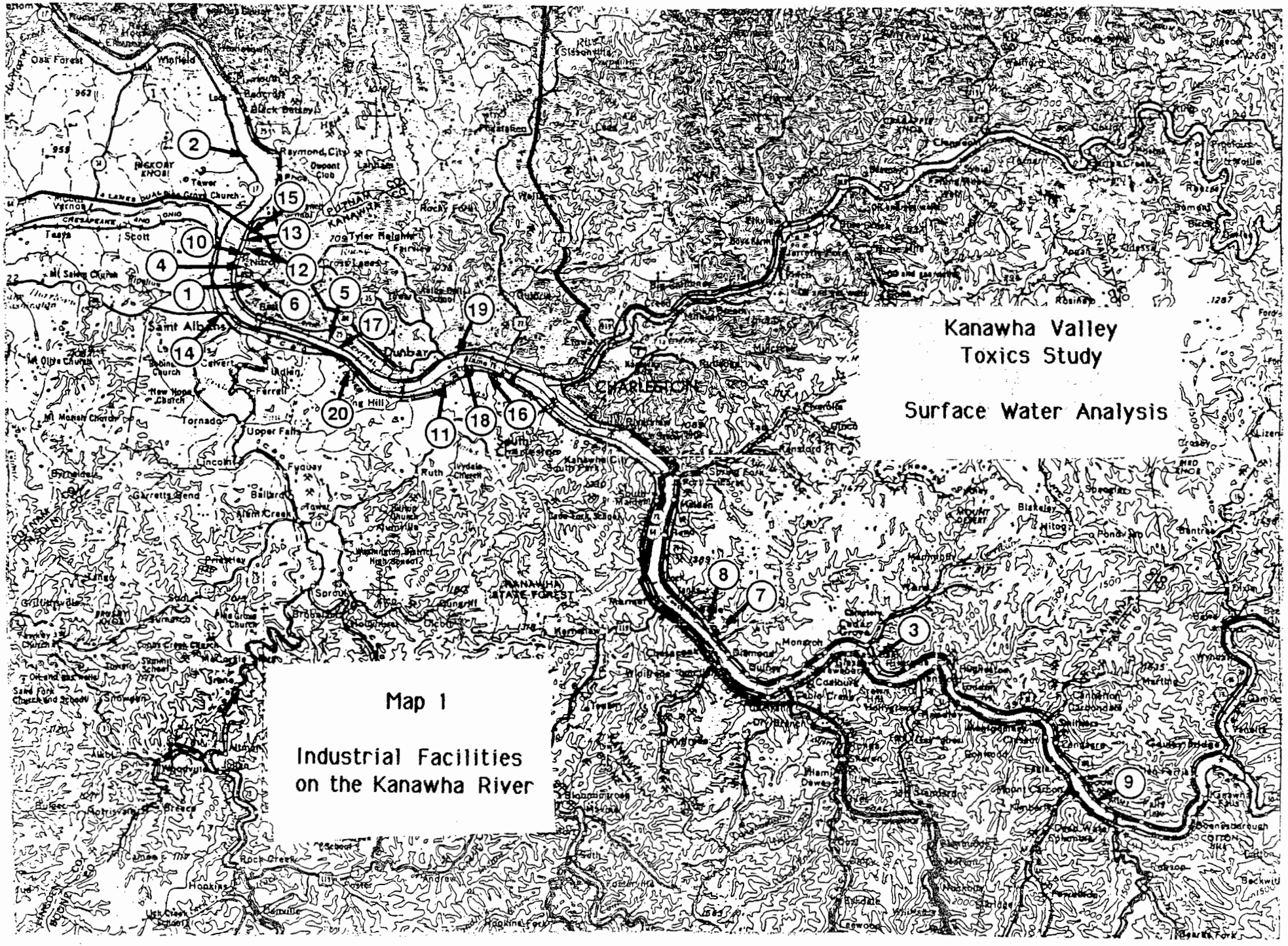
Kanawha Valley
Toxics Study
Surface Water Analysis
Map 1
Industrial Facilities
on the Kanawha River
1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18
19
20
Saint Albans
Dunbar
CHARLESTON
South Charleston
Nitro
Raymond City
Tornado
Upper Falls
Pratt
Sorrel
Alum Creek
Ruth
Lincoln
Fyquay
Scott
Tyler Heights
Cross Lanes
Rocky Fork
Kanawha State Forest
Poca
Cedar Grove
Montgomery
Belle
Marmet
Chesapeake
Clendenin
Gauley Bridge
Kanawha Falls
Oak Forest
Winfield
Buck Bottom
Dupont Club
Tavern
Mount Vernon
Chesapeake and Ohio
Rock Creek
Hopkins
Hopkins Fork
Andrew
Leewood
Whitesville
Blakeley
Hilo
Ponda Bro
Centre
Marmet
Ward
Dixie
Eskdale
Standard
Boomer
Cotton Hill
Beckwith
Cabin Creek
Chelyan
Sissonville
Elkhorn
Ruth
Tiskilwa
Ashton
Alum Creek
Pratt
Sorrel
Brownstown
Nitro
Saint Albans
Dunbar
Kanawha City
South Charleston
Spring Bank
Kanawha Falls
Gauley Bridge
Montgomery
Belle
Marmet
Chesapeake
Clendenin
Cedar Grove
Poca
Dry Branch
Pratt
Sorrel
Alum Creek
Ruth
Lincoln
Fyquay
Tornado
Upper Falls
Scott
Tyler Heights
Cross Lanes
Rocky Fork
Raymond City
Buck Bottom
Winfield
Oak Forest

TABLE 6

KANAWHA VALLEY SURFACE WATER REPORT
PRELIMINARY RISK SCREENING RESULTS

NON-POINT AND POINT SOURCE LOADINGS
FOR METALS AND INORGANICS

INTENDED FOR POLICY DEVELOPMENT

Metal	Estimated Non-Point Source Load[1] (Metric Tons/Yr)	Estimated Point Source Load[2] (Metric Tons/Yr)	Ratio of Non-Point To Point
Antimony	8.3	0.4	21 : 1
Arsenic	1.0	1.4	1 : 1.4
Cadmium	0.4	33.0	1 : 83
Chromium - Total	2.0	0.4	5 : 1
Copper	11.7	1.7	7 : 1
Cyanide	11.3	0.9	13 : 1
Lead	42.4	1.7	25 : 1
Mercury	0.6	<0.1	--
Nickel	3.7	3.5	1 : 1
Selenium	25.6	0.0	--
Silver	0.3	<0.1	--
Zinc	59.8	1.0	60 : 1

[1]See Appendix 1 for derivation of estimates.

[2]Estimated point source loadings are estimated from NPDES permit applications, Discharge Monitoring Reports, and priority pollutant scans. See Appendix 1 for estimation techniques.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA.

to 45 with a monitoring station at KRM 46. Segment 4 corresponds to the St. Albans to Nitro area, (Kanawha Mile 45 to 42). Segment 4 does not have a monitoring station for metals. Finally, Segment 5 corresponds to the Nitro and Winfield areas, KRM 42 to 32. We used ambient water monitoring data from the monitoring station at Kanawha River Mile 31 for this segment.

TABLE 7 presents mean concentration values for these segments. We calculated these mean concentration values from monitored concentrations from these designated monitoring stations within each zone. All monitoring data for metals were obtained from STORET.

For many observations, the concentrations were below the detection limit. In cases where the observation was below the detection limit, we averaged that value at the detection limit concentration. Consequently, these mean values should be considered the maximum possible average based on the available data.

Fish Tissue Concentrations

In September 1985, the Department of Natural Resources sampled fish fillet tissue at Nitro, West Virginia. This is the same database used for the pesticides, dioxin and PCBs analysis. These observed concentrations are presented in TABLE 8 as the range observed in composite samples.

Bioconcentrated metal concentrations appear relatively comparable across these six species of fish with total chromium and arsenic concentrations differing among these species by the largest factors. Other mean metal concentrations differ by no more than a factor of two across these species.

Although this dataset may be appropriate for estimating tissue concentrations in the lower Kanawha River, the limited sampling area precludes drawing inferences for the entire study area.

Potential Human Health Risks

For these metals and inorganics, inorganic arsenic is the only metal which has received a unit risk factor for carcinogenicity. Although inorganic arsenic is a proven carcinogen (weight-of - evidence classification is A), recent laboratoy analyses suggests that arsenic may exist in organic form within fish tissue, a far-less toxic form than inorganic arsenic. For this reason, we do not estimate the likelihood of carcinogenic risk from arsenic in fish tissue.

TABLE 7

KANAWHA VALLEY SURFACE WATER REPORT

ESTIMATED AVERAGE AMBIENT WATER CONCENTRATIONS[1] FOR METALS BY RIVER SEGMENTS FOR 1983 - 1985

INTENDED FOR POLICY DEVELOPMENT

CONCENTRATION (µg/l) BY RIVER SEGMENT (# OF OBSERVATIONS)

Metal	1		2		3		4	5		Range
Antimony	<28.0	(3)	<30.0	(4)	<40.0	(3)	NM	<22.5	(2)	<22.5 - <40.0
Arsenic	3.0	(6)	1.3	(8)	1.6	(6)	NM	1.3	(4)	1.3 - 3.0
Cadmium	2.0	(21)	1.3	(8)	1.3	(6)	NM	1.0	(17)	1.0 - 2.0
Chromium - VI	NM	--	1.3	(6)	1.3	(6)	NM	<1.0	(14)	<1.0 - 1.3
Chromium - Total	11.3	(3)	8.5	(4)	8	(3)	NM	10.0	(2)	8.0 - 11.3
Copper	2.3	(6)	3.0	(8)	2.7	(7)	NM	3.7	(6)	2.3 - 3.8
Cyanide	1.0	(6)	3.0	(8)	2.0	(6)	NM	1.0	(6)	1.0 - 3.0
Lead	13.5	(21)	10.0	(8)	24.5[†]	(11)	NM	11.2	(17)	10.0 - 24.5
Mercury	0.1[†]	(5)	0.2[†]	(6)	0.1	(3)	NM	0.2	(2)	<0.1 - 0.2
Nickel	10.0	(6)	<10.0	(4)	<10.0	(3)	NM	<10.0	(2)	<10.0
Selenium	1.0	(2)	1.1	(4)	<0.8	(3)	NM	1.0	(2)	<0.8 - 1.1
Silver	<2.0	(3)	<2.0	(4)	<2.0	(3)	NM	<2.0	(2)	<2.0
Zinc	10.2	(6)	7.8	(4)	8.3	(3)	NM	10.2	(5)	7.8 - 10.2

[1]Water concentrations are the average of monitored values at STORET river stations within each river segment. All average concentrations are averages of detected concentrations and the detection limit, if the monitoring did not detect the inorganic. If all concentrations are less than the detection limit, average concentration value appears with a less than sign (<). Consequently, these values should be considered the maximum possible average based on the available data.

NM = no monitoring station within the river segment.

[†]Averages are based on 1980 - 1985 data.

TABLE 8

KANAWHA SURFACE WATER REPORT

SEPTEMBER 1985 METAL CONCENTRATION RANGES
IN FISH FILLET COMPOSITES AT NITRO, WEST VIRGINIA

INTENDED FOR POLICY DEVELOPMENT

(mg/kg)

FISH SPECIES* (NUMBER OF COMPOSITES IN RANGE)

METALS	LARGE MOUTH BASS (5)	SMALL MOUTH BASS (1)	SAUGER (2)	CHANNEL CATFISH (2)	SMALL MOUTH BUFFALO (5)	FRESH WATER DRUM (2)
Mercury (inorganic)	Tr - 0.02	Tr	Tr - 0.02	Tr - 0.02	Tr - 0.02	Tr - 0.02
Mercury (organic)	0.1 - 0.17	0.16	0.14 - 0.23	0.15 - 0.22	0.1 - 0.24	0.18 - 0.33
Cadmium	Tr - 0.05	Tr	Tr - 0.05	Tr - 0.05	Tr - 0.05	Tr - 0.05
Chromium	0 - 0.8	ND	Tr - 0.4	Tr - 0.4	Tr - 1.3	0.9 - 2.9
Copper	1.7 - 2.9	2.5	2.5 - 2.8	1.5 - 2.0	1.4 - 2.8	1.7 - 2.3
Lead	Tr - 0.7	0.5	0.5 - 0.7	Tr - 0.4	Tr - 0.4	0.5
Silver	0.1	0.1	0.1	0.1	0.1	0.1
Zinc	8.5 - 12.8	7.7	8.4 - 8.9	4.8 - 8.7	8.3 - 8.9	5.1 - 5.2
Arsenic	0.1 - 0.14 (0.116)'	0.1 (0.1)'	0.02 - 0.03 (0.025)'	0.01 - 0.02 (0.015)'	Tr - .02 (0.018)'	0.04 - 0.05 (0.045)'
Nickel	Tr - 1.1	Tr	Tr - 0.8	Tr - 0.5	Tr - 0.5	Tr - 0.4

* Fish samples obtained by the West Virginia Department of Natural Resources in September, 1985 at Nitro, WV (

Tr = "Trace". A "trace" value indicates the presence of pollutant concentration below the reliable detection lim

ND = Not Detected.

'Mean values were calculated for Arsenic which is considered a carcinogen.
Risk estimates for Arsenic in this analysis use this mean.

In TABLE 9 we present RfDs for possible noncancer health effects for the selected pollutants. The RfDs are benchmark values for the protection of human health from noncancer health effects. Exposures below the RfD are not likely associated with noncancer health risks, As the frequency of exposures exceeding the RfD increases, as well as the magnitude of the exceedance, the probability of adverse noncancer health effects increases.

TABLE 10 presents estimated weekly consumption rates for mercury, silver,and chromium for the highest observed fish tissue concentration from the Nitro Area dataset. To be conservative, we have assumed that organic mercury is methyl mercury and total chromium is hexavalent. It is likely that chromium does not exist in its hexavalent form in fish tissue.

According to TABLE 10 an individual could consume one pound of fish a week with the highest detected concentration of total mercury and exceed the Reference Dose for mercury.

WATER QUALITY EXCEEDANCES

To determine if aquatic life could be affected by metals within the water column, we determined the percentage of observations which exceed ambient water quality criteria for the protection of aquatic life.

Water quality criteria designate ambient water concentrations which are not expected to produce harmful effects in aquatic oganisms. Levels which exceed the criteria may produce harmful effects. These criteria concentrations are often functions of pH, hardness, and chronic toxicity of the pollutant to aquatic organisms. This information is used to calculate the average concentration that should not cause unacceptable levels of chronic toxicity during a long term exposure.

We compared observed values for the years 1983-85 to the chronic toxicity criteria for a pollutant. In many cases, EPA has established 4-day average concentrations for metals; for other metals, 24-hour average concentrations criteria have been determined.

TABLE 11 presents the number of samples analyzed by metals monitored within the Kanawha River, the chronic toxicity criteria

TABLE 9

KANAWHA VALLEY SURFACE WATER REPORT

NONCANCER HEALTH EFFECTS

Pollutant	Health Effect	Reference Dose[1] (mg/kg-day)	Study RfD Based On
Chromium - III	none observed	0.005	Ivankovic and Preussmann (1975)
Chromium - VI	none observed	1.00	MacKenzie et al. (1958)
Mercury (inorganic)	renal and kidney damage	0.002	Fitzhugh et al. (1950)
Methyl Mercury	CNS effects	0.0003	Clarkson et al. (1973)
Silver	argyria	0.003	Gaul and Staud (1935) Blumberg and Carey (1934) East et al. (1980)

[1] EPA/RfD indicates a benchmark value derived from no-effect threshold value. Exposures greater than the RfD are assumed to be associated with an increase in noncancer health effects. EPA reviews and verifies these thresholds internally through the Reference Dose Workgroup.

TABLE 10

KANAWHA VALLEY SURFACE WATER REPORT

LEVELS OF WEEKLY FISH CONSUMPTION
THAT COULD EXCEED NONCANCER HEALTH EFFECTS
REFERENCE DOSES FOR METALS

INTENDED FOR POLICY DEVELOPMENT

Metal	Highest Concentration	Species	lbs/week to Exceed RfD
Mercury(organic)	0.33	Fresh Water Drum	1
Chromium*	2.9	Fresh Water Drum	2
Silver	0.1	Fresh Water Drum†	32

*Concentration is for Total Chromium but reference dose for Chromium-VI used to calculate lbs/week.

†All species showed the same concentration value.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA.

TABLE 11

KANAWHA VALLEY SURFACE WATER REPORT

PERCENTAGE OF SAMPLE OBSERVATIONS FROM 1983-1985
WHICH EXCEED CHRONIC WATER QUALITY CRITERIA
FOR THE PROTECTION OF AQUATIC LIFE

INTENDED FOR POLICY DEVELOPMENT

INORGANIC	NUMBER OF OBSERVATIONS	% OF ACTUAL OBSERVATIONS EXCEEDING CHRONIC CRITERIA†	4 DAY AVERAGES (µg/l) [1]
Antimony	12	0	1600
Arsenic	24	0	190 (2)
Cadmium*	52	44	0.66
Chromium (+VI)	26	0	11
Copper*	26	0	6.5
Cyanide	26	0	5.2
Lead*	46	30	1.3
Mercury II	3	33	0.012

† Concentrations used in this report are total concentrations and in this table are compared to dissolved concentrations used in chronic criteria documents.

* At a hardness of 50 mg/L of $CaCO_3$.

(1) 4 - day averages not to be exceeded more than once every 3 years.

(2) A water quality criterion exists only for Arsenic III. Observed concentrations are for total Arsenic.

INORGANIC	NUMBER OF OBSERVATIONS	% OF ACTUAL OBSERVATIONS EXCEEDING CHRONIC CRITERIA†	24 HOUR AVERAGES (µg/l) [3]
Nickel	12	0	56
Selenium	11	0	35
Silver	12	0	0.12 (4)
Zinc	18	0	47

(3) 24 hour not to be exceeded. These values are used here for inorganics for which 4 day averages are not available.

(4) Available data indicate that this is the concentration to be used for chronic toxicity.

Source: Regulatory Integration Division, Office of Policy Analysis, EPA.

adjusted for water hardness, and the percentage of those samples analyzed which were actually observed to exceed the chronic toxicity criteria. Detection limit values were not counted as exceedances.

According to TABLE 11 three metals have exceeded their criteria during several sampling events: cadmium, lead, and mercury. More than 40 percent of the cadmium samples analyzed and 30 percent of the lead samples analyzed exceeded their respective average chronic criteria. Only three samples were analyzed for mercury; more data are needed to evaluate the potential ecological impact of mercury.

This comparison is only a rough approximation of the potential effect of toxic metal concentrations on aquatic life. We emphasize that we do not know if a metal concentration exceeded a criterion for the appropriate time period corresponding to the criterion. Our observations were only grab samples taken from the Kanawha River and were not designed to determine if such concentrations persisted for a duration of time. Nonetheless, such a comparison suggests that certain pollutants are possibly exceeding their criteria and should be monitored for longer time periods.

Industry with the state is currently conducting a cooperative monitoring project. This project has monitored for metals and organics at six sampling locations from river mile 38 to river mile 91 within the study area. The purpose of this sampling is to detect seasonal variability of water concentrations. Sampling has occurred in May, August, November of 1986 and February 1987. Preliminary results are available for the May and August sampling period, (DNR, 1987). For metals we compared our water concentrations in Table 7 to the observed concentrations from the May and August sampling of the cooperative monitoring project and found the detected ranges comparable, except for cadmium and lead. The cooperative monitoring project has not detected lead nor cadmium the two metals we have identified in our analyses as chemicals requiring further investigation, during the monitoring period.

The Environmental Research Laboratory conducted ambient toxicity tests in the Charleston area of the Kanawha River in 1984. Test organisms were exposed to ambient water from the Kanawha River and were examined for young production and survival after a period of exposure. Although several sample sites did indicate toxicity and reduced young production, the overall results suggested very little ambient toxicity (Personal Communication, Donald Mount, Senior Research Scientist, to Bernard Goldstein, EPA, 1985), (Personal Communication. Scott Heinritz, Aquatic Biologist, to Eli McCoy, Division of Water Resources, DNR).

VII. ORGANICS

Industrial organic chemicals is the third pollutant category reviewed within the surface water analysis of the Kanawha Valley Toxics Study. In this section we briefly outline our methodology

to estimate potential health and ecological effects of organic chemicals in the water column.

The information for organic pollutants was even more limited than the data for pesticides, dioxin, PCBs, and metals within the Kanawha Valley study area. For example, between the years 1983 and 1985, only two sampling stations, both within the St. Albans and Nitro area, monitored for organic pollutants. No fish tissue data for organic pollutants were available, and our ability to determine the water quality for the Kanawha River within the study area was extremely limited. We therefore focused our analysis for organic pollutants on this limited segment of the Kanawha River.

For these two stations, we reviewed monitored ambient water concentrations for selected pollutants. These concentrations were compared to EPA derived ambient water quality criteria for the protection of human health at the 10^{-7}, 10^{-6}, and 10^{-5} risk levels. In addition to human health criteria, we compared observed organic chemical concentrations to organic chemical concentrations known to cause chronic and acute effects in aquatic organisms.

Sources for Organic Pollutants

Although we have measured ambient water concentrations for only a limited segment of the Kanawha River, we do know that organic chemicals are discharged from the major industrial facilities within the study area. We have reviewed discharge monitoring reports, NPDES permit applications and priority pollutant reports for the major facilities discharging into the Kanawha River for the years 1983-85. TABLE 12 lists organic pollutants which have been detected in effluents of the major facilities. Due to the limited data for organic effluent discharges, we could not quantify organic loadings to the Kanawha River.

Ambient Water Concentrations

The Ohio River Valley Water Sanitation Commission (ORSANCO) maintains an organic pollutant monitoring station in the St. Albans area, downriver from the major chemical facilities. Spill detection is the major purpose of this monitoring station. TABLE 13 presents the monitored chemicals, the concentration ranges of those chemicals, the mean concentration value for the monitored pollutants and the standard deviation. Nondetected concentrations are averaged at the detection limit. The ORSANCO station data is considered qualitative data, and may be in error by as much as 100%.

In addition to the ORSANCO river monitoring station, the West Virginia Department of Natural Resources monitored organic pollutants further down river at River Mile 38 for several years. DNR discontinued monitoring in 1984 at this station. TABLE 14 presents the concentration ranges and the mean concentration for

TABLE 12

KANAWHA VALLEY SURFACE WATER REPORT

MAJOR FACILITIES DISCHARGING ORGANICS TO THE KANAWHA RIVER

MAP KEY	FACILITY	BENZENE	BENZO(K)-FLUOR-ANTHENE	BIS(2-CHLORO-ETHYL)ETHER	BROMO-FORM	CARBON TETRA-CHLORIDE	CHLORO-FORM	CHLORO-BENZENE	CHLORO-ETHANE
7	Occidental		✓			✓	✓		
8	Dupont						✓		
11	FMC, South Charleston				✓	✓	✓		
12	CST/Fike Chemicals (Artel)			✓			✓	✓	
13	Kincaid Enterprises								
15	Monsanto	✓					✓		
16	S. Charleston Sewage Treatment Plant				✓		✓	✓	✓
17	Rhone Poulenc						✓		
19	Union Carbide, S. Charleston								

SOURCE: Discharge Monitoring Reports, NPDES permit applications and priority pollutant scans (1983-85).

TABLE 12 (continued)

KANAWHA VALLEY SURFACE WATER REPORT

MAJOR FACILITIES DISCHARGING ORGANICS TO THE KANAWHA RIVER

MAP KEY	FACILITY	DIBROMO-CHLORO-METHANE	DI-CHLORO-BENZENE	DICHLORO-BROMO-METHANE	2,4-DI-CHLORO-PHENOL	DIETHYL PHTHA-LATE	DI(ETHYL-HEXYL)-PHTHALATE	ETHYL BENZENE	ETHYLENE DICHLOR-IDE
7	Occidental								
8	Dupont								
11	FMC, South Charleston	✓		✓					
12	CST/Fike Chemicals (Artel)		✓		✓	✓	✓	✓	
13	Kincaid Enterprises								
15	Monsanto								
16	S. Charleston Sewage Treatment Plant			✓					✓
17	Rhone Poulenc								
19	Union Carbide, S. Charleston								

SOURCE: Discharge Monitoring Reports, NPDES permit applications and priority pollutant scans (1983-85).

TABLE 12 (continued)

KANAWHA VALLEY SURFACE WATER REPORT

MAJOR FACILITIES DISCHARGING ORGANICS TO THE KANAWHA RIVER

MAP KEY	FACILITY	ISOPHOR-ONE	METHYL-ENE CHLOR-IDE	METHOXY-CHLOR	NAPTH-ALENE	TETRA-CHLORO-ETHYLENE	TRI-CHLORO-ETHENE	1,1,1 TRI-CHLORO-ETHANE	1,2,4 TRI-CHLORO-BENZENE	TOLUENE	VINYL CHLORIDE	VINYL-IDENE CHLORIDE
7	Occidental		✓									
8	Dupont		✓		✓							
11	FMC, South Charleston											
12	CST/Fike Chemicals (Artel)		✓						✓	✓		✓
13	Kincaid Enterprises			✓								
15	Monsanto						✓			✓		
16	S. Charleston Sewage											
	Treatment Plant		✓			✓	✓	✓				
17	Rhone Poulenc	✓	✓									
19	Union Carbide, S. Charleston										✓	

SOURCE: Discharge Monitoring Reports, NPDES permit applications and priority pollutant scans (1983-85).

TABLE 13

KANAWHA VALLEY SURFACE WATER REPORT

AMBIENT WATER ORGANIC CONCENTRATIONS AT KANAWHA RIVER MILE 42 (ORSANCO STATION)
1983 - 1985
OBSERVATIONS = 369

ORGANIC	CONCENTRATION RANGE (ug/l)	ESTIMATED MEAN (ug/l)	STANDARD DEVIATION
BROMOFORM	<.5 - 86	8.2	10.9
CARBON TETRACHLHORIDE	<.5 - 18	0.8	1.6
CHLOROFORM	<.5 - 230	3.1	2.7
1,1-DICHLOROETHYLENE	<.5 - 29	0.7	1.7
METHYLENE CHLORIDE	<.5 - 15	0.9	1.3
TETRACHLOROETHYLENE	<.5 - 747	3.6	38.6
TRICHLOROETHYLENE	<.5 - 30	1.6	9.3
1,1-DICHLOROETHANE	<.5 - 14	1.7	1.0
1,2-DICHLOROETHYLENE	<.5 - 7	0.8	0.8
1,2-DICHLOROETHANE	<.5 - 41	1.7	3.2
1,1,1-TRICHLOROETHANE	<.5 - 18	1.0	1.7
BROMODICHLOROMETHANE	<.5 - 96	1.8	5.6
1,2-DICHLOROPROPANE	<.5 - 49	1.2	3.4
1,3-DICHLOROPROPENE	<.5 - 29	0.8	1.9
BENZENE	<.5 - 19	1.7	2.1
TOLUENE	<.5 - 137	6.5	16.9
CHLOROBENZENE	<.5 - 28	1.7	2.7
ETHYLBENZENE	<.5 - 39	1.5	3.0

[1]A less than sign (<) indicates that the concentration is less than the detection limit.

[2]The average is based on all sample concentrations between 1983 and 1985. Values below the detection limit are averaged at the detection limit concentration.

SOURCE: Ohio River Organics Detetion System, ORSANCO, 1986

TABLE 14

KANAWHA VALLEY SURFACE WATER REPORT

AMBIENT WATER ORGANIC CONCENTRATIONS AT KANAWHA RIVER MILE 38 (KRM38)
1983-1984
OBSERVATIONS = 131

ORGANIC	CONCENTRATION RANGE (ug/l)	ESTIMATED MEAN (ug/l)[2]	STANDARD DEVIATION
DICHLOROBROMOMETHANE	<.1 - 1.0	0.22	0.21
BROMOFORM	<.1 - 0.7	0.12	0.08
CHLORODIBROMOMETHANE	<.1 - 0.7	0.13	0.10
CHLOROFORM	<.1 - 9.5	1.32	1.78
CARBON TETRACHLORIDE	<.1 - 2.2	0.61	0.52
METHYLENE CHLORIDE	<.1 - 4.6	0.19	0.48
TETRACHLOROETHYLENE	<.1 - 6.5	0.29	0.63
TRICHOLROFLUOROMETHANE	<.1 - 5.8	0.17	0.52
1,1-DICHLOROETHANE	<.1 - 0.5	0.11	0.04
1,1-DICHLOROETHENE	<.1 - 4.8	0.36	0.57
1,1,1-TRICHLOROETHANE	<.1 - 6.2	0.35	0.75
1,2-DICHLOROETHANE	<.1 - 3.9	0.3	0.46
1,2-DICHLOROPROPANE	<.1 - 5.4	0.2	0.48
TRICHLOROETHYLENE	<.1 - 2.3	0.26	-
CHLOROBENZENE	<.1 - 3.9	1.4	0.42

[1]A less than sign (<) indicates that the concentration is less than the detection limit.

[2]The average is based on all sample concentrations between 1983 and 1984. Values below the detection limit are averaged at the detection limit concentration.

SOURCE: Department of Natural Resources, West Virginia (Storet, 1986)

the years 1983 and 1984. Again, nondetected concentrations are averaged at the detection limit concentration. Therefore these mean concentrations are likely to overstate the true mean value.

When the concentration ranges and mean concentrations from the two monitoring stations are compared, we notice that concentrations are higher at the ORSANCO monitoring station than the DNR station further downstream. Volatilization of these organics from the water column, dilution by tributaries as organic chemicals travel downstream, different sampling techniques, and time periods may account for the concentration differences.

Human and Aquatic Health Effects

TABLE 15 presents water quality criteria for the protection of human health from consumption of contaminated fish tissue, and observed concentration values which have produced harmful effects on aquatic organisms. The water quality criteria were developed by the Office of Research and Development within EPA and published by the Office of Water. These values, based on pollutant-specific bioconcentration factors and a national average individual daily consumption rate, attempt to provide water quality concentrations at which a pollutant may bioaccumlate in fish to various concentrations levels. These water quality criteria are water concentrations representing various levels of 10^{-5}, 10^{-6}, and 10^{-7} risk. In TABLE 15 we present the water quality criteria which represent an estimated conservative lifetime average risk of 10^{-6} for fish consumption.

The values to protect aquatic life are not criteria. For many organic chemicals there are not enough data to establish a water quality criteria for protection of aquatic life. These values represent the lowest observed concentrations to date that have produced harmful effects to aquatic organisms. Only acute effects have been studied for many organic pollutants.

We compared these criteria and observed effect concentrations of TABLE 15 to the observed concentration ranges at the ORSANCO station. TABLE 16 presents the percentage exceedances. We found four pollutants for which the human health criteria set at the 10^{-5} risk level were exceeded; chloroform, tetrachloroethylene, trichloroethylene, and 1,1-dichlorethylene. However, each of these exceedances represents only one (or 0.3%) observation exceeding the criteria. At the 10^{-6} risk level, we find the criteria exceeded by seven pollutants; the four mentioned above plus bromoform, carbon tetrachloride, and bromodichloromethene. All of the pollutants sampled exceeded the criteria set at the 10^{-7} risk level, some quite consistently such as 1,1-dichlorethane (97.9%). Of the three pollutants looked at for noncancer health effects, 1,1,1-Trichloroethane (11.1%) and toluene (3.4%) showed exceedances of the threshold levels. The observed toxic concentrations for aquatic organisms were never exceeded.

TABLE 15

WATER QUALITY CRITERIA FOR THE
PROTECTION OF HUMAN HEALTH AND POTENTIAL TOXIC
WATER CONCENTRATIONS TO AQUATIC ORGANISMS

INTENDED FOR POLICY DEVELOPMENT •

Organic	Ambient Water Concentration Associated With An Individual Lifetime Risk of 10^{-6} For An Assumed Level Of Fish Consumption[1] (µg/l)	Weight of Evidence for Carcinogens	Lowest Observed Toxic Water Concentrations Harmful To Aquatic Organisms (µg/l) Acute	Chronic
Dichlorobromomethane	15.7		11,000	NA
Bromoform	15.7		11,000	NA
Chlorodibromomethane	15.7		11,000	NA
Chloroform	15.7	B2	28,900	1,240[1]
Carbon Tetrachloride	6.94	B2	35,200	NA
Methylene Chloride	15.7	B2	11,000	NA
Tetrachloroethylene	8.85	B2	5,280	840
Trichlorofluoromethane	15.7		11,000	NA
1,1 Dichloroethane	NC		NA	NA
1,1 Dichloroethylene	1.85	C	11,600	NA
1,2 Dichloroethane	243.0	B2	118,000	20,000
1,2 Dichloropropane	NC	C	23,000	5,700
Trichloroethylene	80.7	B2	45,000	21,900[2]
Benzene	40.0	A	5,300	NA
1,3 Dichloropropane	NC		23,000	5,700
1,2 Dichloroethene	NC		NA	NA

	Ambient Water Concentration Associated With Noncancer Effects For An Assumed Level Of Fish Consumption (µg/l)		
1,1,1 Trichloroethane	1.03	18,000	NA
Toluene	424,000	17,500	NA
Chlorobenzene	488.0	250	NA

NC = No Criteria has been calculated for these chemicals.

NA = Not Available.

[1]The Office of Water has set these levels of ambient water concentrations to protect human health at the 10-6 level of risk from consumption of fish tissue, except where noted. Other concentration criteria are set at higher and lower risk levels. To calculate a risk level one order of magnitude higher, increase the concentration by one order of magnitude.

THE UNIT RISK FACTORS USED IN DETERMINING THESE CONCENTRATIONS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE SIGNIFICANTLY LOWER; IN FACT, THEY COULD BE ZERO. THE PROPER FUNCTION OF THE ESTIMATE IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES FOR THE TOPICS EXAMINED.

[1]This concentration is a 27 day LC50 value.

[2]Chronic criteria has not been established; this concentration adversely affected the behavior of one species.

Source: Office of Water. EPA.

TABLE 16

KANAWHA VALLEY SURFACE WATER REPORT

THE PERCENT OF OBSERVATIONS
AT ORSANCO WHICH EXCEED WATER QUALITY CRITERIA
FOR THE PROTECTION OF HUMAN HEALTH AT 10^{-5}, 10^{-6}, 10^{-7}
LEVEL OF RISK AND NONCANCER HEALTH EFFECTS
FROM CONSUMPTION OF FISH TISSUE

% OBSERVATIONS EXCEEDING HUMAN HEALTH CRITERIA SET AT:

	RISK LEVELS			NONCANCER HEALTH EFFECTS THRESHOLD
ORGANIC	10^{-5}	10^{-6}	10^{-7}	
Bromoform	0%	19.0%	62.5%	
Chloroform	0.3%	2.9%	35.9%	
Tetrachloroethylene	0.3%	3.2%	29.0%	
Carbon Tetrachloride	0%	1.3%	11.3%	
Trichloroethylene	0.3%	1.6%	29.0%	
Bromodichloromethane	0%	0.8%	22.2%	
Methylene Chloride	0%	0%	12.9%	
1,1-Dichloroethylene	0.3%	13.4%	97.9%	
1,2-Dichloroethane	0%	0%	0.5%	
Benzene	0%	0%	7.7%	
1,1,1-Trichloroethane				11.1%
Toluene				3.4%
Chlorobenzene				0%

Source: Office of Policy Analysis, Regulatory Integration Division, EPA.

Again, the reader is reminded that conservative assumptions were used in the determination of risk levels. Due to these assumptions and the limited nature of the ORSANCO station data, the actual percentage of exceedances are likely to be lower than TABLE 16 indicates. Furthermore, these water concentration which correspond to various risk levels must, in theory, be maintained over a period of a lifetime to pose the associated cancer risk. The percent exceedances do not indicate a level of risk, but only serves as a way to compare pollutants to each other. The actual risks posed by organics require much more detail. The final results of the cooperative monitoring program should be reviewed, when available, to determine the frequency of exceedances of these pollutants.

SUMMARY AND CONCLUSIONS

In the Kanawha Valley Surface Water Report we have attempted to develop a sense of the potential public health concerns toxic pollutants may pose within the Kanawha River. We emphasize that we examined only one exposure pathway, fish tissue consumption, within the study area. We also emphasize that our analysis is extremely limited due to limited health effects and exposure information. An assessment of potential cancer risk and other potential noncancer health risk posed to a known population is not possible given our inability to characterize actual exposure within the study area. Furthermore, monitoring data are limited in time as well as area with much of the information drawn from monitoring stations on the lower Kanawha River in the study area. Our health effects analysis of pesticides, dioxin, PCB's and metals is based on a one day sampling of fish fillet in the lower Kanawha River while our analysis of industrial discharged organics relies on observed water concentrations from two stations below many of the industrial facilities on the Kanawha River.

Despite these limitations, we believe our limited analysis may provide a general sense of the potential health hazards and may provide some direction and priorities in the collection of data and information needed for assessment of the effects of toxics within the Kanawha River.

We present the following findings from this analysis:

- DNR of West Virginia has detected both pesticides and metal concentrations within fish tissue from samples taken throughout the study area.

  A review of current discharge permits indicate that manufactured pesticides in the Kanawha are not discharged and, therefore, pesticide concentrations within fish tissue are likely a result of non-point source run-off.

  Metal loadings occur from both point and non-point sources within the study area. Based on our review of non-point source information and discharge permits, our analysis

indicates that non-point source run-off, is likely to contribute larger loadings than point sources for most metals. Cadmium is an exception. TABLE 5 presents 22 metals point source discharges.

Industrial discharged organics have been detected in the effluents of 9 point source discharger in the study area (See Table 12). In addition, organics have been detected in the water column at two monitoring stations on the lower Kanawha River in the study area. TABLE 13 and 14 present organics detected.

- Based on a one day sampling on the Nitro area of the Kanawha River, we calculated a weekly consumption rate of .1 to 1 pound, depending on the fish caught (Smallmouth Buffalo, Channel Catfish, Sauger, Largemouth Bass, Spotted Bass, Smallmouth Bass, and Freshwater Drum) which would expose an individual to an upper-bound incremental lifetime risk of 1×10^{-3} or one out of thousand chances of contracting cancer over a lifetime exposure. Dioxin and PCBs are the pollutants primarily responsible for the risks at these various hypothetical consumption rates. We do not know if these concentration levels occur throughout the river.

  For organics, we compared water concentrations obtained from the lower Kanawha River to various levels of water quality criteria set to protect human health. These criteria assume that the average individual consumed approximately one tenth of a pound of fish per week, a rate calculated for the analysis above. Two pollutants, bromoform and 1,1-dichloroethylene exceeded the criteria concentrations to protect human health at the 1×10^{-6} (one out of a million) for more than 10 percent of the observations. Only one observations for each of these pollutants exceeded the concentration representing a 1×10^{-5} (one out of one hundred thousand) risk.

- We calculated consumption rates required to exceed EPA established reference doses for chronic non-cancer health effect for chlordane, which was detected in a one day sampling in the Nitro area. We found that consumption rates ranged from .3 pound per week to eighteen pounds per week, for certain species caught, which would exceed the RfD for chlordane, a nonpoint pollutant. Chlordane was not detected in Small Mouth Bass nor Spotted Bass.

  For mercury, we assumed that the organic mercury was methyl mercury. If an individual were to consume the highest observed concentration for organic mercury, at a consumption rate of one pound per week, the RFD would be exceeded.

- To develop a rough approximation of the potential effect of toxic metal concentration on aquatic life we compared the number of monitored ambient water concentrations of a limited set of metals to their corresponding chronic water quality criteria for the protection for aquatic organisms. During the monitoring period, lead and cadmium concentrations exceeded their criteria 44 and 30 percent of their observations respectively. Cadmium comes predominately from industrial discharges while lead comes from non-point source run-off.

- We compared our limited set of observed organic water concentrations from the lower Kanawha River to the lowest observed toxic water concentrations harmful to aquatic organisms, a proxy for a water quality criteria to protect human health. These levels were not exceeded by the detected concentrations.

DATA GAPS

- We have investigated only one type of exposure, the potential risk from the consumption of fish tissue. There are several other routes of exposure which should be investigated. Dermal contact, water ingestion, and consumption of water fowl are other routes of exposure from pollutants within the Kanawha River.

- Our data on fish fillet concentrations is currently limited to the Nitro Area within the study area. Since whole fish data indicate potential bioaccumulation of many of these pollutants, fillet data should be obtained for the upper reaches of the river. Our analysis suggests that fish tissue contamination by PCBs, dioxin, chlordane, and mercury may pose more of a concern than other pollutants for which we have health effects data.

- The consumption rate of fish tissue is not currently known. The West Virginia DWR Creel Survey will provide consumption information about fish caught from the Kanawha River.

- Recently the State in cooperation with industry has monitored for priority pollutants throughout the entire study area. The database should provide much needed monitoring data on organic and inorganic pollutants in the Kanawha River.

- Finally, health effects information of these pollutants should be continually reviewed as new data and knowledge are developed. Current Reference Dose values for noncancer health effects and cancer potency values may change, and values for new pollutants may be developed. Due to the evolving nature of the knowledge of health effects information, exposure concentration should be continually reviewed with new health effects information.

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Appendix A

Kanawha Point and Non-Point Source Loadings

This document outlines the procedures followed in computing both the annual non-point source loadings and point source loadings to the Kanawha River. These results were presented in the Kanawha Valley Surface Water Analysis Table 2 along with the ratio of non-point to point source loadings.

Non-Point Source Loadings

The non-point source loadings for each pollutant are estimated by multiplying the volume of water that flows into the river via runoff, by the average concentration of each pollutant in stormwater runoff. These calculations were based on data from both the 1981 Geographic Study of the Kanawha Valley (Draft EPA 1981) and the National Urban Stormwater Runoff Program (NURP) (EPA 1982). The NURP data included the mean concentrations (ug/l) of the most frequently detected priority pollutants in NURP urban stormwater runoff. The average flow of runoff to each segment of the river was taken from the 1981 Draft Geographic study.

Although our estimates for non-point source loadings are based on national data, we believe these estimates provide a sense of the possible magnitude of non-point source runoff. Furthermore, these are the best available date for estimating loadings

The NURP Program was conducted from 1980 to 1982, with runoff samples being collected from 51 predominantly residential and commercial areas. Therefore, the results from the NURP effort cannot be attributed to runoff from industrial facilities. Attachment 1 presents the average concentration of heavy metal contaminants from the NURP data set.

The major limitation of the NURP data is that urban areas, with different land uses than in the Kanawha Study area, were sampled to generate the average concentrations. These data probably do not adequately simulate the runoff from the industrial complexes that border the Kanawha River. In addition, loadings of toxic organics such as pesticides and PCBs cannot be quantified using NURP data. Also the large number of CERCLA hazardous waste sites in the study area could contribute a large amount of these types of toxic organics to the river by stormwater runoff. In addition, NURP data is only considered to be accurate to within one order of magnitude.

To develop run-off flows, we used the 1981 Draft Geographic study. In this study, the river was divided into 18 distinct reaches. The division of the river into these reaches reflected discontinuities due to dams, tributaries, and the depth of the river. The direct runoff flow for these 18 reaches was determined as follows. The drainage area of the Kanawha, as well as the total drainage area of tributaries, is known at various points along the river. The drainage area is the watershed area that contributes to the river, i.e. the area where the land slopes toward the Kanawha river and runoff could reasonably be expected to flow into the Kanawha river. The drainage area of the upriver station was subtracted from the downriver station, then the tributary total drainage area was subtracted from that difference leaving the drainage area for direct runoff. The average annual rainfall is known, as is the fraction of that rainfall that runs off (instead of evaporating or infiltrating the ground). The volume of direct runoff is then calculated by the following equation:

$$q = cia \text{ (Merritt 1968)}$$

where i is the rainfall, a is the drainage area and c is the fraction of rain that runs off. This direct runoff was then divided among the 18 reaches depending on the length of each reach. The runoff volumes per reach are presented in attachment 2.

Since the eighteen reaches developed for the 1981 study do not correspond to the five segments in the present analysis, we modified the runoff flow to reflect the new river segment in the following manner. We assigned each of the 1981 reaches to the corresponding river segment in the present study area based on river miles covered:

Segment 1 - Reaches 1-5
Segment 2 - Reaches 6-9
Segment 3 - Reaches 10-11
Segment 4 - Reaches 12-13
Segment 5 - Reaches 14-16

Reaches 17 and 18 are outside the study area. We then divided each runoff flow by its corresponding reach length to give a runoff per mile for each reach. These were then averaged over all the reaches in a segment to give average runoff per mile for every segment. These numbers were then multiplied by the segment length to give average runoff flow in m^3/hr for each segment.

To determine the loadings, the average runoff flow in each segment, described above, was multiplied by the mean concentration of each pollutant from the NURP stormwater runoff data (in cases where a range of concentrations was given, we used the midpoint of the range) along with a conversion factor to give a loading in kkg/yr for each pollutant in each segment. The five segment loadings for each pollutant were summed to give the total non-point source loading to the river of each pollutant.

Point Source Loadings

We developed average loadings for 1984. The only water data available for the Kanawha study area comes from monthly discharge monitoring reports compiled for the National Pollution Discharge Elimination System (NPDES). We first determined, for each outfall which is monitored, the average flow for each month. We then multiplied the reported concentrations in a month by the average flow for that same month to get a loading. We then averaged all the loadings together to get a yearly average. In cases where there are multiple concentration readings for a pollutant in the same month, we kept these distinct when computing the loadings. As a result, the yearly average may be composed of more than 12 individual readings; or conversely, in cases where data for some months was missing, the averages may be composed of less than 12 individual readings.

Attachment 3 lists the loadings by pollutant. Under each pollutant heading it lists the facility outfall number and average yearly loading. We then summed the individual loadings for each pollutant to give the total point source loading to the river.

References

1) U.S. EPA "An Integrated Geographic Study of Potential Toxic Substance Control Strategies in the Kanawha River Valley, West Virginia" Interim Draft Report, July 15, 1981.

2) U.S. EPA, Monitoring and Data Support Division, "NURP Priority Pollutant Monitoring Program Volume I: Findings", September 17, 1982.

3) Merritt, Frederick S. Standard Handbook for Civil Engineers, McGraw-Hill Book Co., New York, 1968, P 14-7.

Attachment 1

Most Frequently Detected Priority Pollutants in NURP Urban Stormwater Runoff Samples[1]

Pollutant	Detection Frequency (%)	Mean Concentration[2] (ug/l)
Copper	96	18.1
Lead	96	65.5
Zinc	95	92.3
Arsenic	58	1.60
Chromium	57	3.13
Cadmium	55	0.57
Nickel	48	5.80
α-Hexachlorocyclohexane	20	0.093
Selenium	19	(2-77)[3]
Beryllium	17	(1-49)[3]
Cyanides	16	(2-33)[3]
Mercury	16	(0.5-1.2)[3]
Pentachlorophenol	15	(1-115)[3]
Antimony	14	(2.6-23)[3]
Bis(2-ethylhexyl)phthalate	13	(7-39)[3]
Silver	12	(0.2-0.8)[3]
Trichloromethane (chloroform)	12	(0.2-12)[3]
Phenanthrene	12	(0.3-10)[3]
β-Hexachlorocyclohexane (Lindane)	12	(0.52-0.1)[3]
Napnthalene	11	(0.8-2.3)[3]
Pyrene	11	(0.3-10)[3]
Thallium	10	(1-14)[3]
Dichloromethane (methylene chloride)	10	(5-14.5)[3]
Fluoranthene	10	(0.3-12)[3]

Source: DON (1982).

1 Pollutants detected in at least 10% of all samples analyzed (86).

2 Geometric mean concentration. Calculated only if pollutant is detected in at least 20% of the acceptable samples. Non-detections were included in calculation as one-tenth of the reported detection limit.

3 Mean concentration not calculated. Range of detected values only reported. For purposes of initial scan analysis the mid-point of the range was used to estimate annual pollutant loads.

ATTACHMENT 2

	Reach (mi)	Direct Runoff (m^3/hr)
1)	73.7 - 69.4	6.66×10^3
2)	69.4 - 68.0	2.17×10^3
3)	68.0 - 67.7	4.64×10^2
4)	67.7 - 66.1	2.48×10^3
5)	66.1 - 57.8	1.28×10^4
6)	57.8 - 56.3	2.32×10^3
7)	56.3 - 54.3	3.09×10^3
8)	54.3 - 52.9	1.49×10^3
9)	52.9 - 49.6	3.52×10^3
10)	49.6 - 48.0	1.71×10^3
11)	48.0 - 45.5	2.67×10^3
12)	45.5 - 43.5	2.13×10^3
13)	43.5 - 41.4	2.24×10^3
14)	41.4 - 39.3	2.24×10^3
15)	39.3 - 32.0	1.83×10^3
16)	32.0 - 31.1	2.25×10^2
17)	31.1 - 18.0	2.82×10^4
18)	18.0 - 0.0	3.88×10^4

Attachment 3

KANAWHA VALLEY, WV STUDY AREA
TOOL 191: MEAN LOADINGS IN KGD BASED ON DMR DATA ONLY

YEAR=84 POLLUTANT=ANTIMONY (SB)

CODE	FACILITY	PIPE	KGD
WV0000078	CARBIDE-SUI DIVISION	24	.
WV0000086	CARBIDE-INSTITUTE	01	0.10196
WV0000086	CARBIDE-INSTITUTE	05	0.80186
WV0000167	ELKEM METALS	06	0.00005
WV0001074	APPALACHIAN 1074-AMOS	04	0.01363
WV0001074	APPALACHIAN 1074-AMOS	11	0.08631
WV0001074	APPALACHIAN 1074-AMOS	27	0.00908
WV0002399	DUPONT -BELLE	62	.
POLLUTANT			1.01289

YEAR=84 POLLUTANT=ARSENIC

CODE	FACILITY	PIPE	KGD
WV0000086	CARBIDE-INSTITUTE	01	0.17112
WV0000086	CARBIDE-INSTITUTE	05	0.26729
WV0000167	ELKEM METALS	06	0.00002
WV0000400	FMC-NITRO	01	1.15589
WV0000400	FMC-NITRO	02	0.01730
WV0000442	FMC-S.CHARLESTON	29	0.56944
WV0001066	APPALACHIAN 1066-KANAWHA	01	0.02030
WV0001074	APPALACHIAN 1074-AMOS	01	1.36078
WV0001074	APPALACHIAN 1074-AMOS	03	0.17205
WV0001074	APPALACHIAN 1074-AMOS	04	0.01817
WV0001074	APPALACHIAN 1074-AMOS	11	0.00303
WV0001074	APPALACHIAN 1074-AMOS	27	0.01514
WV0001651	FIKE CHEMICAL-C.S.T.	01	0.02833
WV0002381	HATFIELD&HENSON	01	0.14839
WV0002399	DUPONT -BELLE	062	.
POLLUTANT			3.94724

YEAR=84 POLLUTANT=BARIUM

CODE	FACILITY	PIPE	KGD
WV0001651	FIKE CHEMICAL-C.S.T.	01	0.04994

YEAR=84 POLLUTANT=CADMIUM

CODE	FACILITY	PIPE	KGD
WVI623382	SMITH FASTENER	01	0.0000
WV0000086	CARBIDE-INSTITUTE	01	0.4758
WV0000086	CARBIDE-INSTITUTE	03	1.6797
WV0000086	CARBIDE-INSTITUTE	05	88.2047
WV0000167	ELKEM METALS	06	0.0000
WV0001074	APPALACHIAN 1074-AMOS	04	0.0227
WV0001074	APPALACHIAN 1074-AMOS	11	0.0076
WV0001074	APPALACHIAN 1074-AMOS	27	0.0151
WV0001651	FIKE CHEMICAL-C.S.T.	01	0.0029
POLLUTANT			90.4087

YEAR=84 POLLUTANT=CHROMIUM TOTAL

CODE	FACILITY	PIPE	KGD
WV0000078	CARBIDE-SUI DIVISION	16	.
WV0000078	CARBIDE-SUI DIVISION	17	.
WV0000078	CARBIDE-SUI DIVISION	18	.
WV0000078	CARBIDE-SUI DIVISION	19	.
WV0000078	CARBIDE-SUI DIVISION	22	.
WV0000078	CARBIDE-SUI DIVISION	23	.
WV0000078	CARBIDE-SUI DIVISION	24	.
WV0000078	CARBIDE-SUI DIVISION	28	.
WV0000078	CARBIDE-SUI DIVISION	29	.
WV0000078	CARBIDE-SUI DIVISION	32	.
WV0000078	CARBIDE-SUI DIVISION	53	.
WV0000078	CARBIDE-SUI DIVISION	74	.
WV0000078	CARBIDE-SUI DIVISION	80	.
WV0000086	CARBIDE-INSTITUTE	02	.
WV0000167	ELKEM METALS	02	0.019491
WV0000167	ELKEM METALS	03	0.000000
WV0000167	ELKEM METALS	05	0.000000
WV0000167	ELKEM METALS	06	0.000000
WV0001074	APPALACHIAN 1074-AMOS	04	0.136274
WV0001074	APPALACHIAN 1074-AMOS	11	0.045425
WV0001074	APPALACHIAN 1074-AMOS	27	0.090850
POLLUTANT			0.292040

YEAR=84 POLLUTANT=CHROMIUM HEXAVALENT

CODE	FACILITY	PIPE	KGD
WV0000400	FMC-NITRO	01	0.000081
WV0000400	FMC-NITRO	02	0.000000
WV0000442	FMC-S.CHARLESTON	22	0.014599
WV0000442	FMC-S.CHARLESTON	23	0.009234
WV0000442	FMC-S.CHARLESTON	29	0.231558
WV0000868	MONSANTO	01	0.453600
WV0001651	FIKE CHEMICAL-C.S.T.	01	0.003539
WV0002372	CHEM LEAMAN TANK LINES	01	0.045425
WV0050130	COASTAL TANK LINES	01	0.000325
POLLUTANT			0.758361

YEAR=84 POLLUTANT=COPPER

CODE	FACILITY	PIPE	MGD
WVI623382	SMITH FASTENER	01	0.00081
WV0000078	CARBIDE-SUI DIVISION	16	.
WV0000078	CARBIDE-SUI DIVISION	17	.
WV0000078	CARBIDE-SUI DIVISION	18	.
WV0000078	CARBIDE-SUI DIVISION	19	.
WV0000078	CARBIDE-SUI DIVISION	22	.
WV0000078	CARBIDE-SUI DIVISION	23	.
WV0000078	CARBIDE-SUI DIVISION	24	.
WV0000078	CARBIDE-SUI DIVISION	28	.
WV0000078	CARBIDE-SUI DIVISION	29	.
WV0000078	CARBIDE-SUI DIVISION	32	.
WV0000078	CARBIDE-SUI DIVISION	53	.
WV0000078	CARBIDE-SUI DIVISION	74	.
WV0000078	CARBIDE-SUI DIVISION	80	.
WV0001066	APPALACHIAN 1066-KANAWHA	01	0.04512
WV0001074	APPALACHIAN 1074-AMOS	01	2.54250
WV0001074	APPALACHIAN 1074-AMOS	03	0.60790
WV0001074	APPALACHIAN 1074-AMOS	04	0.15899
WV0001074	APPALACHIAN 1074-AMOS	11	0.03028
WV0001074	APPALACHIAN 1074-AMOS	27	0.06057
WV0001651	FIKE CHEMICAL-C.S.T.	01	0.01727
WV0001716	PB&S CHEMICAL	01	0.22908
WV0002313	DIAMOND SHAMROCK CHEMICALS CO.	03	0.05596
WV0002381	HATFIELD&HENSON	01	0.13370
WV0002399	DUPONT -BELLE	017	.
WV0002399	DUPONT -BELLE	17	0.00000
WV0002399	DUPONT -BELLE	62	0.29572
WV0023116	S.CHARLESTON POTW	01	0.42813
POLLUTANT			4.60603

YEAR=84 POLLUTANT=CYANIDE

CODE	FACILITY	PIPE	MGD
WV0000868	MONSANTO	01	0.16260
WV0001651	FIKE CHEMICAL-C.S.T.	01	2.16875
WV0002399	DUPONT -BELLE	62	.
POLLUTANT			2.33136

YEAR=84 POLLUTANT=LEAD

CODE	FACILITY	PIPE	KGD
WVI623382	SMITH FASTENER	01	0.00002
WV0000078	CARBIDE-SUI DIVISION	24	.
WV0000078	CARBIDE-SUI DIVISION	29	.
WV0000086	CARBIDE-INSTITUTE	05	2.67287
WV0000167	ELKEM METALS	05	0.68591
WV0000167	ELKEM METALS	06	0.00003
WV0000442	FMC-S.CHARLESTON	18	0.92843
WV0000442	FMC-S.CHARLESTON	29	0.05762
WV0001066	APPALACHIAN 1066-KANAWHA	01	0.04794
WV0001074	APPALACHIAN 1074-AMOS	03	0.10323
WV0001074	APPALACHIAN 1074-AMOS	04	0.04997
WV0001074	APPALACHIAN 1074-AMOS	11	0.03331
WV0001074	APPALACHIAN 1074-AMOS	27	0.01211
WV0001651	FIKE CHEMICAL-C.S.T.	01	0.03342
WV0002381	HATFIELD&HENSON	01	0.04353
POLLUTANT			4.66841

YEAR=84 POLLUTANT=MERCURY

CODE	FACILITY	PIPE	KGD
WV0000167	ELKEM METALS	06	0.00049210
WV0001066	APPALACHIAN 1066-KANAWHA	01	.
WV0001074	APPALACHIAN 1074-AMOS	03	0.00458790
WV0001651	FIKE CHEMICAL-C.S.T.	01	0.00332506
WV0002313	DIAMOND SHAMROCK CHEMICALS CO.	03	0.00009993
POLLUTANT			0.00850500

YEAR=84 POLLUTANT=NICKEL

CODE	FACILITY	PIPE	KGD
WV0000078	CARBIDE-SUI DIVISION	18	.
WV0000078	CARBIDE-SUI DIVISION	19	.
WV0000078	CARBIDE-SUI DIVISION	22	.
WV0000078	CARBIDE-SUI DIVISION	24	.
WV0000078	CARBIDE-SUI DIVISION	28	.
WV0000078	CARBIDE-SUI DIVISION	29	.
WV0000086	CARBIDE-INSTITUTE	01	0.08496
WV0000086	CARBIDE-INSTITUTE	02	5.40858
WV0000086	CARBIDE-INSTITUTE	03	3.05406
WV0000086	CARBIDE-INSTITUTE	05	0.53457
WV0001066	APPALACHIAN 1066-KANAWHA	01	0.05640
WV0001074	APPALACHIAN 1074-AMOS	04	0.13627
WV0001074	APPALACHIAN 1074-AMOS	11	0.03785
WV0001074	APPALACHIAN 1074-AMOS	27	0.06057
WV0001651	FIKE CHEMICAL-C.S.T.	01	0.10662
WV0002313	DIAMOND SHAMROCK CHEMICALS CO.	13	0.17294
WV0002381	HATFIELD&HENSON	01	0.03937
WV0002399	DUPONT -BELLE	62	.
WV0002542	ALLIED CHEMICAL	03	0.13299
POLLUTANT			9.82520

YEAR=84 POLLUTANT=SELENIUM

CODE	FACILITY	PIPE	MGD
WV0000078	CARBIDE-SUI DIVISION	18	.
WV0001074	APPALACHIAN 1074-AMOS	01	3.58099
WV0001074	APPALACHIAN 1074-AMOS	04	0.01817
WV0001074	APPALACHIAN 1074-AMOS	11	0.01514
WV0001074	APPALACHIAN 1074-AMOS	27	0.00606
WV0002399	DUPONT -BELLE	62	.
POLLUTANT			3.62036

YEAR=84 POLLUTANT=SILVER

CODE	FACILITY	PIPE	MGD
WV0000078	CARBIDE-SUI DIVISION	17	.
WV0000078	CARBIDE-SUI DIVISION	18	.
WV0000078	CARBIDE-SUI DIVISION	24	.
WV0000078	CARBIDE-SUI DIVISION	28	.
WV0000078	CARBIDE-SUI DIVISION	29	.
WV0000086	CARBIDE-INSTITUTE	01	0.0142596
POLLUTANT			0.0142596

YEAR=84 POLLUTANT=ZINC

CODE	FACILITY	PIPE	MGD
WV1623382	SMITH FASTENER	01	0.036
WV0000078	CARBIDE-SUI DIVISION	16	.
WV0000078	CARBIDE-SUI DIVISION	17	.
WV0000078	CARBIDE-SUI DIVISION	18	.
WV0000078	CARBIDE-SUI DIVISION	19	.
WV0000078	CARBIDE-SUI DIVISION	22	.
WV0000078	CARBIDE-SUI DIVISION	23	.
WV0000078	CARBIDE-SUI DIVISION	24	.
WV0000078	CARBIDE-SUI DIVISION	28	.
WV0000078	CARBIDE-SUI DIVISION	29	.
WV0000078	CARBIDE-SUI DIVISION	32	.
WV0000078	CARBIDE-SUI DIVISION	53	.
WV0000078	CARBIDE-SUI DIVISION	74	.
WV0000078	CARBIDE-SUI DIVISION	80	.
WV0000086	CARBIDE-INSTITUTE	01	0.156
WV0000086	CARBIDE-INSTITUTE	02	1.352
WV0000086	CARBIDE-INSTITUTE	03	0.764
WV0000167	ELKEM METALS	06	0.000
WV0001066	APPALACHIAN 1066-KANAWHA	01	0.276
WV0001074	APPALACHIAN 1074-AMOS	01	1.289
WV0001074	APPALACHIAN 1074-AMOS	03	0.241
WV0001074	APPALACHIAN 1074-AMOS	04	0.731
WV0001074	APPALACHIAN 1074-AMOS	11	0.174
WV0001074	APPALACHIAN 1074-AMOS	27	0.167
WV0002313	DIAMOND SHAMROCK CHEMICALS CO.	03	0.108
WV0002381	HATFIELD&HENSON	01	0.128
WV0002399	DUPONT -BELLE	017	.
WV0002399	DUPONT -BELLE	046	.
WV0002399	DUPONT -BELLE	62	.
WV0002542	ALLIED CHEMICAL	03	0.341
POLLUTANT			5.764

Chapter Seven

Hazardous Waste Analysis

KANAWHA VALLEY TOXICS SCREENING STUDY/ HAZARDOUS WASTE SITES

I. INTRODUCTION AND PURPOSE

The Kanawha Valley Toxics Screening Study has four stated objectives that have provided overall direction to this technical report. In this hazardous waste site analysis, we attempted to address these objectives to provide a screening analysis for policy development purposes. The four objectives are:

- Using available data bases, identify many of the chemicals routinely released or present within various exposure pathways;
- Develop a sense of the potential public health concerns these toxic pollutants may pose in various pathways, based on health effects and exposure information;
- For a select number of chemicals, provide an initial, conservative assessment of potential cancer risk and potential noncancer health risk for predicted or observed concentrations within exposure pathways; and
- Identify data and information gaps and outline needs and options for future study directions to enable a more detailed investigation of health issues where warranted.

To meet the first objective, we have reviewed available information for hazardous waste sites within the study area to determine the types of chemicals present. To develop a sense of the potential public health concerns these toxic pollutants may pose, we developed a general characterization of the hazardous waste sites within the Kanawha Valley. By organizing sites according to hydrogeology, waste, and facility classes, we developed general categories of waste sites. From these general categories we modeled several site scenarios and estimated potential ground-water contamination and potential risk posed by such contamination. It is important to emphasize that we modeled scenarios representative of sites within the study area, and <u>not</u> specific hazardous waste sites. The limited information available does not allow an accurate assessment of potential cancer risk from specific sites, even for a select number of pollutants. Through our analysis we can only develop a general sense of potential public health concerns that these toxic chemicals may pose in the Kanawha Valley. Finally, to meet the fourth objective we outlined information gaps where more data are needed.

The remainder of the report is divided into five sections. Section II describes the basic site characterization and data collection, and Section III describes the development of hydrogeology, waste, and facility categories. In the fourth section, we discuss the quantitative modeling including: development of representative generic scenarios, methods for exposure and risk modeling, and the results of the analysis. Section V contains a discussion of the limitations of the analysis and identifies areas in which further study is needed; this section also contains a summary of uncertainty issues related to ground-water modeling. The final section presents conclusions for

this phase of the analysis. There are three technical appendices to this report: Appendix A describes in detail our characterization of the hydrogeology of the Kanawha Valley, Appendix B provides a detailed discussion of uncertainty issues in the ground-water modeling, and Appendix C lists our main sources of information.

Limited time and resources required this study to focus solely on potential ground-water contamination. Although we do not review other exposure pathways such as those resulting from chemical volatilization and run-off from hazardous waste sites, these pathways should be considered in a comprehensive assessment of potential effects from hazardous waste sites.

II. HAZARDOUS WASTE SITE CHARACTERIZATION

We characterized sites according to hydrogeologic setting, exposure pathway, and predominant waste types, and grouped sites in three general hydrogeologic categories, five facility types, and four waste categories. While there are a large number of sites in the study area (numerous RCRA generators, approximately 20 RCRA treatment, storage, and disposal (TSD) facilities, and many potential CERCLA sites), we were able to obtain information relevant to risk assessment for only 25 facilities. These facilities include 8 RCRA TSD facilities, 7 RCRA generators, and 10 potential CERCLA sites. Our characterization of these facilities is summarized in Table 1. Figure 1 is a map of the study area showing the locations of the 25 sites.

As indicated in Table 1, each site was identified by name and location, and an attempt was made to classify each site according to whether it was a RCRA or CERCLA facility. While this was obvious in most cases, some sites are active or inactive RCRA facilities that are under investigation as potential CERCLA sites (e.g., Occidental, FMC Corp.). In one case an apparently active RCRA facility, Artel, is also on the CERCLA National Priorities List (NPL). In our characterization we assigned to the RCRA category facilities that had applied for a RCRA permit or were known to generate RCRA wastes; sites used for the dumping of wastes (e.g., municipal landfills) were classified as potential CERCLA sites. Using this methodology, Artel is considered a RCRA site in our characterization.

Based on its location, we assigned each site to one of three general hydrogeologic settings that we developed for the region: (1) Sandstone/Shale-Clymer/Dekalb/Gilpin soil setting, found in the southeast portion of the study area; (2) Shale/Sandstone-Gilpin/Upshur/Vandalia Soil setting found in the northwest portion of the study area; and (3) Alluvium-Kanawha Soil setting, found along the Kanawha River and its major tributaries. These hydrogeologic settings are described in greater detail in Appendix A, "Hydrogeologic Characterization of the Kanawha Valley," and in Section III of this report.

TABLE 1

KANAWHA VALLEY TOXICS SCREENING STUDY/HAZARDOUS WASTE SITES

RCRA AND POTENTIAL CERCLA SITES WITH SUFFICIENT INFORMATION FOR PRELIMINARY RISK ANALYSIS

SITE NAME	LOCATION	RCRA/CERCLA	HYDROGEOLOGIC SETTING	POTENTIAL EXPOSURE PATHWAY	PREDOMINANT WASTES	COMMENTS
1. Heizer Creek Dump	Nitro, WV	CERCLA	Shale/Sandtone - Gilpin-Upshur-Vandalia (GUV) Soils	Consumption of contaminated ground water-private wells	Phenol; N-nitrosodiphenylamine; cresol; tetrachlorobenzene; trichlorobenzene; methylene chloride; 2,4,6-trichlorophenol; cyanide; arsenic (possibly dioxins)	Former disposal area for industrial wastes; ground-water users currently exposed
2. South Charleston Municipal Landfill	South Charleston, WV	CERCLA	Shale/Sandstone-GUV Soils	Consumption of contaminated ground water-private wells	Dichlorobenzene; methylene chloride; aldrin; PCBs; MEK; phenol; cresol; toluene; chlordane	Possible exposure at present time to methylene chloride, chlordane; former industrial waste disposal site
3. Smith Creek Dump	South Charleston, WV	CERCLA	Shale/Sandstone-GUV Soils	Consumption of contaminated ground water-private wells	Bis(2-chloroethoxy)methane; naphthalene; PCBs; chromium; lead; cyanide; TCE	Former industrial waste disposal site (near South Charleston Municipal Landfill)
4. Elkem Metals	Alloy, WV	RCRA generator	Alluvium-Kanawha Soils	Ground-water discharge to Kanawha River-may affect Boomer water supply	Chromium; copper; lead; zinc	
5. Occidental	Belle, WV	RCRA generator/ CERCLA	Alluvium-Kanawha Soils	Ground-water discharge to Kanawha River	Carbon tetrachloride; chloroform; methylene chloride; 2,4- and 2,6-dinitrotoluene; mercury; chromium; hexachloroethane; hexachlorobenzene	Former surface impoundment; ground-water pumping and treatment in progress
6. E.I. DuPont	Belle, WV	RCRA	Alluvium-Kanawha Soils	Ground-water discharge to Kanawha River	Methylene dianiline; nitrosoamines; methacrylate; alcohols; thorium; arsenic; hexamine	Inactive disposal areas
7. Union Carbide	South Charleston, WV	RCRA	Alluvium-Kanawha Soils	Ground-water discharge to Kanawha River	Acrylonitrile; benzene; vinyl chloride a/	
8. FMC Corp., Industrial Chemicals	South Charleston, WV	RCRA/ CERCLA	Alluvium-Kanawha Soils	Ground-water discharge to Kanawha River	Mercury; cadmium; lead; cyanide; lindane; carbon tetrachloride; chloroform; carbon disulfide; MEK; phenol; cresol; trichlorobenzene	Inactive landfills at facility

TABLE 1 (Continued)

KANAWHA VALLEY TOXICS SCREENING STUDY/HAZARDOUS WASTE SITES

RCRA AND POTENTIAL CERCLA SITES WITH SUFFICIENT INFORMATION FOR PRELIMINARY RISK ANALYSIS

SITE NAME	LOCATION	RCRA/CERCLA	HYDROGEOLOGIC SETTING	POTENTIAL EXPOSURE PATHWAY	PREDOMINANT WASTES	COMMENTS
9. Union Carbide, Technical Center	South Charleston, WV	RCRA	Sandstone/Shale-Clymer-Dekalb-Gilpin (CDG) Soils	Consumption of contaminated ground water-private wells	Highly variable as site is a research and development facility	Holz Pond (surface impoundment) located nearby
10. Rhone-Poulenc	Institute, WV	RCRA	Alluvium-Kanawha Soils	Ground-water discharge to Kanawha River	Anthracene; chloroform; chlorobenzene; fluoranthene; N-nitrosodiphenylamine; 1,1,1-TCA; carbon tetrachloride; toluene; ethylbenzene; methylene chloride	Includes Golf Mt. Landfill; Private Trucking Operations (PTO) located nearby; PTO has documented ground-water contamination
11. Chemical Leaman Tank Lines	Institute, WV	RCRA generator	Alluvium-Kanawha Soils	Ground-water discharge to Kanawha River	Phenol; chromium	Tank cleaning facility; no on-site disposal
12. Big Scary Creek Pond	Nitro, WV	CERCLA	Alluvium-Kanawha Soils	Ground-water discharge to Kanawha River	Zinc; unknown organics and heavy metals	Viscose rayon plant; inactive disposal sites (unauthorized) at facility
13. FMC Corp., Specialty Chemical	Nitro, WV	RCRA	Alluvium-Kanawha Soils	Ground-water discharge to Kanawha River	1,1,1-TCA; arsenic; phenol; chromium	2 inactive landfills on-site
14. Monsanto Company	Nitro, WV	RCRA	Alluvium-Kanawha Soils	Ground-water discharge to Kanawha River	2,4,5-T; dioxin; acrolein; TCE; PCE; cyanide; toluene; xylene; chlorobenzene; heavy metals	3 inactive landfills on-site; documented ground-water contamination
15. Artel	Nitro, WV	RCRA (permit denied); CERCLA (National Priorities List);	Alluvium-Kanawha Soils	Ground-water discharge to Kanawha River	Benzene; bis(2-chloroethyl) ether; bis(2-chloroisopropyl) ether; chloroform; 1,2-dichloethane; vinyl chloride; arsenic; ethylbenzene; PCE; carbon tetrachloride; PAHs; DDE; DDD; heptachlor; lindane; mercury; isophorone; lead; cadmium; cobalt; nickel	4 "evaporation" ponds; unlined burial pits; highly contaminated ground water

TABLE 1 (Continued)

KANAWHA VALLEY TOXICS SCREENING STUDY/HAZARDOUS WASTE SITES

RCRA AND POTENTIAL CERCLA SITES WITH SUFFICIENT INFORMATION FOR PRELIMINARY RISK ANALYSIS

SITE NAME	LOCATION	RCRA/CERCLA	HYDROGEOLOGIC SETTING	POTENTIAL EXPOSURE PATHWAY	PREDOMINANT WASTES	COMMENTS
16. Coastal Tank Lines, Inc.	Nitro, WV	RCRA generator	Alluvium-Kanawha Soils	Ground-water discharge to Kanawha River	Phenol; chromium	Tank truck cleaning facility
17. Chem Formulators, Inc.	Nitro, WV	CERCLA	Alluvium-Kanawha Soils	Ground-water discharge to Kanawha River	Methoxychlor; chloroneb; phenol; maleic hydrazine; anisole	Small pesticide manufacturer w/ on-site contamination
18. Mason & Dixon Tank Lines, Inc.	St. Albans, WV	RCRA generator	Alluvium-Kanawha Soils	Ground-water discharge to Kanawha River	Phenol; chromium	Tank truck cleaning facility
19. Nitro Sanitation	Nitro, WV	CERCLA	Alluvium-Kanawha Soils	Ground-water discharge to Kanawha River	Arsenic; phenol; heptone	Municipal landfill that accepted hazardous waste
20. Gary Harris Dump	Lanham, WV	CERCLA	Shale/Sandstone-GUV Soils	Consumption of contaminated ground water-private wells	1,1,1-TCA; benzene; ethylbenzene; dinitrotoluene; carbon tetrachloride; chloroform; toluene; benzo(a)pyrene; PAHs	Uncontrolled disposal area (several hundred drums); high potential for exposure via ground water
21. Western Kanawha County Landfill	Crosslanes, WV	CERCLA	Shale/Sandstone-GUV Soils	Consumption of contaminated ground water-private wells	Heavy metals; phenol; acrolein; toluene; MEK wastes; generally not well characterized	Very high exposure potential; Fike and other companies allegedly disposed of chemical waste here
22. Poca Stripmine Drum Site	Poca, WV	CERCLA	Shale/Sandstone-GUV Soils	Consumption of contaminated ground water-private wells	1,1,1-TCA; cyanide; zinc; chromium; nickel; toluene; methylene chloride; isopropanol	"Midnight dumping"; emergency removal of most of the wastes has been carried out
23. Vimasco Corp.	Nitro, WV	RCRA generator	Alluvium-Kanawha Soils	Ground-water discharge to Kanawha River	Arsenic; cadmium; lead; mercury; zinc; PCBs; toxaphene; xylenol; phenol; MIBK; 1,1,1-TCA; carbon tetrachloride; ethylbenzene; benzene; toluene	Unlined burial pit and three waste ponds on-site

TABLE 1 (Continued)

KANAWHA VALLEY TOXICS SCREENING STUDY/HAZARDOUS WASTE SITES

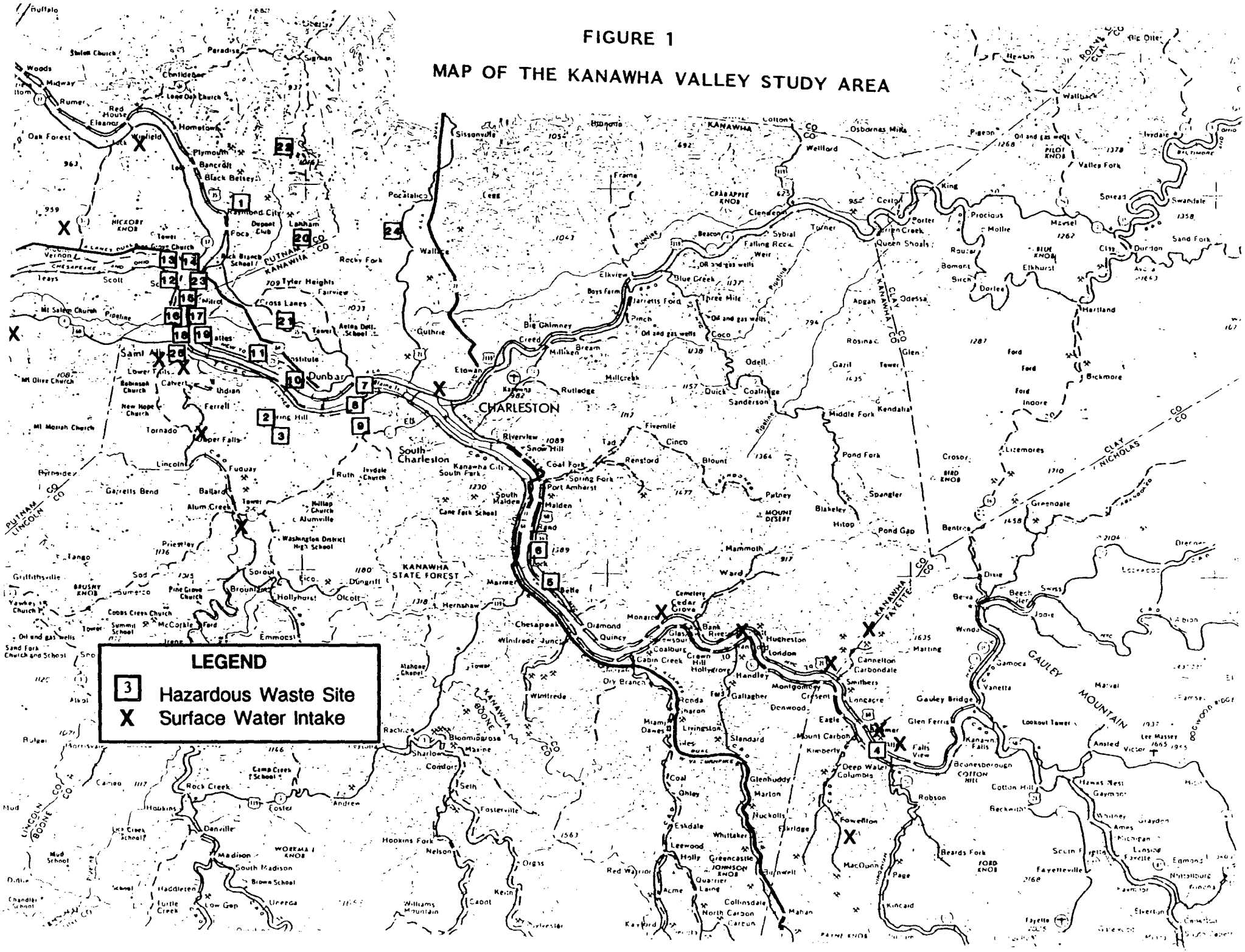
RCRA AND POTENTIAL CERCLA SITES WITH SUFFICIENT INFORMATION FOR PRELIMINARY RISK ANALYSIS

SITE NAME	LOCATION	RCRA/CERCLA	HYDROGEOLOGIC SETTING	POTENTIAL EXPOSURE PATHWAY	PREDOMINANT WASTES	COMMENTS
24. Monday (Mundy) Hollow Landfill	Poca, WV	CERCLA	Shale/Sandstone-GUV Soils	Consumption of contaminated ground water-private wells	Phenol; arsenic	84 ground-water users identified within 1/2 mile of facility; municipal landfill that accepted hazardous waste
25. Markay Chemical Co.	St. Albans, WV	RCRA generator	Alluvium-Kanawha Soils	Ground-water discharge to Coal River	Chlorobenzene; 1,2-dichloropropane; carbon tetrachloride; TCE; chloroform; methylene chloride	Facility operated for 8 years w/o RCRA notification (illegally); halogenated solvents stored in unlined impoundment

a/ These chemicals are used or produced at the facility, and have been suggested as indicator chemicals for ground-water monitoring at the facility. However, it is not known whether these chemicals have actually been disposed of on-site.

FIGURE 1

MAP OF THE KANAWHA VALLEY STUDY AREA



In evaluating potential human exposures via ground water, we considered not only the direct pathway of release, subsurface transport, and consumption of contaminated ground water, but also identified the ground water to surface water migration pathway that exists at many of the sites close to the Kanawha River.

Finally, we attempted to identify the predominant waste types and amounts disposed of at each site. As is frequently the case, this waste characterization proved to be extremely difficult for several reasons: (1) many RCRA facilities perform only the minimum characterization required by law for much of their waste (e.g., determinations of whether the waste is ignitable, EP toxic, corrosive, or reactive); (2) many CERCLA sites have not been extensively sampled; and (3) based on quality assurance (QA) reports, many of the sample analyses from CERCLA sites were suspect. Thus, waste information on the facilities was either missing or substandard in many cases.

III. SITE CLASSIFICATION

We classified the 25 hazardous waste study sites on the basis of hydrogeological setting, facility type, and predominant waste types. These three parameters are important in determining the likelihood of a facility failure, the transport of the pollutant through the ground water, and the potential health effects posed by the transported pollutant if consumed. The classification of study sites according to these parameters provides a qualitative assessment of the potential for ground-water contamination. These general classifications are described below.

Hydrogeologic Setting

Our evaluation of the hydrogeology in the Kanawha Valley and the development of hydrogeologic scenarios are described in detail in Appendix A. Thus, the discussion here focuses mainly on the hydrogeologic settings as they relate to the sites listed in Table 1.

Table 2 presents brief descriptions of the three general hydrogeologic settings we developed for our analysis, and lists the hazardous waste study sites that are found in each of the three settings. As illustrated, all of the RCRA facilities except one and several of the most notable potential CERCLA sites are located in the Kanawha Soil/Alluvium hydrogeologic setting. There is probably no direct ground-water exposure pathway for chemicals released from these sites because: (1) the ground water discharges directly to surface water within a short distance, and (2) people in the Kanawha Valley proper generally do not use ground water for potable water, but rather are served by public water supply systems, which obtain their water from tributaries to the Kanawha River. However, these facilities may be a source of surface water contamination via the pathway of contaminated ground-water discharge.

TABLE 2

KANAWHA VALLEY TOXICS SCREENING STUDY/HAZARDOUS WASTE SITES

HYDROGEOLOGIC SETTING CATEGORIES
IN THE KANAWHA VALLEY

HYDROGEOLOGIC SETTING	DESCRIPTION a/	HAZARDOUS WASTE SITES IN SETTING
1. Clymer-Dekalb-Gilpin Soil/Sandstone-Shale Bedrock	Well drained, acid soils. Bedrock is predominantly sandstone with interbedded shales (Pottsville and Allegheny Groups). Ground-water flow is unconfined b / through fracture systems. Characteristic of SE mountainous portion of study area.	Union Carbide Technical Center
2. Gilpin-Upshur-Vandalia Soil/Shale-Sandstone Bedrock	Moderately to slowly permeable silt loam and silty clay loam soils. Bedrock is predominantly shale with interbedded sandstone (Conemaugh, Monongehala, and Groups). Unconfined flow through fracture systems at 300-400 meters/yr. Characteristic of NW mountainous portion of study area.	Heizer Creek Dump; Gary Harris Dump; Western Kanawha County Landfill; Poca Stripmine Dump Site; Monday (Mundy) Hollow Landfill; South Charleston Municipal Landfill; Smith Creek Dump
3. Kanawha Soil/ Alluvium	Moderately drained to well-drained lime influenced soils. Alluvium consists of sand, silt, gravel, and clay; upper layer of sandy clay with underlying sand and gravel; many lenses and small perched water tables. Total thickness of alluvium is about 60 feet with water table at about 15 feet below surface. Ground-water flow is unconfined through porous media.	Elkem Metals; Occidental; E.I. DuPont; Union Carbide South Charleston; FMC South Charleston; Rhone-Poulenc; Chemical Leaman Tank Lines; Big Scary Creek Pond; FMC Nitro; Monsanto Company; Artel; Coastal Tank Lines; Chem Formulators; Mason and Dixon Tank Lines; Nitro Sanitation; Vimasco; Markay Chemical Co.

a/ For more details see Appendix A, "Hydrogeologic Characterization of the Kanawha Valley." Information was gathered from USGS and state reports, RCRA Part B Applications, CERCLA studies, and academic studies (see reference list given in Appendix C).

b/ An unconfined aquifer is one whose upper surface is a water table free to fluctuate under atmospheric pressure.

Facilities that are located in the mountainous areas outside the main river valley are generally potential CERCLA sites. Of these, most are located in the Gilpin-Upshur-Vandalia Soil/Shale-Sandstone hydrogeologic setting, which covers the northwest portion of the study area. Only one study site (Union Carbide Technical Center) is located in the Clymer-Dekalb-Gilpin Soil/Sandstone-Shale hydrogeologic setting, although two other sites (Diamond Shamrock, E.I. Dupont) are located where the alluvium is not well developed over the Sandstone-Shale hydrogeologic setting. People living outside the main river valley could use ground water for their domestic supply; thus, a direct exposure pathway may exist at some of the facilities in these areas.[1]

Facility Type

An obvious division can be made for the hazardous waste sites in the Kanawha Valley by separating them into RCRA and potential CERCLA sites. However, as noted earlier, some sites are active or inactive RCRA facilities that are also under CERCLA investigation. Also, there is a significant amount of diversity among specific sites in each of these two categories. Thus, we believed that further facility classification was necessary.

We classified RCRA facilities into two major types: (1) permitted treatment, storage, and disposal facilities, and (2) generators who do not treat or dispose wastes and do not store wastes more than 90 days. For potential CERCLA sites, we identified three major categories: municipal landfills that accepted hazardous industrial wastes, inactive chemical plants, and uncontrolled disposal areas[2] (e.g., "midnight dumping" areas, open drum dumps).

These five categories, the typical kinds of release sources for each category, and the sites that are included in each category, are presented in Table 3. It should be noted that these categories were developed to represent the major types of hazardous waste sites in the Kanawha Valley for which there was available information. The fact that one category contains fewer sites does not necessarily indicate that it is less significant; rather, it reflects our lack of information on the majority of hazardous waste sites in the Kanawha Valley. For example, we have information indicating that the Dutch Hollow Drum Site near Poca is an open dump containing 600 to 1000 drums, but have no information that will allow us to characterize the wastes at this

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[1] In our analysis, we were able to identify ground-water users in proximity to several hazardous waste sites in the shale/sandstone and sandstone/shale hydrogeologic settings. Based on Superfund preliminary assessment reports, there is evidence of private well contamination in two cases (Heizer Creek Dump and South Charleston Municipal Landfill). However, an in-depth evaluation of potentially exposed populations and current ground-water contamination was beyond the scope of this analysis.

[2] This category is by necessity somewhat of a catch-all group but mainly addresses sites at which there appears to have been no effort whatsoever to prevent release or migration of wastes.

TABLE 3

KANAWHA VALLEY TOXICS SCREENING STUDY/ HAZARDOUS WASTE SITES

GENERAL FACILITY TYPES FOR HAZARDOUS WASTE SITES IN THE KANAWHA VALLEY

FACILITY TYPE	TYPES OF RELEASE SOURCES	SITES
RCRA treatment, storage, and disposal facilities	Regulated land disposal units, such as landfills and surface impoundments (lined or unlined); also, some old landfills and dumping areas (solid waste management units, or SWMUs)	Monsanto Company E.I. Dupont Union Carbide South Charleston FMC South Charleston Union Carbide Technical Center Rhone-Poulenc FMC Nitro Artel
RCRA generators	Unregulated release sources, such as old landfills and dumping areas	Markay Chemical Elkem Metals Occidental Coastal Tank Lines Chemical Leaman Tank Lines Mason and Dixon Tank Lines Vimasco Corp
Potential CERCLA sites -- municipal landfills that accepted hazardous industrial wastes	Municipal landfills (unlined)	Heizer Creek Dump South Charleston Municipal Landfill Smith Creek Dump Nitro Sanitation Western Kanawha County Landfill Monday (Mundy) Hollow Landfill
Potential CERCLA sites -- uncontrolled disposal	Uncontrolled drum dumping areas	Poca Stripmine Drum Site Gary Harris Dump
Potential CERCLA sites -- inactive chemical plants	Old landfills and dumping areas	Chem Formulators Big Scary Creek Pond

site. As a result, this potentially important site was excluded from further consideration in this study. Thus, given the available information, it would be difficult to estimate the relative importance of each of the five categories.

Waste Type

As expected, the hazardous waste sites we characterized reflect the diversity of chemicals disposed in the Kanawha Valley. A myriad of organic and inorganic chemicals are produced, used, and disposed of in the study area, resulting in a wide variety of chemicals being identified at both RCRA and potential CERCLA sites. A list of the chemicals identified at hazardous waste sites in the study area is presented in Table 4.

After identifying the chemicals present, out next step was to group sites into categories of similar waste types and to designate a set of model constituents (i.e., representative chemical constituents to be modeled) for each waste type. Given the screening nature of the analysis, we minimized the number of waste groupings. Thus, the four groups presented in Table 5 are probably the minimum number into which the 25 sites could be divided while accounting for real differences in waste characteristics that have been observed at the sites.

To group sites into general waste categories, two methods were used. These were: (1) grouping by professional judgment, based on the presence of one or two chemicals at high concentrations; and (2) grouping through the use of a waste/site matrix, in which all of the 25 sample sites included in the analysis were listed along one axis and all chemicals identified at these sites were listed along the other axis. Sites with similar distributions of chemicals were then placed in the same group.

Although the second method would appear to be more objective, both classification approaches necessarily relied heavily on professional judgment, due to the extremely diverse nature of the wastes found at the sites included in the analysis. Thus, both methods used several predominant chemicals as a basis for grouping sites into general categories, with one or more surrogates selected to represent other chemicals found at sites in each group.

To identify model constituents for each group of Kanawha Valley sites, we used the following criteria (roughly in descending order of importance):

- Number of sites in a group in which the chemical was identified (i.e., frequency of occurrence);
- Concentration of the chemical at the sites at which it was identified;

TABLE 4

KANAWHA VALLEY TOXICS SCREENING STUDY/ HAZARDOUS WASTE SITES

PREDOMINANT WASTE TYPES AND CHEMICALS FOUND AT HAZARDOUS WASTE SITES IN THE KANAWHA VALLEY

WASTE TYPE	CHEMICALS
Halogenated aliphatic solvents	Methylene chloride 1,1,1-Trichloroethane Trichloroethylene Carbon tetrachloride Chloroform Tetrachloroethylene 1,2-Dichloroethane 1,2-Dichloropropane
Industrial intermediates, solvents and products	Phenol Cresol Benzene Acrylonitrile Toluene Methyl ethyl ketone Chlorobenzenes Chlorophenols Vinyl chloride Methacrylate Carbon disulfide Acrolein Ethylbenzene Dinitrotoluene Methylene dianiline
Inorganics	Arsenic Mercury Lead Cadmium Chromium Zinc Cyanide Nickel

TABLE 4 (continued)

KANAWHA VALLEY TOXICS SCREENING STUDY/
HAZARDOUS WASTE SITES

PREDOMINANT WASTE TYPES AND CHEMICALS
FOUND AT HAZARDOUS WASTE SITES
IN THE KANAWHA VALLEY

WASTE TYPE	CHEMICALS
Chlorinated pesticides	Chlordane Methoxychlor DDE DDD Dieldrin Aldrin Heptachlor 2,4,5-T Lindane
Highly persistent and toxic by-products	PCBs Dioxin Hexachloroethane Hexachlorobenzene
Industrial carcinogens	Bis(2-chloroethoxy)methane(BCEXM) Bis(2-chloroethyl)ether(BCEE) Bis(2-chloroisopropyl)ether(BCIE) N-Nitrosodiphenylamine Chlorinated azo compounds
Polynuclear aromatic compounds	Naphthalene Benzo(a)pyrene Fluoranthene Chrysene Acenaphthene
Carbamate pesticides	Aldicarb Carbaryl

TABLE 5

KANAWHA VALLEY TOXICS SCREENING STUDY/ HAZARDOUS WASTE SITES

GENERAL WASTE CATEGORIES FOR HAZARDOUS WASTE SITES IN THE KANAWHA VALLEY

WASTE CATEGORY NUMBER	MODEL CONSTITUENTS	HAZARDOUS WASTE SITES
1	Carbon tetrachloride Mercury 2,4-Dinitrotoluene	Occidental; Artel; Union Carbide South Charleston; Rhone-Poulenc; Union Carbide Technical Center; Gary Harris Dump; Markay Chemical Company; Vimasco Corp; FMC-South Charleston
2	Methylene chloride Cyanide Phenol Chlordane	Chem Formulators; Heizer Creek Dump; Smith Creek Dump; South Charleston Municipal Landfill; Poca Stripmine Drum Site; Western Kanawha County Landfill; Monsanto
3	Phenol Arsenic	Chemical Leaman Tank Lines; Mason and Dixon Lines; Coastal Tank Lines; Nitro Sanitation; FMC Nitro; Mundy Hollow Landfill; E.I. DuPont
4	Chromium Zinc	Elkem Metals; Big Scary Creek Pond

- Toxicity of the chemical, with potential carcinogens weighted more heavily than noncarcinogens[3] (carcinogens were scored according to potency values developed by EPA's Carcinogen Assessment Group); and

- Mobility and persistence in ground-water systems.

Waste categories and model constituents are described below. For identification of which sites fall into each general category, refer to Table 5.

Category 1: In this category, carbon tetrachloride, a highly toxic and relatively mobile chlorinated solvent and chemical intermediate, was used as the predominant chemical for grouping hazardous waste sites. Carbon tetrachloride was present at all of the sites in this group at significant concentrations; however, the other two model constituents in this scenario, mercury and 2,4-dinitrotoluene, should be considered surrogates for a variety of chemicals found at these sites. Mercury was selected based on its appearance at several of the sites and its high toxicity, and was chosen as a surrogate for other toxic inorganic contaminants found in lower concentrations or less frequently at sites in this group (e.g., cadmium, chromium, cyanide). 2,4-Dinitrotoluene was identified at two of the sites in this group, and is a potent carcinogen that is relatively mobile in ground water. Thus, 2,4-dinitrotoluene was considered to be a surrogate for other relatively mobile carcinogens, such as acrylonitrile, benzene, and N-nitrosodiphenylamine, that were found at some sites in this group.

Category 2: Sites included in this category were extremely diverse, but were grouped mainly on the basis of three classes of contaminants: (1) chlorinated solvents and intermediates (e.g., methylene chloride); (2) inorganics (e.g., cyanide); and (3) highly persistent chlorinated aromatic compounds (e.g., chlordane). These classes of chemicals were identified at essentially all of the sites in this group. The most common chlorinated solvents and intermediates identified were 1,1,1-trichloroethane, methylene chloride, and di-, tri-, and tetrachlorobenzenes. Inorganics included cyanide, arsenic, chromium, lead, and nickel, while highly persistent chlorinated organics included PCBs, dioxin, chlordane, heptachlor, methoxychlor, lindane, and DDE. In addition to these compounds, phenols and cresols were identified at many of the sites. Based on the criteria cited earlier (i.e., frequency of occurrence, concentration, toxicity, mobility, and persistence), we selected methylene chloride, cyanide, phenol, and chlordane to represent the wastes found at sites in this group.

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[3] Potential carcinogens were weighted more heavily in the selection of model constituents because: (1) in assessing hazardous waste mixtures that contain potential carcinogens, cancer risks usually reach levels of concern before noncancer risks do; and (2) the primary focus of the overall study, including the air, surface water, and drinking water analyses, was on cancer risk.

Category 3: Sites in this category were generally not well-characterized, but the predominant chemicals identified were industrial chemicals such as phenol and heavy metals such as arsenic, chromium, lead, and zinc. Phenol and arsenic were selected to represent this group based on frequency of occurrence, concentration, toxicity, mobility, and persistence.

Category 4: Information on two sites indicated that the predominant hazardous wastes generated at these facilities were heavy metal sludges. Zinc and chromium were identified as the only hazardous constituents of these sludges and therefore were selected to represent this waste category.

It is important to note that certain sites were grouped mainly on the appearance of one or two chemicals (e.g., carbon tetrachloride was the predominant chemical used to group sites into waste category 1), and in some cases there are fairly wide variations in other chemicals identified among the different sites in a particular group. This problem is inevitable in a broad categorization of this type; an attempt was made to minimize the problem by choosing other model constituents that, although they were not always present at a large number of sites, were reasonably representative of the toxicity and mobility of other compounds seen at the sites (i.e., surrogate compounds). For example, 2,4-dinitrotoluene was identified at only two of the nine sites in category 1; however, it is a potent carcinogen that is relatively mobile in subsurface environments, and thus may provide a reasonable conservative representation for some other chemicals seen at other category 1 sites (e.g., acrylonitrile, benzene, vinyl chloride, and N-nitrosodiphenylamine) with respect to mobility and toxicity. Chemicals selected as model constituents for each waste category were chosen primarily based on the degree to which they represented the wastes found at sites in the Kanawha Valley. However, in selecting a surrogate compound to represent a group of chemicals, the most toxic/mobile chemical was chosen.

Also, it should be noted that we did not select many chlorinated aromatic compounds such as PCBs, dioxins, hexachlorobenzene, or dieldrin as model constituents. These types of compounds, which tend to bioconcentrate in aquatic life, have been detected in fish from the Kanawha River. However, these compounds were not identified at high levels at most of the hazardous waste sites for which we had information. Moreover, these compounds, due to their hydrophobic nature, tend to sorb strongly to earth materials and move very slowly through ground water.

IV. QUANTITATIVE MODELING

Two steps were necessary for the quantitative modeling: development of model scenarios and the actual modeling of potential exposures and risks. We discuss these two steps, along with the modeling results, in this section.

Development of Model Scenarios

Although there are a very large number of hazardous waste sites in the Kanawha Valley, sufficient data to perform even a preliminary exposure/risk assessment are available only for a few sites. Most sites have been studied only at the screening level, and, at best, the information available on a particular site consists of: (1) a site report based on a one-day field trip reconnaissance; (2) analytical data on a few samples (usually ten or fewer) collected from various media at the site (ground water, soil, drum, and surface water samples); and (3) a crude hydrogeological characterization and identification of potentially exposed populations for the site (available for only a few sites). Thus, the information available is not adequate to support a site-specific risk analysis, and an extensive data gathering effort would be needed to perform an in-depth analysis of the problem. Therefore, the development and analysis of model scenarios that are representative of different groups of hazardous waste sites was considered the best available method for gaining some sense of which types of site situations have higher risks than others and also to begin to get some sense of the overall environmental problem represented by these sites. Development of the model scenarios is outlined below.

It is important to emphasize that this quantitative modeling effort is a screening-level analysis that is based on limited data. Thus, the study has significant limitations, and definite conclusions with respect to the problem of ground-water contamination by hazardous waste sites cannot be drawn from the results of the analysis. The analysis is, however, considered useful for comparing relative risks between model scenarios, and for providing a quantitative overview of the problem for policy screening purposes.

Review of Preliminary Site Characterization and Classification: As discussed in Section III, similarities exist among the different sites in the Kanawha Valley that allow them to be grouped into categories based on source type, waste characteristics, and hydrogeologic setting. A waste/hydrogeology matrix combining the information in Tables 2 and 5 for the 25 hazardous waste sites considered in this analysis is presented in Table 6, and a grouping of sites according to facility type is presented in Table 3.

The grouping of hazardous waste sites into these categories is based on similarities in source type, waste characteristics, and hydrogeologic setting. For example, two municipal landfill sites that are located in the shale/sandstone hydrogeologic setting and contain carbon tetrachloride wastes would be grouped into the same matrix category. However, it is important to note that these groupings are based only on very general information. This is particularly true for waste characteristics, where in most cases sites have been grouped on the basis of information from only a few samples. More detailed analyses may show that two sites that were grouped together based on screening-level data are in fact quite dissimilar with respect to waste characteristics (e.g., one site may contain carbon tetrachloride wastes, while a second site that was thought to contain similar wastes may actually contain

TABLE 6

KANAWHA VALLEY TOXICS SCREENING STUDY/HAZARDOUS WASTE SITES

HYDROGEOLOGY/WASTE MATRIX
FOR HAZARDOUS WASTE SITES IN
THE KANAWHA VALLEY

SITE CLASSIFICATION INTENDED FOR POLICY DEVELOPMENT PURPOSES

HYDROGEOLOGIC SETTING	PREDOMINANT WASTE CATEOGRY a/			
	1	2	3	4
Sandstone/Shale	Union Carbide Technical Center	--	--	--
Shale/Sandstone	Gary Harris Dump	Heizer Creek Dump; Poca Stripmine Drum Site; Western Kanawha County Landfill; Smith Creek Dump; South Charleston Municipal Landfill	Mundy Hollow Landfill	--
Alluvium	Occidental; Artel; Union Carbide South Charleston; Rhone-Poulenc; Vimasco Corp.; Markay Chemical Co.; FMC South Charleston	Chem Formulators; Monsanto Company	Chemical Leaman Tank Lines; Coastal Tank Lines; Mason and Dixon Tank Lines; Nitro Sanitation; FMC Nitro; E.I. DuPont	Elkem Metals; Big Scary Creek Pond

a/ Model constituents for the four waste categories are:

1 -- carbon tetrachloride, 2,4-dinitrotoluene, mercury
2 -- methylene chloride, phenol, chlordane, cyanide
3 -- phenol, arsenic
4 -- chromium, zinc

predominantly heavy metal sludges). Thus, the grouping of sites into categories is based largely on screening-level information, and more detailed investigations may reveal substantial differences among sites in the same category.

Despite these problems, the grouping of sites into source/waste/hydrogeology categories was considered a useful method for evaluating the potential effects of a large number of sites for which limited data are available. The initial grouping served as the basis for development of model scenarios used in the analysis.

Source/Waste/Hydrogeology Scenarios: Based on the characterization and initial classification of hazardous waste sites outlined above, we developed a group of seven model scenarios that are intended to be representative of different groups of hazardous waste sites in the Kanawha Valley. These scenarios were developed from a compilation of actual site data on source type and size, waste constituents and concentrations, hydrogeologic parameters, and potentially exposed populations. However, the following points should be noted:

- Each scenario represents, in a broad sense, a type of site that we believe exists in the Kanawha Valley, but does not correspond directly to any specific site or group of sites included in the study. Therefore, risks estimated for a scenario are considered to be representative of similar hazardous waste sites in the Kanawha Valley, but should not be associated with any specific individual site in the area.

- The development of all scenarios, while based on available data from Kanawha Valley waste sites, required the use of professional judgment to supplement this information. Thus, parameters that may have a major effect on risk and environmental impact (e.g., waste constituent concentrations) often were estimated using a combination of available data and professional judgment. The significant data limitations introduce considerable uncertainty into the results of the analysis.

In the following sections, we outline our methodology for developing scenarios and for evaluating (semi-quantitatively) the effects of using estimated values for key parameters in our analysis.

Estimation of Parameters for Model Scenarios: In order to use a deterministic mathematical model to estimate risks and other environmental impacts (e.g., the contribution of contaminated ground-water discharge to surface water contamination in the Kanawha River), it was necessary to develop scenarios in which individual source, waste, and hydrogeologic parameters were explicitly identified. As noted above, data from actual waste sites were used

in identifying these parameters; however, actual arithmetic or geometric mean values taken from a group of sites for a particular parameter (e.g., waste constituent concentrations) were not used. Rather, the range of values observed for a given parameter at a group of sites was used to provide an estimate of typical values. The final value for the parameter was based on this observed range and professional judgment. The following example should serve to illustrate this procedure:

> For waste category 1, carbon tetrachloride has been identified as a waste constituent. A range of concentrations has been identified in leachate, soil, drums, and surface streams at sites that serve as the basis for this scenario; however, carbon tetrachloride has been identified only at five of the nine sites in this category.[4] In addition, quality assurance/quality control checks on analytical data show that some of the positive values are questionable. Thus, rather than use a mathematical average of incomplete data of varying quality from various media, we estimated a value for carbon tetrachloride concentration based on the reported range. Our estimated values were close to the observed values in all cases. Site concentration data on model constituents selected for the Kanawha Valley Hazardous Waste Site Study are summarized in Table 7.

This procedure was used not only for identification of waste constituent concentrations, but for identification of other scenario parameters as well (e.g., exposure distance, source size). Thus, parameter values for scenarios do not represent any individual site, but are representative of what one would expect to observe at sites in the Kanawha Valley (i.e., values are not taken from national averages or from sites that are not within the study area).

Parameters for the seven Kanawha Valley source/waste/hydrogeologic setting scenarios are summarized in Table 8. Waste characteristics and hydrogeologic settings are presented in Tables 9 and 10, respectively.[5] As indicated, two sets of values were developed for each scenario, with scenarios designated as "A" representing our best-estimate values for parameters and "B" scenarios representing our conservative values for parameters. The development of conservative and best-estimate scenarios is discussed in the following section.

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[4] As noted in Section III, surrogate compounds were used in some cases to represent groups of similar chemicals; thus, all sites within a particular waste category do not necessarily contain all model constituents in that category.

[5] It should be noted that although our hydrogeologic characterization indicated three different settings, we had information only on sites that were located in the alluvium and shale/sandstone settings. Thus, we did not develop model parameters for the sandstone/shale setting. Based on DRASTIC scores (see Appendix A), it is likely that model parameters for this setting would be very similar to those for the shale/sandstone setting.

TABLE 7

KANAWHA VALLEY TOXICS SCREENING STUDY/HAZARDOUS WASTE SITES

SITE CONCENTRATION DATA FOR CHEMICALS
SELECTED AS MODEL CONSTITUENTS

WASTE CATEGORY a/	MODEL CONSTITUENTS	FREQUENCY OF DETECTION b/	SAMPLE MEDIUM	CONCENTRATION RANGE (mg/l or mg/kg) c/
1	Carbon tetrachloride	5/9	Leachate	0.24
			Soil	2.42
			Drum	NQ d/
			Stream	0.99
	2,4-Dinitrotoluene	2/9	Soil	22.50
			Drum	9.00
	Mercury	6/9	Ground water	0.13
			Soil	1.8-19.0
			Drum	0.2-13.4
			Stream	0.075
2	Methylene chloride	3/7	Ground water	4.57
			Leachate	12.0
			Drum	10,000 - 50,000
	Phenol	2/7	Soil	NQ-22.6
	Chlordane	1/7	Ground water	0.013
	Cyanide	4/7	Soil	NQ-2,800
			Drum	41.8-100,000
3	Phenol	6/7	NA e/	NQ
	Arsenic	5/7	NA	NQ

a/ See Section II for a description of the development of waste categories.

b/ Number of sites in waste category at which contaminant was detected divided by the total number of sites in that waste category.

c/ For sample media in which constituent was detected only once, a single value is reported.

d/ NQ = not quantified. Constituent was detected at the site, but concentration was not reported.

e/ Information on sites in waste category 3 indicated that phenol and arsenic were suspected of being present at nearly all the sites in the group; however, no quantitative sampling information was available for any of these sites.

TABLE 8

KANAWHA VALLEY TOXICS SCREENING STUDY/HAZARDOUS WASTE SITES

SUMMARY OF PARAMETERS FOR
SOURCE/WASTE/HYDROGEOLOGIC SETTING MODEL SCENARIOS

MODEL SCENARIO	SOURCE TYPE a/	OPERATING/ PCC PERIOD b/	WASTE CATEGORY c/	WASTE AMOUNT d/	GROUND-WATER SETTING e/	SIZE (meters)	EXPOSURE DISTANCE f/	PRIMARY EXPOSURE PATHWAY
1A	"Subtitle C" Lined landfill	20 yrs./30 yrs.	1--Average (solid)	22,500 metric tons/ yr	3	300 x 300	200m	Ground-water discharge to surface water
1B	"Subtitle C" Lined landfill (conservative)	20 yrs./30 yrs.	1--Conservative (solid)	50,625 metric tons/ yr	3	450 x 450	60m	Ground-water discharge to surface water
2A	Unlined surface impoundment	20 yrs./0 yrs.	1--Average (liquid)	8000 cubic meters	3	40 x 40	200m	Ground-water discharge to surface water
2B	Unlined surface impoundment (conservative)	20 yrs./0 yrs.	1--Conservative (liquid)	50,000 cubic meters	3	100 x 100	60m	Ground-water discharge to surface water
3A	"Subtitle D" Unlined landfill	20 yrs./0 yrs.	2--Average (leachate)	N/A	3	50 x 50	200m	Ground-water discharge to surface water
3B	"Subtitle D" Unlined landfill (conservative)	20 yrs./0 yrs.	2--Conservative (leachate)	N/A	3	100 x 100	60m	Ground-water discharge to surface water
4A	"Subtitle D" Unlined landfill	20 yrs./0 yrs.	3--Average (leachate)	N/A	3	50 x 50	200m	Ground-water discharge to surface water
4B	"Subtitle D" Unlined landfill (conservative)	20 yrs./0 yrs.	3--Conservative (leachate)	N/A	3	100 x 100	60m	Ground-water discharge to surface water
5A	"Subtitle D" Unlined landfill	20 yrs./0 yrs.	1--Average (leachate)	N/A	2	150 x 150	400m	Consumption of contaminated water
5B	"Subtitle D" Unlined landfill (conservative)	20 yrs./0 yrs.	1--Conservative (leachate)	N/A	2	400 x 400	200m	Consumption of contaminated water

TABLE 8 (continued)

KANAWHA VALLEY TOXICS SCREENING STUDY/HAZARDOUS WASTE SITES

SUMMARY OF PARAMETERS FOR
SOURCE/WASTE/HYDROGEOLOGIC SETTING MODEL SCENARIOS

MODEL SCENARIO	SOURCE TYPE a/	OPERATING/ PCC PERIOD b/	WASTE CATEGORY c/	WASTE AMOUNT d/	GROUND-WATER SETTING e/	SIZE (meters)	EXPOSURE DISTANCE f/	PRIMARY EXPOSURE PATHWAY
6A	"Subtitle D" Unlined landfill	20 yrs./0 yrs.	2--Average (leachate)	N/A	2	150 x 150	400m	Consumption of contaminated water
6B	"Subtitle D" Unlined landfill (conservative)	20 yrs./0 yrs.	2--Conservative (leachate)	N/A	2	400 x 400	200m	Consumption of contaminated water
7A	"Subtitle D" Unlined landfill	20 yrs./0 yrs.	3--Average (leachate)	N/A	2	150 x 150	400m	Consumption of contaminated water
7B	"Subtitle D" Unlined landfill (conservative)	20 yrs./0 yrs.	3--Conservative (leachate)	N/A	2	400 x 400	200m	Consumption of contaminated water

a/ "Subtitle C" landfills are considered to be in compliance with current RCRA liner requirements for hazardous waste landfills. "Subtitle D" landfills are unlined.

b/ PCC = post-closure care.

c/ Solid = waste constituent concentration specified for solid material disposed at site; liquid = waste constituent concentration specified for liquid material disposed in surface impoundments; leachate = waste constituent concentration specified for infiltrating liquid (leachate) at base of landfill. See Table 9 for characteristics of waste categories 1, 2, and 3.

d/ Specified only for model RCRA facilities ("Subtitle C" landfills and surface impoundments).

e/ Setting 3 = alluvium; Setting 2 = shale/sandstone. Insufficient information on the few sites located in Setting 1 (sandstone/shale) was available to develop a generic scenario for this group. See Table 10 for details about ground-water settings.

f/ Distance from source of contamination to (1) Kanawha River (scenarios 1-4); or (2) drinking water well (scenarios 5-7).

TABLE 9

KANAWHA VALLEY TOXICS SCREENING STUDY/ HAZARDOUS WASTE SITES

WASTE CHARACTERISTICS FOR MODEL SCENARIOS

WASTE a/ CATEGORY	MODEL CONSTITUENTS	CONCENTRATION (BEST-ESTIMATE SCENARIO (A)) (ppm) b/	CONCENTRATION (CONSERVATIVE SCENARIO (B))(ppm) b/
1 (solid)	Carbon tetrachloride	10	1000
	2,4-Dinitrotoluene	1	100
	Mercury	1	100
1 (liquid)	Carbon tetrachloride	300	3000
	2,4-Dinitrotoluene	0.05	5
	Mercury	0.1	10
1 (leachate)	Carbon tetrachloride	0.25	25
	2,4-Dinitrotoluene	0.01	1
	Mercury	0.05	5
2 (leachate)	Methylene chloride	5	500
	Phenol	10	1000
	Chlordane	0.01	1
	Cyanide	2	200
3 (leachate)	Phenol	5	500
	Arsenic	0.05	5

a/ See Section III for a description of the development of waste categories.

b/ For solid and liquid wastes, ppm refers to the constituent mass fraction in the waste stream itself. For these wastes, the Liner Location Model calculates the mass fraction in leachate as part of the modeling process. For leachate wastes, ppm refers to the constituent mass fraction in leachate at the bottom of the disposal unit.

TABLE 10

KANAWHA VALLEY TOXICS SCREENING STUDY/HAZARDOUS WASTE SITES

GROUND-WATER CHARACTERISTICS
FOR HYDROGEOLOGIC SETTINGS a/

GROUND-WATER SETTING 2 - SHALE/SANDSTONE

	Bulk Density (p) b/	Porosity (n) c/	Fraction Organic Carbon Content (Foc) b/	Hydraulic Conductivity c/ (m/sec)	Depth to Ground Water (m) c/	Horizontal Ground-Water Velocity c/ (m/year)
Unsaturated zone	2.0	0.25	0.02	2.5×10^{-4}	1	N/A
Saturated zone	2.0	0.25	0.005	2.5×10^{-4}	N/A	100

GROUND-WATER SETTING 3 - ALLUVIUM

	Bulk Density (p) b/	Porosity (n) b/	Fraction Organic Carbon Content (Foc) b/	Hydraulic Conductivity d/ (m/sec)	Depth to Ground Water (m) d/	Horizontal Ground-Water Velocity d/ (m/year)
Unsaturated zone	2.0	0.25	0.02	3×10^{-4}	5	N/A
Saturated zone	2.0	0.25	0.005	3×10^{-4}	N/A	10

a/ Parameters not given for setting 1 (sandstone/shale) because it was not included in any of the seven model scenarios.

b/ Identified from Soil Conservation Service Reports, USGS and state Geological Survey Reports, and literature estimates (Freeze and Cherry, 1980).

c/ Identified from "Hydrogeologic Investigations of Hazardous Waste Landfills Near South Charleston, WV (Ahmad and Hughart, 1981).

d/ Identified from "Fike Chemical Hydrogeologic Investigation" (prepared for EPA by JRB Associates, 1983).

Best-Estimate and Conservative Scenarios: As noted previously, professional judgment was used in some cases to identify values for scenario parameters. Some of these parameters have a major effect on the results of the analysis. Therefore, it was considered desirable to have some estimate of the degree of uncertainty that was introduced to the analysis by estimating these parameters based on professional judgment. However, formal sensitivity analysis techniques (e.g., Monte Carlo simulation) were not feasible, given the limited resources allocated for this portion of the overall study and the major data gaps that exist for some parameters.[6]

Therefore, to gain some estimate of the degree to which our identification of scenario parameters affected the outcome of the analysis, we developed best-estimate and conservative scenarios. Best-estimate scenarios were developed as outlined above, by using available data and professional judgment to construct scenarios that we believe to be most representative of a particular type of hazardous waste site found in the Kanawha Valley. In conservative scenarios, we used the same data and professional judgment to construct a conservative yet reasonable scenario for each type of hazardous waste site we identified. For example, in Scenario 1A we estimated carbon tetrachloride concentration in the waste stream to be 10 mg/kg, landfill size to be 300 x 300 meters, and exposure distance to be 200 meters. In Scenario 1B, we increased carbon tetrachloride concentration to 1000 mg/kg, increased landfill size to 450 x 450 meters, and decreased exposure distance to 60 meters. Thus, in Scenario 1B there is a considerably larger volume of material, a higher concentration of carbon tetrachloride, and a shorter distance between source and exposure point, resulting overall in a situation where one would expect higher risks than in Scenario 1A.

In using best-estimate and conservative scenarios, we have attempted to develop what we believe are representative situations for different types of hazardous waste sites in the Kanawha Valley. However, it is important to note once again that these scenarios are based on limited information and are intended to represent a type of sites rather than any individual site. Although our conservative settings are generally representative of the "highest risk" conditions for sites on which we had information, we do not believe that the conservative scenarios necessarily represent actual worst-case conditions that may be encountered at individual sites; therefore, we would caution against considering risk estimates for conservative scenarios to be upper-bound limits for hazardous waste sites in the Kanawha Valley. Conditions at an individual site could be worse than conditions specified in our conservative scenarios (e.g., much higher concentrations of chemicals, shorter exposure distances), resulting in risks significantly higher than those estimated in our analysis.

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[6] A sensitivity analysis using a technique such as Monte Carlo simulation is useful only if valid probability distributions can be constructed for each variable. The Kanawha Valley data were insufficient for this purpose in most cases.

In the next section, we present a description of the exposure/risk model used in the analysis and discuss the migration and exposure pathways considered in the study.

Exposure/Risk Modeling

In order to evaluate potential risks and environmental impacts for the Kanawha Valley model scenarios, it was necessary to: (1) identify the most important migration and exposure pathways for chemicals released from hazardous waste sites in the Valley; and (2) choose a mathematical model to quantitatively evaluate these pathways and estimate risks from chemical exposures. In the following sections, we describe each of these steps.

Identification of Migration and Exposure Pathways: Risk assessment scientists generally consider an exposure pathway to consist of four basic elements: (1) a source and mechanism of chemical release to the environment; (2) an environmental receiving and transport medium for the released chemical; (3) a point of potential exposure by humans or other receptors; and (4) a route of exposure to the contaminants. An exposure pathway is considered "complete" if all of these elements are present. A migration pathway consists only of a source and mechanism by which a chemical can be released and transported (e.g., miscible transport in ground water) and does not describe potential receptors or routes of exposure.

In this analysis, we have considered one exposure pathway and one migration pathway. The exposure pathway consists of the following:

- Release of chemicals to ground water from hazardous waste sites as a result of rainwater infiltration (or liquid infiltration from surface impoundments), either through failed liner systems or directly to soil in the case of unlined facilities;

- Transport through the unsaturated zone to the water table, followed by miscible transport in ground water to nearby drinking water wells; and

- Human exposure resulting from consumption of contaminated ground water from these wells.

Thus, this exposure pathway is considered "complete," and risks can be quantitatively estimated based on predicted exposures.

There are a number of other potential exposure pathways for contaminants escaping from hazardous waste sites in the Kanawha Valley, including: (1) direct contact (ingestion or dermal contact) with hazardous waste or contaminated soil; (2) inhalation of contaminated dust particles; (3) inhalation of volatile chemicals; (4) consumption or direct contact with surface water contaminated by run-off or ground-water discharge; and (5) consumption of biota that have been contaminated with chemicals. However, we have selected consumption of contaminated ground water as the exposure pathway of most concern in this analysis for the following reasons:

- A review of the data for sites in the Kanawha Valley has revealed that there are people living close to some sites who use ground water from private wells as their source of drinking water.[7] (This is therefore a complete pathway at some of the sites on which we have information.);

- Based on our experience with Superfund and RCRA sites, we have found that this pathway often results in the highest potential human exposures; and

- The quantitative evaluation of other pathways requires more site-specific information than is available for this study.

In addition to the ground-water exposure pathway, we considered one migration pathway in our analysis: miscible transport of contaminants in ground water and discharge of this contaminated ground water to surface water. This pathway was included because many of the chemical plants and RCRA facilities considered in our analysis are very close to the Kanawha River or its tributaries, and therefore it was considered important to evaluate the potential contribution of contaminated ground-water discharge to the contamination problem observed in the Kanawha River.

Use of the OSW Liner Location Model: To predict risks to ground-water users and estimate mass loadings of contaminants from ground water to surface water, we selected the Liner Location Risk and Cost Analysis Model (LLM) developed by EPA's Office of Solid Waste (OSW). The LLM is an integrated computer model that consists of release, transport, exposure, and risk component submodels. It was designed to analyze generic scenarios representative of conditions at land disposal facilities throughout the United States. Thus, the model is appropriate for screening-level, generic analyses such as the Kanawha Valley study, and it allows both the ground-water and ground-water/surface water pathways to be quantitatively evaluated.

The LLM includes the following component submodels:

- A stochastic failure model that predicts time to failure and leachate release volumes for various types of lined and unlined landfills and surface impoundments;

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[7] As stated previously, exposure through consumption of contaminated ground water is more likely for sites located outside the Kanawha Valley proper. People in the Kanawha Valley generally receive their drinking water from a municipal supply, while people living in mountainous areas outside the valley may obtain their water from private ground-water wells.

- A mass balance model that predicts contaminant concentrations in leachate released from a land disposal unit;

- Unsaturated zone and saturated zone contaminant transport models (modified versions of the McWhorter-Nelson Wetting Front and Prickett-Lonnquist Random Walk Models, respectively);

- An exposure model based on consumption of contaminated ground water; and

- Carcinogenic and noncarcinogenic risk models.

The model also provides the user with the ability to calculate contaminated ground-water discharge to surface water.

The LLM is described in detail in the Draft Liner Location Risk and Cost Analysis Model Report (EPA, 1985). As with any hazardous waste risk model, the LLM incorporates many assumptions. It is believed that the LLM generally overestimates risk levels (i.e., is a conservative model) because many of its assumptions, algorithms, and default values are conservative.

Results of the Screening-Level Analysis

This section presents our results for the Kanawha Valley model scenarios. As noted, we used the LLM to estimate carcinogenic risks and exposures to noncarcinogens resulting from consumption of contaminated ground water for scenarios in which it was determined that a potentially exposed population could exist (scenarios 5 through 7). We also estimated loadings and potential effects on the Coal River for scenarios 5 through 7. For scenarios 1 through 4, we evaluated the potential contribution of contaminated ground-water discharge to contamination in the Kanawha River. The results of these analyses are discussed below.

Exposure/Risk Analysis for Ground-Water Users: For scenarios 5 through 7 we estimated carcinogenic risks and evaluated noncarcinogenic exposures for an individual living relatively close to a hypothetical hazardous waste site represented by each scenario. For our modeling effort, we used the waste constituents and concentrations, ground-water settings, and exposure distances outlined for each scenario in Table 8. LLM assumptions regarding contaminant transport and fate were used, as were typical assumptions about human exposure and chemical absorption. These assumptions are as follows:

- Contaminant transport in ground water is controlled primarily by two factors: (1) the retardation factor of the particular contaminant of interest; and (2) the ground-water velocity of the aquifer in which transport is occurring. Aquifer properties that affect transport (e.g., velocity, fraction organic

carbon content) are considered to remain constant over the entire distance from source to exposure point, and a specific retardation factor is used for each contaminant (based on organic carbon-water partition coefficients for organics and distribution coefficients for metals). The aquifer is considered to be a homogeneous, isotropic porous medium. Contaminants are assumed to degrade at a rate specified by a single first-order constant that accounts for all chemical and biological degradation processes. Rate constants are identified from literature estimates; chemicals for which degradation data are unavailable are assumed to be infinitely persistent.

- Humans are assumed to drink 2 liters of water per day, and to have a body weight of 70 kilograms. One hundred percent absorption is assumed for all contaminants, and each individual is assumed to be exposed for 70 years.

These assumptions are necessary in a screening-level, non-site-specific analysis such the Kanawha Valley Toxics Screening Study, and they are consistent with assumptions made in similar EPA policy analyses. However, without detailed site-specific information the degree of uncertainty introduced to the analysis by the use of these assumptions cannot be fully evaluated.

The total modeling time used for the exposure/risk analysis was 100 years, which is long enough to allow ground-water transport of moderately mobile chemicals. Carcinogenic risks were estimated using the one-hit equation, EPA Carcinogen Assessment Group (CAG) potency values for all contaminants, and a 70-year moving average lifetime dose.[8] After a time profile of drinking water concentration (in mg/l) was estimated, we followed three steps to compute cancer risk: (1) convert each year's concentration to an equivalent human dose (in mg/kg-day) by multiplying by 2 liters per day intake and dividing by 70 kilograms average body weight; (2) average the annual doses over each 70-year period being analyzed to obtain average lifetime doses; and (3) calculate the risk using the one-hit equation (linear at low doses), the average lifetime dose, and the appropraite CAG upper-bound potency value. To evaluate noncarcinogenic risk, we compared yearly doses of noncarcinogenic chemicals to risk reference doses (RfDs) for these chemicals. The toxicity parameters used for specific chemicals included in the analysis are given in Tables 11 and 12.

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[8] For a more detailed discussion of exposure and risk modeling used in the analysis see the Draft Liner Location Risk and Cost Analysis Model Report developed by EPA's Office of Solid Waste (EPA, 1985).

TABLE 11

KANAWHA VALLEY TOXICS SCREENING STUDY/ HAZARDOUS WASTE SITES

UPPER-BOUND UNIT CANCER RISK VALUES FOR POLLUTANTS AT HAZARDOUS WASTE SITES

CHEMICAL	UNIT CANCER RISK VALUE a/ (mg/kg-day)-1	WEIGHT-OF-EVIDENCE RATING b/
Arsenic c/	15	A
Carbon tetrachloride	0.13	B2
Chlordane	1.3	B2
Methylene chloride	0.0075	B2
2,4-Dinitrotoluene	0.31	B2

a/ Potency values are upper-bound estimates derived by EPA's Carcinogen Assessment Group (CAG).

b/ Weight-of-Evidence rating derived by CAG, based on EPA's classification system: A = proven human carcinogen; B = probable human carcinogen (B1 indicates limited evidence from human studies, B2 indicates sufficient evidence from animal studies but inadequate evidence from human studies); C = possible human carcinogen; D = not classifiable, and E = no evidence of carcinogenicity.

c/ CAG has calculated a potency score for arsenic based on epidemiological evidence of skin cancer. However, some scientists believe that arsenic may not be carcinogenic and may be a necessary element at low levels.

TABLE 12

KANAWHA VALLEY TOXICS SCREENING STUDY/ HAZARDOUS WASTE SITES

VERIFIED REFERENCE DOSES FOR NONCARCINOGENIC EFFECTS OF CHEMICALS INCLUDED IN THE ANALYSIS

CHEMICAL	VERIFIED REFERENCE DOSE (mg/kg-day) a/
Arsenic	b/
Carbon tetrachloride	0.0007
Chlordane	0.00005
Chromium VI	0.005
Cyanide	0.02
Methylene chloride	0.06
2,4-Dinitrotoluene	b/
Mercury	0.002
Phenol	0.10
1,1,1-Trichloroethane	b/
Zinc	b/

a/ All reference doses derived for oral exposures, and verified by EPA's Reference Dose Workgroup in 1985 and 1986.

b/ Verified reference dose unavailable.

The results of our analysis are summarized in Tables 13 and 14 and Figure 2. Table 13 presents maximum and average risks for an individual living directly downgradient from a hypothetical waste site and using contaminated ground water as his/her source of drinking water. Risk estimates for all carcinogenic chemicals contributing to risk are presented for each scenario. As illustrated, risks are significantly greater for Scenarios 5A and 5B than for the other scenarios. This is a result of the wastes found in Scenario 5 (particularly 2,4-dinitrotoluene, a potent carcinogen);[9] all risk-influencing parameters other than waste type are constant across the three scenarios.

Figure 2 is a bar graph comparing peak individual carcinogenic risks for best-estimate and conservative scenarios. As indicated, risks vary from 2 to 4 orders of magnitude between best-estimate and conservative scenarios, with the greatest difference between scenarios 7A and 7B, and the least difference between scenarios 5A and 5B. These differences illustrate that the specified waste constituent concentrations for a particular scenario have a major effect on the outcome of the analysis. Thus, this comparison provides a qualitative estimate of the degree of uncertainty that is involved in this screening-level analysis. There are of course other elements of uncertainty.[10]

A summary of predicted exposures to noncarcinogenic chemicals for scenarios 5 through 7 is presented in Table 14. RfDs are not exceeded for any of the chemicals for which exposure is predicted to occur.

Analysis of Mass Loadings to Surface Water: For scenarios 1 through 4, we identified no potentially exposed populations for the ground-water exposure pathway. The sites that were used to develop these scenarios are located within the Kanawha River Valley proper, rather than in nearby mountainous areas, and residents in the river valley generally obtain their drinking water from municipal supplies.[11] However, many of these sites are located very close to the Kanawha River, and it is likely that contaminated ground water from these sites is discharging to the river. Also, the Kanawha Valley surface water contamination study performed by EPA indicated high levels of certain contaminants in fish tissues and sediments that could not be fully explained from NPDES effluent monitoring reports. Thus, it was hoped that an analysis of river contamination by ground-water discharge would provide some insight as to the source of these chemicals.

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[9] Carbon tetrachloride, also found in scenario 5, is a fairly potent carcinogen, but is assumed to degrade in ground water.

[10] As noted previously, conservative scenario risk estimates should not be regarded as upper-bound limits for risks from hazardous waste sites in the Kanawha Valley. Risks at specific individual sites could conceivably be greater than those estimated for a model scenario.

[11] Municipal supply for the Charleston-Institute-Nitro area is obtained mainly from the Elk and Coal Rivers.

TABLE 13

KANAWHA VALLEY TOXICS SCREENING STUDY/HAZARDOUS WASTE SITES

PRELIMINARY RISK SCREENING: INTENDED FOR POLICY DEVELOPMENT PURPOSES

ESTIMATES OF LIFETIME INDIVIDUAL CANCER
RISK FOR HAZARDOUS WASTE SITE MODEL SCENARIOS a/

MODEL SCENARIO b/	POTENTIAL CARCINOGEN (WEIGHT-OF-EVIDENCE)	INITIAL EXPOSURE YEAR	PEAK CONCENTRATION (mg/l) c/	PEAK INDIVIDUAL RISK c/	AVERAGE CONCENTRATION (mg/l) d/	AVERAGE INDIVIDUAL RISK d/
5A	2,4-Dinitrotoluene (B2)	11	6×10^{-4}	5×10^{-6}	3×10^{-4}	3×10^{-6}
	Carbon tetrachloride (B2)	e/	--	--	--	--
5B	2,4-Dinitrotoluene (B2)	11	9×10^{-2}	8×10^{-4}	5×10^{-2}	4×10^{-4}
	Carbon tetrachloride (B2)	e/	--	--	--	--
6A	Methylene chloride (B2)	5	1×10^{-6}	2×10^{-10}	2×10^{-7}	4×10^{-11}
	Chlordane (B2)	e/	--	--	--	--
6B	Methylene chloride (B2)	5	9×10^{-4}	2×10^{-7}	9×10^{-5}	2×10^{-8}
	Chlordane (B2)	e/	--	--	--	--
7A	Arsenic (A)	94	2×10^{-10}	9×10^{-11}	7×10^{-12}	3×10^{-12}
7B	Arsenic (A)	89	2×10^{-6}	9×10^{-7}	9×10^{-8}	4×10^{-8}

a/ RISKS ARE LIFETIME INCREMENTAL RISKS ESTIMATED FOR INDIVIDUALS EXPOSED BY GROUND-WATER PATHWAY (i.e., CONSUMPTION OF CONTAMINATED GROUND WATER). THE CARCINOGENIC POTENCY FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND CONTAMINANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE HIGHER OR LOWER; IN FACT, THEY COULD BE ZERO. THE PROPER USE OF THESE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES.

b/ Scenarios 5A through 7B are located in the shale/sandstone hydrogeologic setting, with a ground-water velocity of 100 m/yr.

c/ Peak refers to the highest concentration or risk occurring over the 100-year modeling period.

d/ Average refers to the concentration or risk averaged over the entire 100-year modeling period.

e/ Chemical did not reach exposure point within modeling period because of either retardation or degradation.

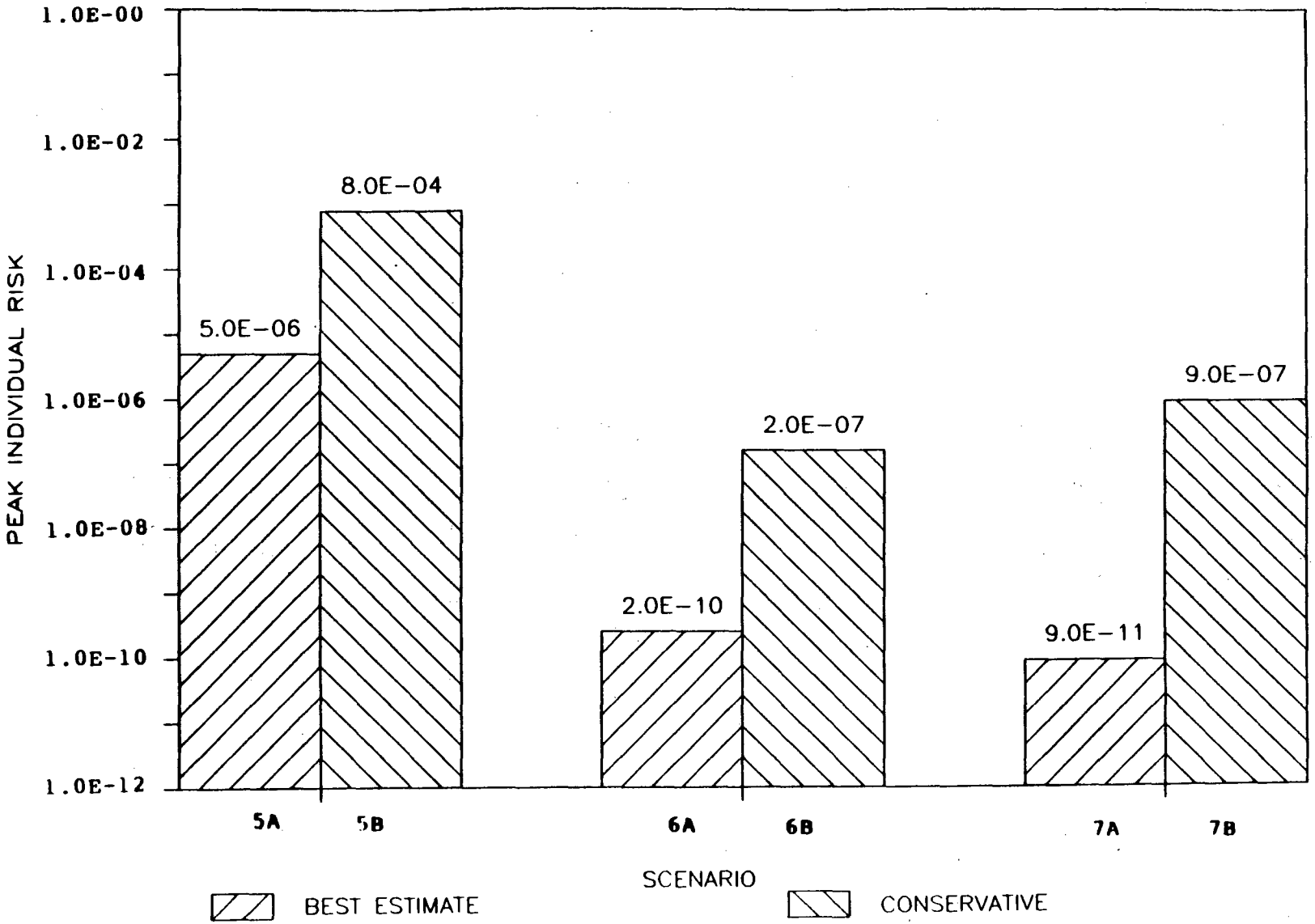
SOURCE: Regulatory Integration Division, Office of Policy Analysis, U.S. EPA, 1987.

FIGURE 2

KANAWHA VALLEY TOXICS SCREENING STUDY/HAZARDOUS WASTE SITES

PRELIMINARY RISK SCREENING: INTENDED FOR POLICY DEVELOPMENT PURPOSES

COMPARISON OF PEAK INDIVIDUAL CANCER RISKS FOR MODEL SCENARIOS a/



a/ RISKS ARE LIFETIME INCREMENTAL RISKS ESTIMATED FOR INDIVIDUALS EXPOSED BY GROUND-WATER PATHWAY (I.E., CONSUMPTION OF CONTAMINATED GROUND WATER). THE CARCINOGENIC POTENCY FACTORS USED IN THIS ANALYSIS ARE BASED ON CONSERVATIVE ASSUMPTIONS THAT GENERALLY PRODUCE UPPER-BOUND ESTIMATES. BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL AREAS OF THE ANALYSIS, SUCH AS EXPOSURE CALCULATIONS AND CONTAMINANT SELECTION, RISK ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL CANCER RISKS IN THE KANAWHA VALLEY. ACTUAL RISKS MAY BE HIGHER OR LOWER; IN FACT, THEY COULD BE ZERO. THE PROPER USE OF THESE ESTIMATES IS TO HELP LOCAL OFFICIALS SELECT AND EVALUATE ISSUES AND SET PRIORITIES.

TABLE 14

KANAWHA VALLEY TOXICS SCREENING STUDY/HAZARDOUS WASTE SITES

PRELIMINARY RISK SCREENING: INTENDED FOR POLICY DEVELOPMENT PURPOSES

SUMMARY OF NONCARCINOGEN EXPOSURES FOR HAZARDOUS WASTE SITE MODEL SCENARIOS a/

MODEL SCENARIO	DISTANCE FROM SOURCE TO EXPSOURE POINT (m)	CONTAMINANT b/	RISK REFERENCE DOSE (RfD) (mg/kg-day)	PEAK CONCENTRATION (mg/l)	PEAK EXPOSURE DOSE c/ (mg/kg-day)	EXCEEDS RfD? (YES/NO)
5A	400	2,4-Dinitrotoluene	d/	6×10^{-4}	2×10^{-5}	N/A
5B	200	2,4-Dinitrotoluene	d/	9×10^{-2}	3×10^{-3}	N/A
6A	400	Methylene chloride	6.0×10^{-2}	1×10^{-6}	3×10^{-8}	No
		Phenol	0.10	4×10^{-4}	1×10^{-5}	No
		Cyanide	0.02	1×10^{-18}	3×10^{-20}	No
6B	200	Methylene chloride	6×10^{-2}	9×10^{-4}	3×10^{-5}	No
		Phenol	0.10	5×10^{-2}	1×10^{-3}	No
		Cyanide	0.02	2×10^{-14}	6×10^{-16}	No
7A	400	Phenol	0.10	2×10^{-4}	6×10^{-6}	No
		Arsenic	d/	2×10^{-10}	6×10^{-12}	N/A
7B	200	Phenol	0.10	2×10^{-2}	6×10^{-4}	No
		Arsenic	d/	2×10^{-6}	6×10^{-8}	N/A

a/ BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL PARTS OF THE ANALYSIS, EXPOSURE ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL EXPOSURES IN THE KANAWHA VALLEY.

b/ Only contaminants reaching the exposure point within the modeling period are listed.

c/ Calculated from peak predicted well concentrations of contaminants, assuming consumption of 2 liters of water/day per person and an average body weight of 70 kg.

d/ Verified RfD not available for this contaminant.

SOURCE: Regulatory Integration Division, Office of Policy Analysis, U.S. EPA, 1987.

To evaluate the importance of this contamination source to overall contamination problems observed in the Kanawha River, we used the LLM to estimate yearly mass loadings of contaminants from scenarios 1 through 4 to the river. We then obtained average initial concentrations of these contaminants in the river resulting from contaminated ground-water discharge by dividing yearly mass loadings by an average annual Kanawha River flow of 1.5×10^{10} m^3 (based on an average hourly flow of 1.7×10^{6} m^3).

The results of this analysis are presented in Table 15. As illustrated, only a few of the contaminants considered in the analysis were of sufficient mobility and persistence to migrate to surface water during the 100-year time period used for the study. Contaminants that tend to bioconcentrate in fish tissues (e.g., chlordane) are generally very hydrophobic and thus are relatively immobile in ground water because they sorb strongly to soil and aquifer materials. Of the chemicals that did migrate to the river, mass loadings were not sufficient to cause significant water quality problems in the river (contaminant concentrations were below federal ambient water quality criteria in all cases), and in most cases river concentrations of contaminants resulting from ground-water discharge would be below detection limits.[12] This is not surprising, given that most ground-water plumes are relatively small and contain small quantities of contaminants when compared to flow volumes in a large river such as the Kanawha. Also, it should be noted that many of the RCRA land disposal units used to develop scenarios 1 and 2 have ground-water monitoring systems that should allow contamination to be detected before a plume could migrate to the Kanawha River. Detection would allow for corrective action to be taken (e.g., ground-water pumping and treatment), thus minimizing surface water contamination via this pathway.

We also estimated mass loadings to and initial concentrations in the Coal River for scenarios 5 through 7. Table 16 presents these results. None of the model results for these scenarios indicate a significant surface water quality problem, even though the Coal River has approximately an order of magnitude lower flow than the Kanawha River. In all cases concentrations were below federal ambient water quality criteria, and in most cases the predicted concentrations would be below analytical detection limits.

V. UNCERTAINTY ISSUES IN GROUND-WATER MODELING

In the hazardous waste part of the Kanawha Valley Toxics Screening Study, we used limited data on site characteristics and contaminants at 25 hazardous waste sites in the Kanawha Valley to develop a group of model scenarios that we believe are representative of the different types of sites in the area. Based on information from characterization surveys, sampling reports, and hydrogeologic investigations of specific sites, we assigned model chemical and hydrogeologic characteristics to each scenario and used the EPA Phase II Liner Location Model (LLM) to evaluate: (1) exposure and risk for scenarios where

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[12] Our preliminary concentration estimates do not account for background contamination in either the Kanawha or Coal River.

TABLE 15

KANAWHA VALLEY TOXICS SCREENING STUDY/HAZARDOUS WASTE SITES

PRELIMINARY EXPOSURE SCREENING: INTENDED FOR POLICY DEVELOPMENT PURPOSES

LOADINGS TO THE KANAWHA RIVER FROM CONTAMINATED GROUND WATER a/

MODEL SCENARIO	CONTAMINANTS	AVERAGE MASS LOADING (kg/yr)	PEAK MASS LOADING (kg/yr)	DURATION OF MASS LOADING OVER 100-YEAR MODELING PERIOD (yrs.)	RESULTANT INITIAL KANAWHA RIVER CONTAMINANT CONCENTRATION (mg/l) b/ c/
1A	2,4-Dinitrotoluene	3.6×10^{-4}	1.4×10^{-3}	22	2.4×10^{-11}
1B	2,4-Dinitrotoluene	$1.5 \times 10^{+2}$	$2.8 \times 10^{+2}$	39	1.0×10^{-5}
2A	2,4-Dinitrotoluene	1.0×10^{-3}	1.7×10^{-3}	70	6.7×10^{-11}
2B	2,4-Dinitrotoluene	1.2	4.7	87	8.0×10^{-8}
3A	Methylene chloride	6.2×10^{-13}	4.3×10^{-12}	68	4.1×10^{-20}
	Phenol	3.1×10^{-14}	1.2×10^{-13}	69	2.1×10^{-21}
3B	Methylene chloride	2.1×10^{-6}	3.4×10^{-5}	79	1.4×10^{-13}
	Phenol	5.2×10^{-9}	2.7×10^{-8}	40	3.5×10^{-16}
4A	Phenol	1.6×10^{-14}	6.0×10^{-14}	69	1.1×10^{-21}
4B	Phenol	2.6×10^{-9}	1.3×10^{-8}	40	1.7×10^{-16}

a/ BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL PARTS OF THE ANALYSIS, SURFACE WATER LOADING ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL EXPOSURES IN THE KANAWHA VALLEY.

b/ Based on average mass loading and average flow of 1.7×10^{6} cubic meters/hour in the Kanawha River (equals approximately 1.5×10^{10} cubic meters/year). These estimates assume dilution by uncontaminated water (background contamination in the Kanawha River is not accounted for). The computation formula is average mass loading (kg/yr) times 1,000,000 mg/kg, divided by [average flow (cubic meters/year) times 1000 liters/cubic meter].

c/ None of the values exceed federal ambient water quality criteria either for aquatic life or human health (at 10^{-6} cancer risk level). Assuming 2 liters per day drinking water exposure, the highest individual cancer risk would be approximately 9×10^{-8} (scenario 1B).

SOURCE: Regultory Integration Division, Office of Policy Analysis, U.S. EPA, 1987.

TABLE 16

KANAWHA VALLEY TOXICS SCREENING STUDY/HAZARDOUS WASTE SITES

PRELIMINARY EXPOSURE SCREENING: INTENDED FOR POLICY DEVELOPMENT PURPOSES

LOADINGS TO THE COAL RIVER FROM CONTAMINATED GROUND WATER a/

MODEL SCENARIO	CONTAMINANTS	AVERAGE MASS LOADING (kg/yr)	PEAK MASS LOADING (kg/yr)	DURATION OF MASS LOADING OVER 100-YEAR MODELING PERIOD (yrs.)	RESULTANT INITIAL COAL RIVER CONTAMINANT CONCENTRATION (mg/l) b/ c/
5A	2,4-Dinitrotoluene	3.3×10^{-2}	5.4×10^{-2}	90	1.7×10^{-8}
5B	2,4-Dinitrotoluene	$1.2 \times 10^{+1}$	$2.2 \times 10^{+1}$	90	6.3×10^{-6}
6A	Methylene chloride	2.2×10^{-5}	9.0×10^{-5}	95	1.2×10^{-11}
	Phenol	9.0×10^{-3}	3.6×10^{-2}	68	4.7×10^{-9}
	Cyanide	2.2×10^{-17}	9.0×10^{-17}	97	1.2×10^{-23}
6B	Methylene chloride	2.3×10^{-2}	2.2×10^{-1}	95	1.2×10^{-8}
	Phenol	3.0	$1.2 \times 10^{+1}$	64	1.6×10^{-6}
	Cyanide	1.2×10^{-12}	4.8×10^{-12}	97	6.3×10^{-19}
7A	Arsenic	8.9×10^{-9}	1.8×10^{-8}	7	4.6×10^{-15}
	Phenol	4.5×10^{-3}	1.8×10^{-2}	68	2.4×10^{-9}
7B	Arsenic	1.8×10^{-4}	4.8×10^{-4}	12	9.5×10^{-11}
	Phenol	1.2	4.8	64	6.3×10^{-7}

a/ BECAUSE OF LIMITATIONS IN DATA AND METHODS IN SEVERAL PARTS OF THE ANALYSIS, SURFACE WATER LOADING ESTIMATES WERE CALCULATED AS AIDS TO POLICY DEVELOPMENT, NOT AS PREDICTIONS OF ACTUAL EXPOSURES IN THE KANAWHA VALLEY.

b/ Based on average mass loading and average flow of 2.2×10^{5} cubic meters/hour in the Coal River (equals approximately 1.9×10^{9} cubic meters/year). These estimates assume dilution by uncontaminated water (background contamination in the Coal River is not accounted for). The computation formula is average mass loading (kg/yr) times 1,000,000 mg/kg, divided by [average flow (cubic meters/year) times 1000 liters/cubic meter].

c/ None of the values exceed federal ambient water quality criteria either for aquatic life or human health (at 10^{-6} cancer risk level). Assuming 2 liters per day drinking water exposure, the highest individual cancer risk would be approximately 6×10^{-8} (scenario 5B).

SOURCE: Regulatory Integration Division, Office of Policy Analysis, U.S. EPA, 1987.

direct exposure to contaminated ground water was plausible; and (2) chemical mass loadings to the Kanawha and Coal Rivers resulting from ground-water discharge for site scenarios in the alluvial setting.

The quantitative analysis described in Section IV is limited by the modeling assumptions used and the uncertainties inherent in using mathematical models as predictive tools. In general, modeling assumptions tend to oversimplify environmental fate and transport (which may result in over-prediction or under-prediction of exposure point concentrations). Also, risks are estimated based on a number of conservative assumptions, including 100 percent absorption of contaminants and the use of CAG upper-bound potency values for carcinogens. Unfortunately, these multiple factors interact in a complex manner, and although risk modeling assumptions and data limitations introduce a significant amount of uncertainty to the analysis, it is impossible to state categorically that the results of the risk analysis are conservative (i.e., that risk estimates are higher than one would expect to find at most sites in the Kanawha Valley).

Appendix B provides a detailed discussion of the uncertainty involved in our quantitative ground-water modeling. Qualitative/semi-quantitative evaluations of ground-water modeling results for the hazardous waste part of the Kanawha Valley Toxics Screening Study indicate that the results have substantial uncertainty associated with them. This uncertainty is partially attributable to assumptions and limitations inherent to the models used in the analysis. However, the greatest contributor to uncertainty in the analysis is probably the lack of comprehensive quantitative information on hazardous waste sites in the Kanawha Valley. Because of this lack of key information necessary for ground-water modeling, the scenarios we developed to represent typical groups of hazardous waste sites were based only on limited data from a relatively small number of sites. We are unable to quantitatively evaluate the degree to which our model scenarios are representative of actual sites in the Kanawha Valley.

From our evaluation of uncertainty issues related to ground-water modeling in the Kanawha Valley Toxics Screening Study, which is included as Appendix B, we have drawn the following conclusions:

- Uncertainty inherent to the LLM version of the Random-Walk Model used in our ground-water analysis is probably not a major contributor to overall uncertainty in the study. For situations in which model conditions and assumptions are met, the model is not likely to be in error by more than an order of magnitude.

- Application of the model to ground-water situations not well matched to model conditions and assumptions may contribute greatly to the overall uncertainty of the ground-water results. Predicted concentrations could be in error by several orders of magnitude for situations where model scenarios do not accurately reflect site conditions. Potential sources

of error include incorrect matching of the model's generic hydrogeologic settings with site hydrogeology and misidentification of retardation factors and degradation potential of chemical contaminants.

- Chemical release modeling algorithms used in the analysis are simplistic representations of complex and possibly transient phenomena. Even under the most ideal circumstances, estimation of a source term for hazardous waste sites is likely to be a substantial contributor to uncertainty in ground-water modeling and exposure assessment.

- Data on geohydrologic and chemical characteristics of hazardous waste sites in the Kanawha Valley were limited and of variable quality. As noted, this lack of data makes it difficult to evaluate the representativeness of our model scenarios, and is a source of considerable uncertainty in the analysis.

The multiple factors in the model interact in a complex manner, and linear propogation of errors cannot be assumed. For example, under-estimation of certain parameters and over-estimation of others may fortuitously produce correct results. Thus, we are unable to estimate quantitatively the overall uncertainty associated with ground-water modeling results in this study. We have described in Appendix B, however, the modeling areas that we believe are responsible for most of the uncertainty.

VI. SUMMARY AND CONCLUSIONS

We structured this analysis to develop a sense of the potential public health concerns related to ground-water contamination from hazardous waste sites within the study area. A review of available information on sites, collected by EPA Region III and the West Virginia Department of Natural Resources, provided data on characteristics of and chemical contaminants detected at 25 hazardous waste sites in the study area. From this information, we developed a group of model scenarios that we believe are representative of hazardous waste sites present in the Kanawha Valley, and quantitatively evaluated exposure and risk for these scenarios. This modeling exercise can only provide a general sense of the potential public health concerns these sites may present, and accounts for only two potential exposure pathways within the Kanawha Valley. We cannot provide a quantitative estimate of potential cancer risk for specific sites from this analysis; however, we believe our analysis of model scenarios may provide a general sense of direction and priorities in the collection of data and information for such an assessment.

The specific findings from our study are as follows:

- The data available for assessing the potential health risks from hazardous waste sites in the Kanawha Valley are limited. Our data base included only 25 RCRA and potential CERCLA sites, which are listed in Table 1. By contrast, EPA Region III has identified 56 uncontrolled waste sites that require a Preliminary Assessment/Site Inspection through the CERCLA program, in addition to numerous RCRA facilities throughout the study area.[13] Our data base represents only a subset of these CERCLA and RCRA sites for which sufficient data to perform a characterization were available.

- Of the 25 sites for which data were available, 17 are located in the river valley alluvial setting and seven are in the shale/sandstone bedrock setting in the northwestern part of the study area. Only one site is located in the sandstone/shale bedrock setting in the southeastern part of the study area.

- Most of the RCRA sites are located in the alluvial setting, while many of the potential CERCLA sites are in the non-alluvial settings outside the valley walls.

- Site monitoring studies have detected many hazardous chemicals present at the 25 sites reviewed in this analysis, as presented in Table 4. These chemicals represent a wide variety of hazardous substances and include a number of potential carcinogens.

- Residents living in the alluvium are served primarily by public water supply systems. Some residents living in the other two hydrogeologic settings, shale/sandstone and sandstone/shale, receive drinking water from private wells.

- Because exposure to contaminated ground water is possible in the non-alluvial regions of the study area, we developed hazardous waste site scenarios that included drinking water exposure for the shale/sandstone setting. For various chemical contaminants typically found at hazardous waste sites in this setting, we estimated a wide range of risks. Our modeling analysis indicates that individual

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[13] The 56 sites either are on the ERRIS site inventory (a listing of all known potential Superfund sites) or have been recommended for the inventory (Source: NUS Corp., Kanawha Valley Study - Phase II Screen of Non-ERRIS Listed Sites, EPA Contract No. 68-01-6699, December 10, 1986).

incremental risks could range from 10^{-12} to 10^{-3} probability (a one in one trillion to a one in one thousand chance) of contracting cancer during a lifetime as a result of consumption of contaminated water. The exposure point concentration and chemical type were the most significant variables affecting risk levels. No noncancer effects due to chronic exposure were predicted.

- Tributaries of the Kanawha River are the primary sources of drinking water for the public water systems. As these tributaries flow to the Kanawha River, they could be affected by discharged ground water contaminated by hazardous waste sites along their banks. To investigate the potential effects of hazardous wastes sites on tributaries, we modeled ground-water discharge to the Coal River, which supplies two public water systems. The highest modeled concentration could result in an individual incremental risk of 6×10^{-8} probability (a six in one hundred million chance) of contracting cancer over a lifetime exposure. This level of risk is driven primarily by chemical type and concentration, as well as the dilution effect of the tributary. The Elk River, another tributary that serves a larger public water supply system, has a much larger flow than the Coal. Consequently, we would expect risks to be lower for the modeled scenarios discharging contaminated ground water to the Elk River.

- The model scenario analysis suggests that mass loadings of contaminants to the Kanawha River via ground-water seepage from hazardous waste sites in the alluvium are likely to be relatively small. In general, loadings from site scenarios were less than one kilogram per year of a modeled constituent discharged to the river. One conservative scenario, a RCRA-type landfill, yielded discharge of approximately 280 kilograms per year of 2,4-dinitrotoluene.

VII. STUDY LIMITATIONS, DATA GAPS, AND RECOMMENDATIONS

This Kanawha Valley hazardous waste site study is a screening-level analysis based on representative model scenarios, and as such provides only limited insight into the problem of hazardous waste contamination in the Kanawha Valley. The study is constrained by a lack of complete data on most sites, by the use of model scenarios to represent groups of what may in some cases be dissimilar sites, and by uncertainties attributable to the

methodologies used to predict exposure and risk. This section outlines limitations of the study caused by information gaps and provides some guidance for areas where further study is needed.

Specific limitations and data gaps in the study can be grouped into the following general areas: (1) limitations in the analysis of exposure and risk via the ground-water pathway; and (2) limitations resulting from focusing on ground-water exposure pathways. These areas are described below.

Limitations and Data Gaps in the Ground-Water Analysis

Although many hazardous waste sites have been identified in the Kanawha Valley, sufficient data for a detailed exposure/risk assessment are available for very few sites. Information on local hydrogeology, types and amounts of wastes present, and potentially exposed populations is lacking for many of the sites used to develop the model scenarios used in the analysis; thus, the quantitative results of the analysis must be viewed with caution. Specific areas in which more data are needed to improve our knowledge of the potential effects of hazardous waste on ground water include the following:

- Potentially exposed populations for the ground-water consumption pathway have not been fully identified for any of the hazardous waste sites in the Kanawha Valley. In some cases, a single ground-water user or a small group of users has been identified near the site, but in other instances there is no population information. It is very important that the number of people potentially affected by these sites be identified, so that the scope of the hazardous waste problem can be better evaluated. A review of potential ground-water users indicated that private wells serve a population outside the valley walls, away from the public water supply system. This study suggests that a survey of private well users in the Kanawha Valley would be a useful first step in any further studies.

- The majority of potential hazardous waste sites in the Kanawha Valley have been identified only by aerial photography. These sites need to be verified as hazardous waste sites or removed from EPA's list of potential sites. The very large number of sites on which there is essentially no information makes the actual magnitude of the hazardous waste problem in the Kanawha Valley difficult to determine.

- Although a number of parameters affect the risk attributable to hazardous waste sites, none have a greater effect than the types and concentrations of contaminants present. Unfortunately, this is the area

of our analysis in which the data are weakest. Future studies at hazardous waste sites in the Kanawha Valley should concentrate on gathering reliable chemical information, preferably from a number of media (e.g., surface soil, subsurface soil, ground water, surface water, biota).

- Detailed hydrogeologic studies were available for only two hazardous waste sites in the area (Artel in the alluvium, and Smith Creek Dump in the shale/ sandstone). Both studies provide a great deal of useful information on the hydrogeology of the area; however, more studies are needed to determine whether these sites are representative of general hydrogeologic conditions in the Kanawha Valley.

- For the effects of ground water on rivers and tributaries, results of the model analysis suggest that sources of pollutants other than ground-water discharge may be more significant and should be investigated. Although we cannot draw firm conclusions on the significance of the modeled discharge rates relative to other sources, it is likely that pollutant loadings from industrial discharges, contaminated sediments, and non-point source run-off, including run-off from hazardous waste sites and industrial facilities, may be more important as major loading sources to the Kanawha River.

Limitations and Data Gaps for Other Potential Exposure Pathways

Exposure via consumption of ground water contaminated by hazardous waste site leachate was the primary exposure pathway considered in this screening-level analysis (surface water contaminated by ground-water seepage was also examined). Although we believe that this pathway is generally the most important for many hazardous waste sites, it is possible that there are other pathways resulting in significant exposure to contaminants released from the Kanawha Valley sites. For example, chemicals may volatilize from surface impoundments, or from contaminated soil or drums, and may be transported to a human receptor by air. Other potential pathways that were not considered in the analysis include wind entrainment of contaminated dust particles, migration of contaminants via surface run-off, and direct contact with materials at the site.

Additional information on hazardous waste sites in the Kanawha Valley would need to be gathered to allow a quantitative evaluation of potential exposures and risks resulting from these other pathways. To evaluate air pathways (volatilization and wind entrainment of dust), information on the following parameters would be needed:

- Wind speed and direction;
- Site topography and vegetation:
- Particle size distribution and moisture content of site soils;
- Climate (e.g., temperature, rainfall, stability class);
- Identity and concentrations of chemicals; and
- Potentially exposed populations (number of people living in proximity to the site).

Some of this information is readily available (e.g., climate information), while obtaining other data (e.g., site topography and vegetation, potentially exposed populations) would require site-specific investigations.

In evaluating the importance of surface run-off as a potential exposure pathway, similar information would be necessary. Data on site topography and vegetation, soil infiltration rate and run-off coefficient, run-off patterns, and rainfall would need to be gathered, as would information on downstream uses of surface water that could be contaminated by this run-off. Potentially exposed populations would need to be identified, and the fate in surface water of the various hazardous contaminants from the site would need to be considered.

To evaluate exposure via direct contact, information on populations living nearby would be needed, as would data on site security, physical barriers surrounding the site, and the ages of people who may come in contact with the site (children are much more likely than adults to be exposed at a significant level via soil ingestion).

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HAZARDOUS WASTE ANALYSIS

Technical Appendices

(available upon request)

Appendix A	Hydrogeologic Characterization of the Kanawha Valley
Appendix B	Uncertainty Issues in Ground-Water Modeling
Appendix C	Hazardous Waste Site Reference List and Source Information

Chapter Eight

Putting Risk In Context

CHAPTER EIGHT

PUTTING RISKS IN CONTEXT

There are several ways to compare environmental risks, each providing a different perspective. One way is to compare risk estimates for the Kanawha Valley to similar risk estimates in other geographic areas, although one must exercise caution in doing so. Studies done for different regions with different objectives often have exposure and risk assumptions that are not easily comparable. However, EPA has estimated some ambient concentrations and risks associated with certain chemicals in different areas of the country. Where those chemicals and exposure assumptions were similar to those studied in Kanawha Valley, we have attempted to present a comparison.

In general, the ambient concentrations and estimated risks from many of the toxic chemicals which we evaluated in the Kanawha Valley appear to be within the range we see in other areas where EPA has data for similar individual pollutants. However, we see concentrations in the Kanawha Valley at both the high and low end of this range.

The risks associated with the toxics studied in public drinking water in the Kanawha Valley appear to be about the same as in other urban areas in the United States which also use surface water as a source. Trihalomethane concentrations measured in Kanawha Valley public water supplies fall in the middle of our comparative range. A comparison of ambient drinking water concentrations of some common pollutants found in different urban areas is presented in Table 1.

For cancer risks from toxic air pollutants, we compared the upper-bound risks estimated in the Kanawha Valley Toxics Study with risks estimated in national studies (NESHAP analyses) conducted by EPA for evaluating various chemicals as potential national Hazardous Air Pollutants under Section 112 of the Clean Air Act. The NESHAP analyses model all major industrial plants within the country emitting a specific pollutant, and estimate national cancer incidence and a range of individual risks to populations surrounding these industrial plants. For the Kanawha Valley Study, we estimated risks to individuals living near industrial plants, and incidence, the increased number of cancer cases attributed to a pollutant within the Kanawha Valley study area.

Table 2 presents a comparison of incidence rate estimates obtained from NESHAP analyses to incidence estimates obtained from the Kanawha Valley Study. Incidence rates are presented in terms of cases per million per year. According to this comparison, seven pollutants found in the Kanawha Valley are approximately equal to or exceed the calculated incidence rate for their respective NESHAP analysis. Risks estimated for three pollutants studied in Kanawha Valley - Ethylene Oxide, 1,3-Butadiene, and Acrylonitrile - significantly exceed their NESHAP analysis. Incidence rate estimates for five pollutants found in the Kanawha Valley are less than their corresponding NESHAP analyses.

Table 3 presents a comparison of increased risks to individuals living within the vicinity of the plants. For the NESHAP analysis, these are the considered most exposed individuals (MEIs), presented as a range to indicate the MEI risks calculated for a set of processes emitting a pollutant. For the Kanawha Valley Study, these estimates are not necessarily for the most exposed individual, but for people who likely received a higher ambient exposure than an average individual within the Kanawha Valley. According to this comparison, several Kanawha Valley pollutants are at the upper end (higher risk) of the range estimated for their NESHAP analyses.

Another perspective on air risks is to consider what people may actually be exposed to in a typical day, including residential, occupational and outdoor air exposures. Since people generally spend a majority of their time indoors, national estimates of the risks from indoor air exposure have been compiled through several studies. These studies have shown that indoor levels of many organic compounds are frequently higher than levels found outdoors. However, these generalized findings may not apply to several of the highest risk Kanawha Valley air pollutants (of those studied), which originate primarily from outdoor point sources.

A third perspective on risk is to compare the risks estimated from environmental exposures to mortality and sickness (morbidity) attributable to a variety of other causes. When considering this type of comparison, keep in mind that mortality and morbidity statistics describe actual occurrences of disease in the past. The risks calculated for the Kanawha Valley Toxics Screening Study are plausible, upper-bound estimates and may be overstated. For a limited number of pollutants, the Kanawha Valley Study estimated 0.3 additional annual cancer cases from toxic pollutants in drinking water, and 0.7 to 1.8 in air. This compares to between 275 to 650 estimated cancer cases which occur annually in the Valley from all causes.

Even when we have considered our risk estimates in the context of other risks, and formed an opinion about the significance of those risks, we are still faced with the question of whether or not the risks are acceptable. Central to this question is how risk is perceived. For instance, people may find it hard to relate to large, abstract estimates such as a 1 in 10,000 chance of contracting cancer over a lifetime.

Table 4 presents some common risks we face. Risk may be influenced by factors such as voluntariness, controllability, familiarity or by the immediacy, magnitude, and reversability of the consequences of the risks faced. For example, people may find some risks more acceptable if benefits accrue directly to them for incurring this risk.

Having considered the severity and significance of the risks in question still leaves the management decision of whether to act to control the risk now, engage in further study, or accept the risks. The technical feasibility of control, the availability of resources, and the economic implications are some of the most important factors, particularly given the conservative nature of the risk estimates. Public concern and perception will always weigh heavily in these risk management decisions.

TABLE 1

DRINKING WATER

COMPARISON OF AMBIENT LEVELS OF SELECTED TOXICS IN KANAWHA VALLEY PUBLIC WATER SUPPLIES TO LEVELS IN OTHER AREAS

All Values in Micrograms/Liter (ug/l)

SUBSTANCE	SANTA CLARA COUNTY 1	NATIONAL STUDIES 2	KANAWHA VALLEY 3
Trihalomethanes	4 - 80	NA	42-77
Arsenic	0.3 - 15	10 - 1000	<1 -10
Barium	<100 - <1000	0 - 172	<1 -100
Cadmium	0 - 10	NA	<.1-.2
Chromium	1 - 8	0 - 112	< 1- 2
Lead	1 - 40	NA	< 1- 1
Mercury	0 - 6	0.1- 1.8	<.2-.4
Selenium	1 - 10	0 - 10	< 2- 6
Silver	1 - 20	NA	<.2
Zinc	1 - 90	NA	< 5 -10

1 SOURCE: Santa Clara Valley Integrated Environmental Management Project; Tables 4 - 11, Exposure to metals and minerals
2 SOURCE: National Organics Monitoring Survey, 1977
3 SOURCE: Kanawha Valley Toxics Screening Study
4 NA : Not Available

TABLE 2

COMPARISON OF ANNUAL CANCER INCIDENCE
FOR SELECTED AIR TOXICS IN
KANAWHA VALLEY WITH INCIDENCE RATES
FROM NATIONAL EMISSION STANDARDS
FOR HAZARDOUS AIR POLLUTANTS (NESHAP)

(Annual Incidence per Million Population)

POLLUTANT	NESHAP STUDY[1]	KANAWHA VALLEY[2]
Ethylene oxide	.23	3.48
Chloroform	.10	.58
1,3-Butadiene	.35	1.15
Acrylonitrile	.01	.81
Carbon tetrachloride	<.01	.08
Methylene chloride	.14	.1
Trichloroethylene	.02	<.001
Vinylidene chloride	.02	.01
Vinyl chloride	2.34	<.001
Benzene	>.2	<.001
Ethylene dichloride	.01	<.001
Allyl Chloride	Not Available	<.001

[1] Source: Communication from Pollutant Assessment Branch, OAQPS. Estimates developed from "Intent to List" Federal Register Notices or equivalent information.

[2] Source: Kanawha Valley Toxics Study

TABLE 3

COMPARISON OF UPPER-BOUND

ESTIMATES OF LIFETIME CANCER RISKS

TO THE MOST EXPOSED INDIVIDUAL

POLLUTANT	NESHAP Range [1]	Kanawha Valley (Zone) [2]
Ethylene oxide	10^{-7}-10^{-3}	5×10^{-3} (S. charleston)
Chloroform	10^{-6}-10^{-2}	3×10^{-3} (Belle)
1,3-Butadiene	10^{-6}-10^{-1}	6×10^{-3} (Institute)
Methylene chloride	10^{-5}-10^{-3}	5×10^{-4} (Belle)
Carbon tetrachloride	10^{-6}-10^{-3}	4×10^{-4} (Belle)
Acrylonitrile	10^{-6}-10^{-3}	9×10^{-4} (S. Charleston)
Trichloroethylene	10^{-5}-10^{-3}	6×10^{-6} (Nitro)
Vinylidene chloride	10^{-5}-10^{-4}	1×10^{-5} (S. Charleston)
Vinyl chloride	10^{-8}-10^{-3}	2×10^{-8} (Nitro)
Benzene	10^{-8}-10^{-3}	3×10^{-7} (Institute)
Ethylene dichloride	10^{-7}-10^{-2}	9×10^{-7} (Nitro)

[1] Source: Communication from Pollutant Assessment Branch, OAQPS. Estimates developed from "Intent to List" Federal Register Notices or equivalent information.

[2] Source: Kanawha Valley Toxics Study

TABLE 4

SELECTED INVOLUNTARY RISKS

	Source for Wilson Derivation
Lightening Striking	
-10-6 risk of death in 2 years	U.S. Census (1975)
10-5 risk of death in 20 years	
Motor Vehicle Accidents	
-10-6 risk of death in 1.5 days	National Safety Council (1950-78)
-10-3 risk of death in 4 years	
Sulfate Air Pollution	
-10-3 risk of death in a lifetime	Derived from several sources
Radon	
-10-2 to 10-3 risk of lung cancer in a lifetime in the overall population	Office of Policy Analysis derived from EPA published information

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Appendices

APPENDIX A

SOME ENVIRONMENTAL ACTIVITIES NOT ADDRESSED IN THE KANAWHA VALLEY TOXICS SCREENING STUDY REPORT

I. Accidental Release Prevention, Preparedness and Response

- Federal/State

  - The Chemical Emergency Prepardeness Program (CEPP) was established by EPA to provide guidance to local communities for the preparation of contigency plans to handle accidental air releases of toxic substances. The CEPP criteria document includes the list of 402 acutely toxic chemicals, which is used in planning operations.

  - EPA is initiating The Accidental Release Information Program (ARIP) designed to compile data on the nature and causes of accidental releases and the measures taken by industry to prevent them. The early stages of data gathering is being executed by letters to selected facilities requesting information on releases. However, the ultimate goal of the program is to publish regulations requiring companies to report certain data subsequent to a spill or release.

  - The recently approved amendments (SARA) to the Superfund law (CERCLA) includes a provision entitled Title III which institutionalizes and expands CEPP by setting up reporting requirements for facilities that handle hazardous materials. The facilities will report to a Governer appointed State Commission which oversees emergency prepardeness and prevention activities. The State Commission will designate local emergency planning districts and local emergency planning commities who will collect information and data through the right-to-know provisions of the statute.

  - EPA has established an Emergency Systems Review Workgroup to review, on a national level, emergency systems for monitoring, detecting and preventing releases of toxic substances. It is expected that a wide range of potential accident prevention techniques will emerge from the workgroup project.

- EPA Region III and The West Virginia Department of Natural Resources are parts of a Regional Response Team (RRT), established to respond to chemical emergencies that are a imminent treat to the public health or the environment. The RRT has limited contractor authority and can call on the resources of various State and Federal agencies for the purpose of mitigating a hazardous situation.

- The West Virginia State Legislature passed a Right-to-Know Law which calls for certain reporting requirements for facilities that handle hazardous materials. This information is submitted to The State Department of Health in the form of Material Safety Data Sheets.

- The prevention of transportation accidents is enhanced through the hazardous substances handling and shipping requirements administered by the U.S. Department of Transportation (DOT). The DOT has set regulations on the classification of hazardous substances as well as standards for packing, labeling, and shipping of the different classes of materials.

- The National Institute for Chemical Studies (NICS) was formed in early 1985 by West Virginia Governor Arch Moore to serve as a "bridge" between the general public and the chemical industry, and to assess the seriousness of known or perceived risks in the Kanawha Valley to determine how effectively those risks are being managed. NICS Safeguard Report, published and released in November 1986, examines the onsite accident prevention and risk management activities of Valley chemical companies.

- Local

  - Kanawha County and the City of Charleston are cooperating with the Kanawha Valley Emergency Planning Council (KVEPC) in building a new, state-of-the-art, Metro Emergency Operations Center, which will be the headquarters for all response activities in the Kanawha Valley. The Center is scheduled for opening in the Spring of 1987.

  - The Kanawha County Office of Emergency Services has developed one mile emergency planning zones (EPZs) around each major chemical plant. These EPZs will become the focus of special efforts in emergency response planning.

  - Agreements have been reached with the Kanawha County Board of Education and the Kanawha Regional Transit (KRT) to assure their cooperation in the event of an emergency. Two way radios have been placed on all KRT buses and 67 radios have installed in Kanawha County schoolbuses.

- Industry

  - The Kanawha Valley Emergency Planning Council (KVEPC) is an area coalition of chemical companies whose purpose is to share knowledge and resources for preventing and responding to emergencies at member facilities. The KVEPC is also interested in outside response in the Kanawha Valley and has produced the "Path Forward" Report, which presents a point-by-point plan to upgrade emergency response in the region.

  - In an attempt to prevent accidents before they occur, several companies in the area are performing formal engineering risk assessments at the design stage for each major high hazard chemical process. In addition the American Institute of Chemical Engineers Center for Chemical Process Safety (CCPS) is planning to publish a manual of engineering guidelines for detection, containment and isolation of accidental releases, which could lead to some additional safety modifications and retrofitting at Valley chemical plants.

  - Several accident preventative quality control programs are being conducted by many companies in the Valley. The Process Hazard Management Review Program at DuPont and Union Carbide systematically identifies and evaluates the impacts of potential process hazards. The Tentative Process Amendment Program at Monsanto is used to plan and implement significant changes or major modifications to exsisting chemical processes, and FMC has commissioned a full scale corporated-led process hazards review project at their facilities.

  - Some chemical companies are effectively reducing the risk of an accidental release occuring by reducing their inventories of hazardous materials. Some plants utilize the "just-in-time" method of scheduling raw material or product shipments in order to minimize storage. Other chemical plants are specifically designed to completely use up quantities of chemicals immediately subsequent to receiving them.

  - Since most accidents at chemical plants are caused by human error, many facilities are upgrading their operator training programs. Union Carbide has plans to implement an operator training, testing, and certification program which utilizes a simulator unit equivilant to those used by airline pilots. Monsanto and DuPont are using on-the-job evaluation techniques to rate operator performance. The results are communicated to workgroups for corrections and follow-up.

II. Indoor Air

- The Kanawha Valley Toxics Study did not include sampling of indoor air. The EPA has studied indoor air in six U.S. cities over the past few years and exposure data has been remarkably consistent from city to city. Charleston was not involved in these TEAM (Total Exposure Assessment Methodology) studies, however, relative assumption indoor air quality may be made due to the data consistency.

- The EPA TEAM Study is the largest of the personal exposure studies performed to date. The study measured personal exposure of about 600 residents of five geographical areas between 1980-84. Concurrent outdoor air measurements were made near 200 of the homes, allowing direct comparison of indoor-outdoor levels for the TEAM target compounds, which included many of the Kanawha target compounds. In essentially all cases, the indoor levels of the target chemicals outweighed the outdoor levels, often by factors of 2-5. These basic findings have been repeated in a number of studies by different investigators in different countries.

- The sources of the high indoor and personal exposures have been identified for some carcinogenic target compounds (benzene, chloroform, tetrachloroethylene) but not for others (trichloroethylene, carbon tetrachloride). The major source of benzene exposure is undoubtedly mainstream cigarette smoke, which contains about 55 ug/cigarette. Other main sources of benzene include environmental tobacco smoke and auto-related activities such as extended commuting time and filling a gas tank at a service station.

- The major source of airborne chloroform exposure appears to be the use of hot water in the home. Recent unpublished work by EPA/ORD indicates that washing clothes or dishes is correlated with higher levels of chloroform in the home than taking showers or tub baths. In addition, use of chlorine bleaches may create and release considerable quantities of chloroform. The major source of tetrachloroethylene is wearing and storing dry-cleaned clothes. Levels in homes remain elevated for at least a week following storage of freshly dry-cleaned clothes in a closet.

- It would seem that, for the chemicals described above, exposures from indoor sources and activities are normally dominant over exposures from outdoor concentrations, even when one's residence is in the heart of a chemical manufacturing district. With the exception of chloroform, the outdoor levels observed in the Kanawha Valley during this study are typical of those observed in many other areas of the country. Therefore it may be concluded that the indoor concentrations and resulting personal exposures of Kanawha Valley residents, as in all other areas of the country so far studied, are important in terms of long-term risks and should not be taken lightly when considering total exposure risk reductions.

III. Epidemiological Health Effects Study

- The Kanawha Valley Toxics Screening Study was not epidemiological in nature; that is, it did not examine current rates or patterns of disease incidence in the resident population and attempt to link these health conditions with previous levels of chemical exposure.

- The U.S. Congress through the work of Senator Robert Byrd appropriated $250,000 to explore possible designs for an epidemiological health effects study in the Kanawha Valley.

- The study design is being conducted by a consortium of the National Institute for Chemical Studies (NICS), the West Virginia Department of Health and the Harvard University School of Public Health (HSPH) through a grant administered by EPA.

- Phase I of this study was initiated in October, 1986 and will be implemented for a period of approximately fourteen months ending in December, 1987. This effort includes: identifying chemicals of significant concern in the ambient atmosphere; identifying potential health endpoints relating to chemical exposure versus time; and defining exposure gradients in the study population which would optimize the sensitivity and validity of the study.

- Additional Phase I activities include: conducting ambient air monitoring, and possibly indoor air and water quality monitoring to better define an exposure gradient; organizing two workshops convening experts in the fields of toxicology and epidemiology to meet with local health officials to review and interpret available health and exposure data; and establishing a process to continually assess new health and exposure information from a toxicological perspective.

IV. Occupational Risk

- The Occupational Safety and Health Administration (OSHA) has the responsibility for assuring that workers in the Kanawha Valley are provided with safe and healthful working conditions. OSHA works toward this goal by conducting safety and health inspections in the workplace.

- OSHA has established permissable exposure limits for approximately four hundred chemical substances. These permissable exposure limits (PELs) are eight hour time weighted average airbourne concentrations to which most workers may be exposed on a daily basis without experiencing adverse health effects. This simply means that a chemical may exceed a PEL for a short period of time, however, the chemical may not exceed the PEL when taking the average concentration over an eight hour period. For a limited number of chemicals ceiling levels (levels which cannot be exceeded for any period of time) have also been established.

- In order to further address the unique potential hazards that exist in the chemical industry, OSHA developed a Pilot Special Emphasis Program for inspections of certain chemical establishments. These inspections are designed to focus on process safety, backup system, preventive maintenance programs and emergency response procedures. OSHA developed and used a High Hazard Chemical List to further concentrate on those facilities that produce high risk chemicals. The Charleston OSHA office was one of ten offices chosen to participate in the pilot program which resulted in some notable inspections conducted in chemical plants in the Kanawha Valley.

- Four seperate inspections were conducted at the Union Carbide plant in Institute between December 1984 and April 1986. The inspections covered such things as the methyl isocyanate manufacturing and handling operations and five other process units in the plant. Emergency response procedures were also reveiwed by OSHA.

- Two inspections of the Union Carbide South Charleston Plant were concluded in August 1985 and October 1986. The first inspection was a result of a alledged release of mesityl oxide and acetone in which OSHA recommended design improvements that could be made to the distillation equipment. The second inspection scrutinized the acrylonitrile processing unit in which several respirator violations were cited, however, there were no potential exposures noted from company sampling data.

- The Artel Chemical Co. (formerly Fike Chemical) was inspected between May - July 1986 because of an accidental release of chlorine gas from the plant. Actual exposure levels could not be documented and no OSHA sampling was conducted.

- The Occidental Chemical Corporation inspection ending in December 1986 was initiated as a result of an accidental release of chlorine, carbon tetrachloride, chloroform and methylene chloride. Again actual exposure levels could not be documented. Process safety deficiencies were documented and cited during the inspection.

APPENDIX B

HISTORY OF DEVELOPMENT AND EMPLOYMENT IN KANAWHA VALLEY[1]

The history of the Kanawha Valley is extremely interesting, involving names such as Daniel Boone, George Washington, William McKinley, and Rutherford B. Hayes. Events such as the French and Indian War, the Revolutionary War, the Civil War, and the two World Wars also played a major role in shaping the development of the Valley.

Although not established by an act of the Virginia General Assembly until 1794, Charleston's origins date back to the pre-Revolutionary War days. Originating as a military outpost in the Virginia system of the frontier, Charleston and its surrounding communities developed into a major industrial and commercial area strategically located near the large urban areas of the East, South and Midwest.

Charleston's origins reach back to the closing days of the French and Indian War. The first white man of record to view the site was Matthew Arbuckle, who, in 1764, passed from the Greenbrier Levels (Lewisburg) down the Kanawha Valley to a French trading post on the site of Point Pleasant.

In April, 1788, a company of Virginia Rangers under Colonel Charles Clendenin erected a stockade fort at the corner of the present Brooks Street and Kanawha Boulevard, officially known as Fort Lee, which was a part of the frontier defense until the close of the Indian wars in 1795. In 1794, forty acres of the Clendenin holdings were laid out in lots, and a town was authorized by the Virginia Assembly to be known as Charleston in honor of Charles Clendenin. On January 19, 1818, Charleston, by name, was officially established.

The early years of Charleston were much interwoven with the story of the great salt industry of the Kanawha Valley. As early as 1796, salt was manufactured at the mouth of Campbells Creek, almost directly opposite the homeplace of Daniel Boone, who resided at the present Kanawha City from 1790 until 1795. This industry rose to great proportions. By 1860, salt was shipped to all parts of the U.S. from some 49 great furnaces. In addition to the salt industry, natural curiosities known as "burning springs" were found, from which exuded natural gas. Two such areas, above the suburb of Malden, were embraced in a "military survey" owned jointly by General George Washington and General Andrew Lewis.

During the Civil War, the town of some 1,500 population was much divided, and many citizens served in the Federal Army and many in the Confederate Army. It was an important military post and the nominal head of navigation for river transportation of troops. The Battle of Scary was fought a few miles below the city in 1861, and

Camp Platt was located a few miles above. Fort Scammon, located in the city, was an important post, and the remains may still be seen. Camp White, on the "South Side", was occupied by many notable men, including William McKinley and Rutherford B. Hayes.

The Chesapeake & Ohio Railroad was completed through the city in the spring of 1873. This was followed by construction of the Ohio Central Railroad, between 1880 and 1883, which is now part of the Consolidated Rail Corporation. The Coal & Coke Railroad was opened in 1906, from Charleston to Elkins, using in part an earlier railroad constructed in the Elk River Valley. This rail system is now part of the Baltimore & Ohio System.

The industrial and commercial history of Charleston falls logically into three phases since its founding by the location of Fort Lee in 1788. The first era, from the early time of the city to as late as 1859, centers around the great salt industry. The second era saw the development of Charleston as a market and wholesale town to serve the rapidly developing coal mining industry in southern West Virginia. The third, or present era is marked by the development of Charleston and environs as one of the great chemical centers of the U.S., based upon the manufacturing use of the natural resources which surround the city.

The present era is further divided into the gas and glass period, the war period and the synthetic period. The gas and glass period was a result of the extensive development of oil and gas fields around Charleston. The war period saw the location and expansion of a number of manufacturing plants which were designed to be converted easily into war munitions plants. The synthetic period, or the present period, is marked by the location of internationally known chemical industries in the Valley. Like earlier industries, most of these are based upon the salt brine and other natural resources underlying the Valley.

The presence of manufacturing plants, the nearby location of natural resources such as coal, oil and gas, and the provision of facilities for transportation, led to the location of the U.S. Naval Ordnance Plant at South Charleston and the U.S. Government's Nitrocellulose Plant at Nitro, both in 1917. As part of the war production effort in 1942-43, the nation's then largest installations for the production of bulk synthetic rubber were built at Institute.

The old Naval Ordnance Plant complex in South Charleston has been completely renovated and is now called the Charleston Ordnance Center, providing space for over 30 small companies as well as the Charleston Stamping Plant of Volkswagen of America, Inc. The nitrocellulose plants have been cleared from their original sites at Nitro, but

FMC, Monsanto and other major companies have developed facilities in the same area. The bulk synthetic rubber property at Institute today belongs to the Union Carbide Corporation, the Kanawha Valley's largest employer.

While major industrial activity and employment have declined in recent years (see TABLE 1 on next page), the Valley remains one of the largest industrial complexes in the United States.

TABLE B-1

ANNUAL AVERAGE
EMPLOYMENT TRENDS 1972 - 1983
CHARLESTON SMSA - KANAWHA & PUTNAM COUNTIES

	1972	1973	1974	1975	1976	1977	1978	1979*	1980*	1981**	1982***	1983****
LABOR FORCE	102,800	105,900	107,500	111,400	113,800	117,500	118,800	122,600	127,300	121,100	119,100	118,000
EMPLOYMENT	97,600	100,300	102,800	105,100	106,200	112,800	114,300	117,900	119,700	112,900	110,300	106,300
UNEMPLOYMENT	5,200	4,600	4,800	6,400	5,300	4,700	4,500	4,700	7,500	8,100	8,900	11,700
% UNEMPLOYMENT	5.1%	4.3%	4.3%	5.7%	4.7%	4.0%	3.8%	3.9%	5.9%	6.7%	7.4%	9.9%
SELECTED SECTORS												
WHOLESALE & RETAIL TRADE	20,900	21,900	22,300	22,650	23,300	25,300	25,500	27,300	26,700	26,200	25,700	25,600
MANUFACTURING	17,500	17,800	18,400	17,600	19,000	19,700	19,600	19,600	18,100	16,700	16,100	14,300
GOVERNMENT	15,700	16,100	17,200	17,500	18,600	18,700	18,800	21,600	23,000	20,600	20,400	20,500
SERVICES	14,200	14,700	15,700	15,800	17,000	18,500	18,500	20,100	20,400	20,700	21,300	21,200

SOURCE: West Virginia Department of Employment Security
Labor & Economic Research Section
*Revised to March, 1980 Benchmark
**Revised to March, 1981 Benchmark
***Revised to March, 1982 Benchmark
****Preliminary, based on March, 1982 Benchmark

Somewhat surprisingly, a look at Charleston's employment structure reveals that the most identifiable enterprises - manufacturing - actually account for a relatively small portion of the region's total economic activity. This holds for both the data given in Table 1 above, and for 1984 figures. In fact, out of all persons employed in the Charleston SMSA on the average for 1984:

> Wholesale and retail trade employed more persons than any other sector of the local economy in 1984. Approximately 27,600 persons had jobs in retail establishments, warehouses, and distribution centers. This is equal to 25.9% of all employed persons in 1984.
>
> The second largest sector was services, which employed 22,100 persons, or 20.7% of all those employed in the region in that year. The major group within this sector was health services, which employed 9,200 persons.
>
> The next largest sector of employment was government, which employed 21,300 persons or 20% of the total employed. While much of this was state government employment, since Charleston is the capital city, it also included a substantial number of city, county and federal employees.
>
> The fourth largest economic sector was manufacturing, with 13,300 persons, or 12.5% of all those employed. Chemical manufacturing dominated this sector with over 9,800 employees.[2]

In terms of average weekly wages, however, manufacturing has accounted for the highest incomes. Table 2 breaks down the average weekly wages, by industry or business, for Putnam and Kanawha Counties for 1983, 1984, and 1985.[3]

TABLE B-2

	Putnam County			Kanawha County		
	1983	1984	1985	1983	1984	1985
Mining	N.A.	252.04	357.92	N.A.	588.27	600.23
Construction	N.A.	374.02	362.48	416.68	421.90	428.23
Manufacturing	493.14	539.69	568.81	N.A.	571.44	604.10
Trade	N.A.	257.31	260.94	N.A.	266.04	267.63
Retail	N.A.	213.26	211.25	N.A.	203.90	203.93
Finance	N.A.	292.52	299.19	N.A.	352.92	374.44
Services	N.A.	267.42	310.96	N.A.	304.42	314.90
Government	N.A.	302.33	325.28	N.A.	323.22	346.60

With respect to specific industries identified in the Kanawha Valley study, available employment figures for industries in the two counties are given in Table 3.[4]

TABLE B-3

Putnam County		Kanawha County	
Company	Employees	Company	Employees
Allied Chemical	71	Diamond Shamrock (Occindental)	74
FMC	160	Dupont	1061
Monsanto	548	FMC	806
		Union Carbide (South Charleston)	1172
		Rhone-Polenc (Institute)	1408
		Union Carbide (Technical Center)	3629

In terms of employment outlook, it has already been indicated that the region has been declining in both labor force and employment since 1980, as compared to increases in both categories from 1972 up to, and including, 1980. Compared to the 1984 data given above, however, it appears that the decline in the trade, government, and service industries has been reversed. By comparison, the manufacturing sector has continued to decline, based on the available data. The trickle down effect of an improved national economy may have a positive effect on the local manufacturing sector. However, it might be presumptuous to expect marked improvement.[5]

APPENDIX C

SCIENCE ADVISORY BOARD REVIEW

INTEGRATED ENVIRONMENTAL MANAGEMENT SUBCOMMITTEE

- Final Comments - May 27, 1987
- List of IEM Subcommittee Members

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

JUL 14 1987

THE ADMINISTRATOR

Ronald Wyzga, Chairman
Integrated Environmental
Management Subcommittee
Science Advisory Board
U.S. Environmental Protection Agency
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Washington, D.C. 20460

Dear Dr. Wyzga:

Thank you for your letter of May 27, 1987, transmitting final comments on the Kanawha Valley Toxics Screening Study. I originally requested a Science Advisory Board review of this Study because of the complex technical questions concerning toxics pollution in the Kanawha Valley and my desire to base any decisions concerning future EPA efforts there on sound scientific analyses. I was, therefore, gratified to note in your letter that "the study reaches a number of scientifically supportable conclusions about health risks from cancer in the Kanawha Valley. The study also points EPA and other interested parties in a direction for conducting further analyses of problems . . .".

The specific technical points raised in Attachment A of your letter have been reviewed by the EPA offices responsible for conducting the Study (Region III's Environmental Services Division and the Office of Policy, Planning, and Evaluation). Many of these points have already been incorporated as part of revisions to the Draft Report. In addition, we will continue to evaluate the resource implications and future possibilities for improved study results from implementing the suggestions contained in your letter, including:

- Replacing several of the Study's "large assumptions" with improved monitoring data to more precisely characterize exposure;
- Replacing the Study's hypothetical facility approach for estimating hazardous waste risks with one that examines specific hazardous waste sites; and
- Examining potential acute and non-cancer health effects and risks from accidental releases of toxic contaminants.

The Kanawha Valley Toxics Screening Study identified several potentially serious risks which suggest the need for further analysis and possibly risk management actions. We are now working with the appropriate West Virginia agencies to develop specific plans for responding to the risks and needs identified in the Study. Industry within the Valley has shown a willingness to take voluntary steps to reduce emissions where they contribute to significant risks. Other ongoing work will also contribute to a better understanding of environmental risks and control options, including the health effects feasibility study now underway through the National Institute for Chemical Studies.

Again, I appreciate the efforts, conclusions, and insights of the IEM Subcommittee concerning the Kanawha Valley Study. I also look forward to receiving your report on the full Integrated Environmental Management Program in the near future.

Sincerely,

Lee M. Thomas

cc: A. James Barnes
Terry Yosie
IEM Subcommittee Members
James Seif
John Campbell

APPENDIX C

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

May 27, 1987

SAB-EC-87-031

OFFICE OF
THE ADMINISTRATOR

Honorable Lee M. Thomas
Administrator
U. S. Environmental Protection Agency
401 M Street, S. W.
Washington, D. C. 20460

Dear Mr. Thomas:

The Science Advisory Board's Integrated Environmental Management Subcommittee has completed its review of EPA's Draft Kanawha Valley Toxics Screening Study and is pleased to transmit its final report to you. The Subcommittee met in public session on March 16, 1987 in Philadelphia, Pa., to review the study. During March 11-13, 1987, three representatives of the Subcommittee visited the Kanawha Valley to become more familar with its environmental problems.

The Subcommittee unanimously concludes that the Kanawha Valley study represents an important component of EPA's overall effort to develop methodologies to define public health and environmental priorities. Studies such as this provide valuable technical challenges and experiences to EPA staff, particularly those working in regional offices. And, finally, they provide a valuable means for developing closer working relationships with state and local officials and the general public.

This letter is the Subcommittee's second communication to you. On July 30, 1986 it expressed "many concerns about the ability of the current study to satisfy a number of technical issues. A chief concern is the incongruity between [the study's] .... objectives and the fact that the study design itself is not an integrated multimedia effort, nor a response to Bhopal."

Since the transmittal of that letter, EPA staff have modified the study's objectives and technical design, and have conducted supplementary analyses to support the revised objectives and design. In general, the Subcommittee believes that the staff have made appropriate responses to its major concerns. The study reaches a number of scientifically supportable conclusions about health risks from cancer in the Kanawha Valley. The study also points EPA and other interested parties in a direction for conducting further analyses of problems related to accidental releases of pollutants and acute health effects.

Specific issues addressed during the Subcommittee's review include: the study's objectives and scope; pollution sources; pollution transport and fate by media; health effects; risk communication; and recommendations for additional follow-up efforts. Attachment A presents additional, more-detailed recommendations for modifying the current study and future activities in the Kanawha Valley. Attachment B lists the Subcommittee members.

In general, the Subcommittee views the Draft Kanawha Valley Toxics Screening Study as one step of a continuing process to assess risks. The current study addresses chronic health exposures to carcinogens which represent one of many public health concerns in the Valley. As a follow-up to the current study, the Subcommittee recommends two additional steps that include:

- o Expanded monitoring of air toxics, and use of monitored values to obtain more precise estimates of exposure and health risks.

- o Greater focus on accidental releases and fugitive emissions as areas of public health concern.

The Subcommittee appreciates the opportunity to conduct an independent scientific review of these important public health issues in the Kanawha Valley. We request that EPA formally respond to our scientific advice.

Sincerely,

Ronald Wyzga, Chairman
Integrated Environmental
Management Subcommittee
Science Advisory Board

Norton Nelson, Chairman
Executive Committee
Science Advisory Board

SAB-EC-87-031

MAJOR FINDINGS AND RECOMMENDATIONS

OF THE

INTEGRATED ENVIRONMENTAL MANGEMENT SUBCOMMITTEE

ON THE

U. S. ENVIRONMENTAL PROTECTION AGENCY'S DRAFT

KANAWHA VALLEY TOXICS SCREENING STUDY

INTEGRATED ENVIRONMENTAL MANAGEMENT SUBCOMMITTEE

SCIENCE ADVISORY BOARD

U. S. ENVIRONMENTAL PROTECTION AGENCY

May, 1987

U. S. ENVIRONMENTAL PROTECTION AGENCY

NOTICE

This report has been written as a part of the activities of the Science Advisory Board, a public advisory group providing extramural scientific information and advice to the Administrator and other officials of the Environmental Protection Agency. The Board is structured to provide a balanced expert assessment of scientific matters related to problems facing the Agency. This report has not been reviewed for approval by the Agency, and hence the contents of this report do not necessarily represent the views and policies of the Environmental Protection Agency, nor of other agencies in the Executive Branch of the Federal government, nor does mention of trade names or commercial products constitute endorsement of recommendation for use.

Study Objectives and Scope

The objectives of the Kanawha Valley study are limited, but reasonably well-defined. In most instances, the study seeks to derive an upper bound for the health risks associated with airborne carcinogens for which EPA's Cancer Assessment Group has derived potency estimates. Other potential carcinogens are minimally considered, and the health risks of non-carcinogens, including those risks associated with the accidential release of chemicals such as occurred at Bhopal, are not considered. Hence, the health assessment of airborne toxics is far from complete, but this is clearly articulated in the study report. Available resources did not allow a more comprehensive assessment.

The study attempts "to develop a sense of potential public health concerns" associated with carcinogens in drinking water, surface water and hazardous wastes. The efforts are not multimedia efforts, but medium-specific efforts based upon very limited data; thus, conclusions from these efforts are subject to considerable uncertainty.

Sources

The air analyses depend very heavily upon an emissions inventory of some 450 substances developed by the West Virginia Air Pollution Control Commission (APCC). The inventory is as extensive and comprehensive as any other available information. Nevertheless, there exist some uncertainties in the inventory, particularly with respect to fugitive emissions, which the study identifies as a major source of health risk in some Kanawha Valley communities. The possibility that the inventory is incomplete is also suggested by the fact that ethylene oxide was not included for either the Belle or Nitro communities despite some limited monitoring evidence that it may be present. If a compound was not in the inventory it was not included in subsequent EPA modeling. This discrepancy underlines the need for including ethylene oxide in future monitoring programs.

The drinking water and surface water analyses depend upon monitored levels of toxics in water supply systems and fish fillets, respectively. Data are limited to a subset of all public water suppliers, with no private well samples, and to a very small number of fish sampled from only one location for a very limited number of toxic substances. The hazardous waste inventory is based upon a priority pollutant screening of inventories for a subset of RCRA and potential CERCLA sites. No information was available on the total quantity and overall composition of toxic wastes that may be entering surface or ground water. For this reason alone, the results of this part of the study are, at best, suggestive.

Transport and Exposure

The transport models used in the studies generally appear to be congruent with the study objectives. The air transport modeling addresses the concerns of the Subcommittee in its July 30, 1986 letter, although better

documentation of this modeling is needed. There is a factor of two uncertainty on point source air emissions and another uncertainty of a factor of two in dispersion modeling. The current modeling efforts do not address these potential uncertainties, although "worst case" scenarios should recognize their existence. Drinking water exposure was estimated by assuming that individuals consume two liters of the water delivered to their neighborhoods. Similar assumptions are often made in risk assessments. The surface water and hazardous waste studies are greatly hampered by a lack of data, making large assumptions necessary to estimate exposure to toxics.

Health Effects

The study evaluated 20 known or suspected cancer causing chemicals from the West Virginia APCC inventory of more than 450 compounds. The Subcommittee concludes that the current study provides useful information on health effects from cancer and environmental loadings of these 20 compounds. After finalizing the current study, EPA should conduct additional efforts that include:

- Using the APCC inventory and information on toxicity to evaluate the potential health effects of some of the remaining compounds. Of the remaining 430 or so compounds, relatively few merit further attention, but EPA and APCC should work together to identify compounds that need additional evaluation. These should be identified by defining the set of those compounds to which some exposure may be likely at known toxic levels.

- Broadening the health endpoints of concern to include non-cancer and acute effects. Concern about the potential effects from acute releases is strong within the community; hence, some priority should be given to addressing this issue. The methodologies used to address these endpoints require further development, particularly in estimating the effects of accidential releases. Some fault-tree or alternative analysis should be designed to address this possibility. Experts from other groups within the EPA should be enlisted in this effort.

- Incorporating frequency plots of pollutant concentrations versus time, in addition to stating average pollutant concentrations.

- Assessing the conversion of reference doses from the ingestion to the inhalation pathway, where reference dose information for the inhalation pathway is not available.

- Evaluating whether to develop or use biological markers for health assessment.

- Comparing risks from high mass emissions of pollutants with low toxicity, with low mass emissions of pollutants with high toxicity as a means to identify priority risk management needs.

- o Exploring other potentially useful sources of data for compounds of concern, including monographs prepared by the International Agency for Research on Cancer, Health Effects Profiles developed by the Office of Research and Development, Reportable Quantities for hazardous compounds and gaining access to information through the community right-to-know provision of Superfund.

Communication of Risks

The Subcommittee encourages EPA to continue its efforts of working with officials and citizens of the Kanawha Valley to update them on the sources and magnitude of risks they experience. In particular, EPA should seek to further improve its presentation of technical information to better enable lay persons to understand the results of technical analyses and to ensure it is understood that the risk numbers reflect upper bound estimates. Clarification of the latter issue is also needed in the executive summary of the study.

It is important for citizens, scientists and public officials to understand that the principal value of the Kanawha Valley study is as a screening study of airborne carcinogens. As the study acknowledges, a screening study should strive to ensure that all potential risks are identified even at the expense of calling attention to risks that subsequent analysis may not confirm, or will be less than indicated in the screening study. Accordingly, assumptions in screening studies are conservative in nature; assumptions should be avoided that might cause potential risks to be ignored. Within the stated scope of the study, conservative assumptions are made; for example, individuals are assumed to be exposed continuously to ambient outdoor levels of industrially emitted toxics and upper bound risk estimates are given. There are a few instances, however, where the study did not rigorously pursue conservative assumptions. These include potential uncertainties or omissions in the emissions inventory. The study suggests that point estimates could be too small (or too large) by a factor of two. For fugitive emissions it could be greater. It is important that these uncertainties and their likely direction be clearly articulated in the report along with a discussion about whether additional scenarios are necessary to consider these uncertainties.

In addition, the air quality models are equally likely to under-and-over predict ambient concentrations. The biases of the models are fairly predictable. Exposures are likely underestimated at the peaks of ridges where the river turns and when overlapping models were not used. On the other hand, the use of the Box model probably overpredicts exposure in some neighborhoods on the Valley floor, which are not adjacent to emissions sources. Although it is to the study's credit to have implemented two different modeling approaches to estimate exposure, further discussion in the report is merited on the potential model biases and on their implications for the risk estimates.

ADDITIONAL RECOMMENDATIONS TO THE AGENCY

A. Current Report

1. The technical assumptions for the underlying transport models should be documented and made accessible to readers of the report.

2. Given the comprehensive nature of the airborne toxic risk assessment in contrast to the rudimentary nature of the other three studies, it may be desirable to more clearly separate the air toxic studies from the others; moreover, the various studies are undertaken for differently defined geographic areas.

3. The risk estimate bounds are probably more clearly defined than in most similar documents; nevertheless, further clarification may be necessary. Cases could be presented as <X rather than the number X; attribution of cases/risk bounds by categories (industrial sources, fugitive vs. point emissions) should be more carefully qualified. Moreover, the conservatism of total case estimates is likely to be greater than estimates attributed to a single substance because of the joint probability that all substances require conservative assumptions is lower than that for a single substance.

4. Parts of Appendix C might be moved to the body of the report.

B. Future Work

1. A major public health concern among the residents of the Kanawha Valley is the risk associated with sudden accidental releases of airborne toxics. There is an urgent need to address this issue. Moreover, the current study addresses chronic health exposures, which are only one component of the many public health concerns in the Valley. A simple first step is to obtain some index of the toxicity of the remaining compounds. Information sources such as Health Effects Profiles, monographs of the International Agency for Research on Cancer, Reportable Quantities and data obtained from community right-to-know efforts, should be used. The preparation of exposure analyses will be more difficult as potential exposures to various lengths of time, including acute exposures, are estimated. Methodological help should be sought from other parts of the Agency.

2. The West Virginia Air Pollution Control Commission has developed an air emissions inventory, but the inventory represents an approximation of emissions from stationary sources. For the chemicals of the greatest public health concern, officials should undertake further monitoring to help validate the inventory. Where discrepancies arise, additional efforts will be warranted to more accurately determine sources and emissions levels.

3. The hazardous waste data considered are very limited. CERCLA requirements can perhaps provide some useful information. Other parts of EPA should be enlisted to improve the source inventory for these data. Analysis of historical operations and land use may also be useful to characterize the types of chemicals in waste sites. The fundamental approach to consider risk from hazardous waste should be replaced, however, by one that examines specific waste sites.

4. Increased monitoring data can aid the analysis of drinking water, surface water, and ground water. For chemicals of concern in the Valley, such efforts should be instituted to help ensure that no major problems are overlooked.

5. Health surveys and measurement of biological markers could provide some validation of the estimated health profile of the Valley. Such efforts will not, however, be useful when incremental risk estimates are small.

Attachment B

U. S. ENVIRONMENTAL PROTECTION AGENCY

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APPENDIX D

METHODLOGY FOR EVALUATING HEALTH RISKS: A GENERAL OVERVIEW

EPA is charged with controlling the levels of exposure to thousands of chemicals in the environment. Decisions about which chemicals to control are based, in large part, upon the population risks associated with exposure to those chemicals. This is because we want to control first those problems that pose the greatest and most controllable risks, and because the amount of resources we will apply to a problem should bear some relationship to the health threat it presents.

Population risks in the simplest sense, are a function of two basic factors: hazard and exposure. To cause a risk, a chemical has to be both toxic (present an intrinsic hazard), and be present in the human environment at some significant level (provide opportunity for significant human exposure). Risk assessment interprets the evidence on these two points, judging the probability of whether or not an adverse effect may occur.

To judge whether a chemical may cause a particular health effect, EPA must first consider the inherent toxicity of the chemical. In practice, this has been done in two quite different ways, depending on whether or not the chemical has been identified as a carcinogen, a substance that may cause cancer in animals or people. Toxicological research has led the Agency to assume that the exposure to any amount of a carcinogen is associated with the possibility, however small, that some person in the exposed population may develop cancer.

For many other health effects, in contrast, the Agency assumes that there is some level of exposure that causes virtually no harm. This level of exposure is called a "threshold." In almost all people, exposures below the threshold should not cause adverse health effects, while exposures above the threshold may lead to such effects in some exposed ivdividuals.

For each of these groups of chemicals, the Agency has adopted a standard process for estimating how potent a substance might be. For non-carcinogens, the Agency uses the concept of the threshold to determine Reference Doses, or RfDs (previously called ADIs).

For carcinogens, the EPA Cancer Assessment Group (CAG) is responsible for assessing dose-response relationships from exposure to carcinogens. The problem is that most of the information that we have about the effects of carcinogens relates to high doses, in animal studies or human occupational exposures. Alsmost all environmental exposures occur at much lower levels. For years, the scientific community has struggled with the problem of extrapolating effects at low doses from information about high doses. Also, for many substances, the only information that we have is from animal studies. Here a further uncertainty arises because scientists are uncertain about how to apply quantitative information from animal experiments to predict the effects of carcinogens on people.

CAG must make assumptions based on the best available scientific evidence. It uses mathematical models that are based on scientifc understanding of biochemical mechanisms to "fill in" the missing lowdose data. Intrinsically, the assumptions and application of such models in developing estimates of low dose effects results in some uncertainty. In general, when making a choice among such assumptions, concern for the public health drives Agency policy to select the one that generally turns out to be "conservative" or pessimistic, i.e., the one most protective of human health. The Agency, therefore, has chosen to define the CAG scores as a "plaisible upper bound." That is, if the application of the so-called "CAG numbers" predicts, say, three cancers from a certain exposure, we state that it is not likely to be more than that, but may be much less, or even zero.

CAG numbers undergo substantial review within the Agency. In a similar fashion, the Agency has established a review process for RFDs. CAG numbers are often reviewed further by EPA's Science Advisory Board. In this process they are often modified as a result of addiitional research findings. Over time, CAG's and RFDs come to represent a consensus about what EPA thinks about the tendency of various substances to produce adverse health effects. As such they are used as the scientific underpinnings of regulatory policy making, and although dose-response and risk assessment are not perfect, they represent the best quantitative decision aid we have for discriminating among risks.

The Kanawha Valley study uses a risk assessment screening methodology to evaluate and compare, in a very limited fashion, the potential health risks from exposure to a limited set of pollutants. The purpose of this methodology is to permit a comparison of one risk with another and to provide a general sense of the risk a substance may present. This methodology does not make a definitive statement concerning the absolute risk posed by a particular pollutant, source or exposure pathway.

Risk to an individual is defined as the increased probability that an individual exposed to one or more pollutants will experience a particular adverse health effect during his or her lifetime because of that exposure. Several measures of carcinogenic risk are used in this analyses, including estimated risk to an individual, and incidence, defined as the increased number of cases projected in a given population.

This risk screening methodology involves both a qualitative and quantitative assessment of the potential carcinogenicity of the selected pollutants. As a screening study, this analysis employs both types of pollutants. The Carcinogen Assessment Group (CAG) reviews the evidence of carcinogenicity for selected pollutants and classifies pollutants as human carcinogens (Group A), probable human carcinogens (Group B), possible human carcinogens (Group C), not classified as carcinogens due to inadequate evidence (Group D), and not carcinogenic to humans (Group E).

For those chemicals in group A,B, and C, CAG provides quantitative, conservatives upper-bound estimates of carcinogenic unit risk factors. A unit risk factor allows the calculation of the estimate individual risk posed by exposure by a chemical given certain exposure assumptions. To calculate individual risks, a chemical's unit risk factor is multiplied by the estimated concentration of the pollutant.

Individual Lifetime Risk = Unit risk factor $(uq/m^3)^{-1}$ x Concentration (ug/m^3)

The concentration is simply the predicted concentration of the chemical in the ambient air. For this analysis, we use the long-term average concentration predicted by the model. The unit risk factor which provides a measure of the chemical potency for this analysis assumes that an average person weighs 70 Kg and breathes 20 m^3 of air each day.

SAMPLE CALCULATION
POSSIBLE RISK OF CANCER ASSOCIATED WITH INHALATION OF CHLOROFORM IN AMBIENT AIR

Estimated Average Individual Lifetime Risk	=	Unit Risk Factor x Average Concentration
Cancer Unit Risk Factor for the Inhalation of Chloroform	=	2.3×10^{-5} $(ug/m^3)^{-1}$
Concentration	=	For this Example assume 1 ug/m^3 of chloroform in the ambient air
Estimated Average Individual Lifetime Risk	=	2.3×10^{-5} $(ug/m^3)^{-1} \times 1$ (ug/m^3)
	=	2.3×10^{-5}

This estimated average individual risk, based on an upper bound unit risk factor, indicates that, concervatively, there is approximately a 2 in one hundred thousand increased chance of developing cancer over a 70 year lifetime of constant exposure to this concentration of chloroform. The actual risk could be considerably lower.

Incidence, another measure of risk, is the probable number of cancer cases expected when a population is exposed. Incidence is calculated by multiplying individual risk by the population number and can be annualized by dividing by 70 to obtain cases per year.

APPENDIX E

RISK ASSESSMENT FOR NON-CANCER EFFECTS

REFERENCE DOSE (RfD): DESCRIPTION AND USE IN HEALTH RISK ASSESSMENTS

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I. INTRODUCTION

This concept paper describes the U.S. Environmental Protection Agency's principal approach to and rationale for assessing risks for health effects other than cancer and gene mutations from chronic chemical exposure. By outlining principles and concepts that guide EPA risk assessment for such systemic* effects, the report complements the new risk assessment guidelines, which describe the Agency's approach to risk assessment in other areas (carcinogenicity, mutagenicity, developmental toxicity, exposure, and chemical mixtures.)

A. Background

Chemicals that give rise to toxic end points other than cancer and gene mutations are often referred to as "systemic toxicants" because of their effects on the function of various organ systems. It should be noted, however, that chemicals which cause cancer and gene mutations also commonly evoke other toxic effects (systemic toxicity). Generally, based on our understanding of homeostatic and adaptive mechanisms, systemic toxicity is treated as if there is an identifiable exposure threshold (both for the individual and for the population) below which effects are not observable. This characteristic distinguishes systemic end points from carcinogenic and mutagenic end points, which are often treated as nonthreshold processes.

Systemic effects have traditionally been evaluated in terms of concepts such as "acceptable daily intake" and "margin of safety." The scientific community has identified certain limits on some of these approaches, and these limits have been borne out in EPA's experience. Nonetheless, EPA is called upon to apply these concepts in making and explaining decisions about the significance for human health of certain chemicals in the environment.

To meet these needs, the RfD Work Group has drawn on traditional concepts, as well as on recommendations in the 1983 National Academy of Sciences (NAS) report on risk assessment, to more fully articulate the use of noncancer, nonmutagenic experimental data in reaching decisions on the significance of exposures to chemicals. In the process, the Agency has coined new terminology to clarify and distinguish between aspects of risk assessment and risk management. EPA has tested and implemented these innovations in developing consistent information for several recent regulatory needs, for instance under RCRA.

B. Overview

This Appendix consists of four parts in addition to this introduction. In Section II, much of the traditional information on assessing risks of systemic toxicity is presented, with the focus on the concepts of "acceptable daily intake (ADI)" and "safety factor (SF)." Issues associated with these approaches are identified and discussed.

In Section III, the Agency's approach to assessing the risks of systemic toxicity is presented in the context of the NAS scheme of risk assessment and risk management in regulatory decision-making. This approach includes recasting earlier ADI and SF concepts into the less value-laden terms "reference dose (RfD)" and "uncertainty factor (UF)." A new term, "margin of exposure,"** as utilized in the EPA regulatory context, is introduced to avoid some of the issues associated with the traditional approach.

Section IV examines how these new concepts can be applied in reaching risk management decisions, while Section V briefly mentions some of the additional approaches the Agency is using and exploring to address this issue. Section VI provides a sample RfD calculation.

---

*In this document the term "systemic" refers to an effect other than carcinogenicity or mutagenicity induced by a toxic chemical.

**In this Appendix, the ratio of the NOAEL to the estimated exposure (often referred to as "margin of safety") is referred to as the "margin of exposure (MOE)" in order to avoid confusion with the original use of the term "margin of safety" in pharmacology (i.e., the ratio of the toxic dose to the theraputic dose) and to avoid the use of the value-laden term "safety."

II. TRADITIONAL APPROACH TO ASSESSING SYSTEMIC (NONCARCINOGENIC) TOXICITY

The Agency's approach to assessing the risks associated with systemic toxicity is different from that for the risks associated with carcinogenicity. This is because different mechanisms of action are thought to be involved in the two cases. In the case of carcinogens, the Agency assumes that a small number of molecular events can evoke changes in a single cell that can lead to uncontrolled cellular proliferation. This mechanism for carcinogenesis is referred to as "nonthreshold," since there is essentially no level of exposure for such a chemical that does not pose a small, but finite, probability of generating a carcinogenic response. In the case of systemic toxicity, organic homeostatic, compensating, and adaptive mechanisms exist that must be overcome before the toxic end point is manifested. For example, there could be a large number of cells performing the same or similar function whose population must be significantly depleted before the effect is seen.

The threshold concept is important in the regulatory context. The individual threshold hypothesis holds that a range of exposures from zero to some finite value can be tolerated by the organism with essentially no chance of expression of the toxic effect. Further, it is often prudent to focus on the most sensitive members of the population; therefore, regulatory efforts are generally made to keep exposures below the population threshold, which is defined as the lowest of the thresholds of the individuals within a population.

A. The Traditional Approach

In many cases, risk decisions on systemic toxicity have been made by the Agency using the concept of the "acceptable daily intake (ADI)." This quantity is derived by dividing the appropriate "no-observed-adverse-effect level (NOAEL)" by a "safety factor (SF)" as follows:*

$$\text{ADI (human dose)} = \text{NOAEL (experimental dose)} / \text{SF} \qquad (1)$$

The ADI is often viewed as the amount of a chemical to which one can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect. Often, the ADI has been used as a tool in reaching risk management decisions; e.g., establishing allowable levels of contaminants in foodstuffs and water.

Once the critical study demonstrating the toxic effect of concern has been identified, the selection of the NOAEL derives from an essentially objective, scientific examination of the data available on the chemical in question.

Generally, the SF consists of multiples of 10, each factor representing a specific area of uncertainty inherent in the available data. For example, an SF may be developed by taking into account the expected differences in responsiveness between humans and animals in prolonged exposure studies; i.e., a 10- fold factor. In addition, a second factor of 10 may be introduced to account for variability among individuals within the human population. For many chemicals, the resultant SF of 100 has been judged to be appropriate. For other chemicals, with a less complete data base (e.g., those for which only the results of subchronic studies are available), an additional factor of 10 (leading to an SF of 1,000) might be judged to be more appropriate. On the other hand, for some chemicals, based on well-characterized responses in sensitive humans (e.g., effect of fluoride on human teeth), an SF as small as 1 might be selected.

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*A NOAEL is an experimentally determined dose at which there was no statistically or biologically significant indication of the toxic effect of concern. In an experiment with several NOAELs, the regulatory focus is normally on the highest one, leading to the common usage of the term NOAEL as the highest experimentally determined dose without statistical or adverse biological effect. In some treatments, the NOAEL for the critical toxic effect is simply referred to as the NOEL. This latter term, however, invites ambiguity in that there may be observable effects which are not of toxicologic significance; i.e., they are not "adverse." In order to be explicit, this Appendix uses the term NOAEL and it refers to the highest NOAEL in an experiment. Further, in cases in which a NOAEL has not been demonstrated experimentally, the formulation calls for use of the "lowest-observed-adverse-effect level (LOAEL)." In order to focus on the major concepts, however, we will use NOAEL as a general example.

While the original selection of SFs appears to have been rather arbitrary (Lehman and Fitzhugh, 1954)*, subsequent analysis of data as reviewed by Dourson and Stara (1983) lends theoretical (and in some instances experimental) support for their selection. Further, some scientists, but not all, within the EPA interpret the absence of widespread effects in the exposed human populations as evidence of the adequacy of the SFs traditionally employed.

B. Some Difficulties in Utilizing the Traditional Approach

1. *Scientific Issues*

While the traditional approach has performed well over the years and the Agency has sought to be consistent in its application, observers have identified scientific shortcomings of the approach. Examples include the following:

- By focusing on the NOAEL, information on the shape of the dose-response curve is ignored. Such data could be important in estimating levels of concern for public safety.
- As scientific knowledge is increased and the correlation of precursor effects (e.g., enzyme induction) with frank toxicity becomes known, questions about the selection of the appropriate "adverse effect" arise.
- Guidelines have not been developed to take into account the fact that some studies have used larger numbers of animals and, hence, are generally more reliable than other studies.

These and other "generic issues" are not susceptible to immediate resolution, because the data base needed is not yet sufficiently developed or analyzed. Therefore, these issues are beyond the scope of this Appendix. However, the Agency has established a work group to consider them.

2. Management-related Issues

a. The use of the term "safety factor"

The term "safety factor" suggests, perhaps inadvertently, the notion of absolute safety, i.e., absence of risk. While there is a conceptual basis for believing in the existence of a threshold and "absolute safety" associated with certain chemicals, in the majority of cases a firm experimental basis for this notion does not exist.

b. The implication that any exposure in excess of the ADI is "unacceptable" and that any exposure less than the ADI is "acceptable" or "safe"

In practice, the ADI is viewed by many as an "acceptable" level of exposure, and, by inference, any exposure greater than the ADI is seen as "unacceptable." This strict demarcation between what is "acceptable" and what is "unacceptable" is contrary to the views of most toxicologists, who typically interpret the ADI as a relatively crude estimate of a level of chronic exposure not likely to result in adverse effects to humans. The ADI is generally viewed as a "soft" estimate, whose bounds of uncertainty can span an order of magnitude. That is, within reasonable limits, while exposures somewhat higher than the ADI are associated with increased probability of adverse effects, that probability is not a certainty. Similarly, while the ADI is seen as a level at which the probability of adverse effects is low, the absence of risk to all people cannot be assured at this level.

c. Possible limitations imposed on risk management decisions

Awareness of the "softness" of the ADI estimate (see b. above) argues for careful case-by-case consideration of the implications of the toxicological analysis as it applies to any particular situation. To the degree that ADIs generated by the traditional approach are the determining factors in risk

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*Lehman, A.J. and Fitzhugh, O.G. (1954). Association of Food Drug Officials. USQ Bulletin 18:33-35.

management decisions, they can take on a significance beyond that intended by the toxicologist or merited by the underlying scientific support.

Further, in administering risk/benefit or cost/benefit statutes, the risk manager is required to consider factors other than risk (e.g., estimated exposures compared to the ADI) in reaching a decision. The ADI is only one factor in a management decision and should not prevent the risk manager from weighing the full range of factors.

d. Development of different ADIs by different programs

In addition to occasionally selecting different critical toxic effects, Agency scientists have reflected their best scientific judgments in the final ADI by adopting factors different from the standard factors listed in Table A-1. For example, if the toxic end point for a chemical in experimental animals is the same as that which has been established for a related chemical in humans at similar doses, one could argue for an SF of less than the traditional 100. On the other hand, if the total toxicologic data base is incomplete, one could argue that an additional SF should be included, both as a matter of prudent public policy and as an incentive to others to generate the appropriate data.

Such practices, as employed by a number of scientists in different programs, exercising their best scientific judgment, have in many cases resulted in different ADIs for the same chemical. The fact that different ADIs were generated (e.g., by adopting different SFs) can be a source of considerable confusion when the ADIs are applied in risk management decisionmaking (see c. above). For example, although they generally agree on the experimental data base for 2,3,7,8-TCDD, regulatory agencies within the United States and around the world have generated different ADIs by selecting different "safety factors"; specifically, 1000, 500, 250, and 100. These different ADIs have been used to justify different regulatory decisions. The existence of different ADIs need not imply that any of them is more "wrong"--or "right"--than the rest. It is more nearly a reflection of the honest difference in scientific judgment.

These differences, which may reflect differences in the interpretation of the scientific data, can also be characterized as differences in the management of the risk. As a result, scientists may be inappropriately impugned,and/or perfectly justifiable risk management decisions may be tainted by charges of "tampering with the science." This unfortunate state of affairs arises, at least in part, from treating the ADI as an absolute measure of safety.

III. EPA ASSESSMENT OF RISKS ASSOCIATED WITH SYSTEMIC TOXICITY

In 1983, the National Academy of Sciences published a report which discusses the conceptual framework within which regulatory decisions on toxic chemicals are made; see Figure A-1. The determination of the presence of risk and its potential magnitude is made during the risk assessment process, which consists of hazard identification, dose-response assessment, exposure assessment, and risk characterization. Having been apprised by the risk assessor that a potential risk exists, the risk manager answers the question: "What, if anything, are we going to do about it?"

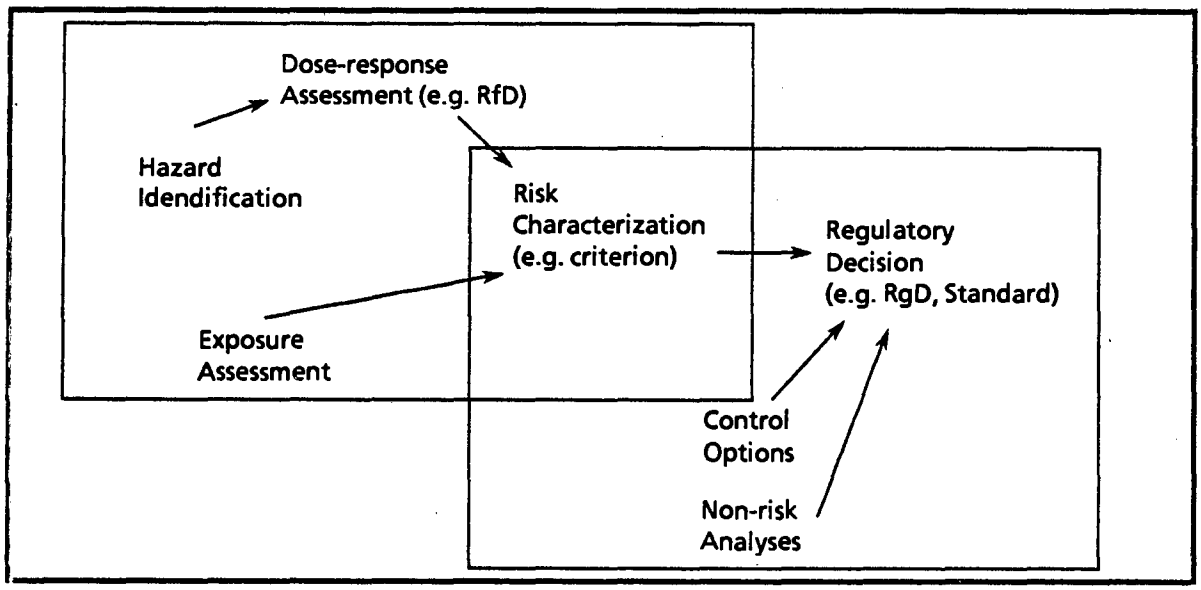
A. Hazard Identification

1. Evidence

a. Type of effect

Exposure to a given chemical, depending on the dose employed, may result in a variety of toxic effects. These may range from gross effects, such as death, to more subtle biochemical, physiologic, or pathologic changes. The risk assessor considers each of the toxic end points from all studies evaluated in assessing the risk posed by a chemical, although primary attention usually is given to the effect exhibiting the lowest NOAEL, often referred to as the critical effect. For chemicals with a limited data base, there may be a need for more toxicity testing.

FIGURE A-1



b. Principal studies

Principal studies are those that contribute most significantly to the qualitative assessment of whether or not a particular chemical is potentially a systemic toxicant in humans. In addition, they may be used in the quantitative dose-response assessment phase of the risk assessment. These studies are of two types:

(1) Human studies

Human data are often useful in qualitatively establishing the presence of an adverse effect in exposed human populations. Further, when there is information on the exposure level associated with an appropriate end point, epidemiologic studies can also provide the basis for a quantitative dose-response assessment. Use of these latter data avoids the necessity of extrapolating from animals to humans, and therefore, human studies, when available, are given first priority, with animal toxicity studies serving to complement them.

In epidemiologic studies, confounding factors that are recognized can be controlled and measured, within limits. Case reports and acute exposures resulting in severe effects provide support for the choice of critical toxic effect, but they are often of limited utility in establishing a quantitative relationship between environmental exposures and anticipated effects. Available human studies on ingestion are usually of this nature. Cohort studies and clinical studies may contain exposure-response information that can be used in estimating effect levels, but the method of establishing exposure must be evaluated for validity and applicability.

(2) Animal studies

Usually, the data base on a given chemical lacks appropriate information on effects in humans. In such cases, the principal studies are drawn from experiments conducted on non-human mammals, most often the rat, mouse, rabbit, guinea pig, hamster, dog, or monkey.

c. Supporting studies

Supporting studies include information from a wide variety of sources. For example, metabolic and other pharmacokinetic studies can provide insights into the mechanism of action of a particular compound. By comparing the metabolism of the compound exhibiting the toxic effect in the animal

with the metabolism found in humans, some light may be cast on the potential for the toxic manifestation in humans or for estimating the equitoxic dose in humans.

Similarly, *in vitro* studies can provide insights into the compound's potential for biological activity, although a definite connection to the human experience cannot be drawn. Under certain circumstances, consideration of structure-activity relationships between the chemical under test and the effects of structurally related agents can provide a clue to the biological activity of the former.

At the present time, these data are supportive, not definitive, in assessing risk. However, there is focused activity aimed at developing more reliable *in vitro* tests to minimize the need for live-animal testing. Similarly, there is increased emphasis on generating mechanism-of-action and pharmacokinetic information as a means of increasing the fundamental understanding of toxic processes in humans and nonhumans. It is expected that in the future these considerations will play a larger role in our determination of toxicity of chemicals.

d. Route of exposure

The Agency often approaches the investigation of a chemical with a particular route of exposure in mind; e.g., an oral exposure for a drinking water contaminant or a residue in food. Although the route of exposure is oral in both cases, specific considerations may differ. For example, the bioavailability of the chemical administered in food may differ from that when administered in water or inhaled. Usually, the toxicologic data base on the compound does not include detailed testing on all possible routes of administration.

In general, it is the Agency's view that the potential for toxicity manifested by one route of exposure is relevant to any other route of exposure, unless convincing evidence exists to the contrary. Consideration is always given to potential differences in absorption or metabolism resulting from different routes of exposure, and whenever appropriate data (e.g., comparative metabolism studies) are available, the quantitative impacts of these differences on the risk assessment are fully delineated.

e. Length of exposure

The Agency is concerned about the potential toxic effects in humans associated with all possible exposures to chemicals. The magnitude, frequency, and duration of exposure may vary considerably in different situations. Animal studies are conducted using a variety of exposure durations (e.g., acute, subchronic, and chronic) and schedules (e.g., single, intermittent, or continuous dosing). Information from all of these studies is useful in the hazard identification phase of risk assessment. For example, overt neurological problems identified in high-dose acute studies tend to reinforce the observation of subtle neurological changes seen in a low-dose chronic study. Special concern exists for low-dose, chronic exposures, however, since such exposures can elicit effects absent in higher-dose, shorter exposures, through mechanisms such as accumulation of toxicants in the organisms.

f. Quality of the study

Evaluation of individual studies in humans and animals requires the consideration of several factors associated with a study's hypothesis, design, execution, and interpretation. An ideal study addresses a clearly delineated hypothesis, follows a carefully prescribed protocol, and includes sufficient subsequent analysis to support its conclusions convincingly.

In evaluating the results from such studies, consideration is given to many other factors, including chemical characterization of the compound(s) under study, the type of test species, similarities and differences between the test species and humans (e.g., chemical absorption and metabolism), the number of individuals in the study groups, the number of study groups, the spacing and choice of dose levels tested, the types of observations and methods of analysis, the nature of pathologic changes, the alteration in metabolic responses, the sex and age of test animals, and the route and duration of exposure.

2. Weight-of-Evidence Determination

As the culmination of the hazard identification step, a discussion of the weight-of-evidence summarizes the highlights of the information gleaned from the entire range of principal and

supporting studies. Emphasis in the analysis is given to examining the results from different studies to determine the extent to which a consistent, plausible picture of toxicity emerges. For example, the following factors add to the weight of the evidence that the chemical poses a hazard to humans: similar results in replicated animal studies by different investigators; similar effects across sex, strain, species, and route of exposure; clear evidence of a dose-response relationship; a plausible relation between data on metabolism, postulated mechanism-of-action, and the effect of concern; similar toxicity exhibited by structurally related compounds; and some link between the chemical and evidence of the effect of concern in humans. The greater the weight-of-evidence, the greater one's confidence in the conclusions drawn.

B. Dose-Response Assessment

1. Concepts and Problems

Empirical observation generally reveals that as the dosage of a toxicant is increased, the toxic response (in terms of severity and/or incidence of effect) also increases. This dose-response relationship is well-founded in the theory and practice of toxicology and pharmacology. Such behavior is observed in the following instances: in quantal responses, in which the proportion of responding individuals in a population increases with dose; in graded responses, in which the severity of the toxic response within an individual increases with dose; and in continuous responses, in which changes in a biological parameter (e.g., body or organ weight) vary with dose.

However, in evaluating a dose-response relationship, certain difficulties arise. For example, one must decide on the critical end point to measure as the "response." One must also decide on the correct measure of "dose." In addition to the interspecies extrapolation aspects of the question of the appropriate units for dose, the more fundamental question of administered dose versus absorbed dose versus target organ dose should be considered. These questions are the subject of much current research.

2. Selection of the Critical Data

a. Critical study

Often animal data are selected as the governing information for quantitative risk assessments, since available human data are generally insufficient for this purpose. These animal studies typically reflect situations in which exposure to the toxicant has been carefully controlled and the problems of heterogeneity of the exposed population and concurrent exposures to other toxicants have been minimized. In evaluating animal data, a series of professional judgments are made that involve, among others, consideration of the scientific quality of the studies. Presented with data from several animal studies, the risk assessor first seeks to identify the animal model that is most relevant to humans, based on the most defensible biological rationale, for instance using comparative pharmacokinetic data. In the absence of a clearly most relevant species, however, the most sensitive species (i.e., the species showing a toxic effect at the lowest admininistered dose) is adopted as a matter of scientific policy at EPA, since no assurance exists that humans are not innately more sensitive than any species tested. This selection process is made more difficult if animal tests have been conducted using different routes of exposure, particularly if the routes are different from those involved in the human situation under investigation.

In any event, the use of data from carefully controlled studies of genetically homogeneous animals inescapably confronts the risk assessor with the problems of extrapolating between species and the need to account for human heterogeneity and concurrent human exposures to other chemicals, which may modify the human risk.

While there is usually a lack of well-controlled cohort studies that investigate non-cancer end points and human exposure to chemicals of interest, in some cases human data may be selected as the critical data (e.g., in cases of cholinesterase inhibition). Risk assessments based on human data have the advantage of avoiding the problems inherent in interspecies extrapolation. In many instances, use of such studies, as is the case with the animal investigations, involves extrapolation from relatively high doses (such as those found in occupational settings) to the low doses found in the environmental situations to which the general population is more likely to be exposed. In some

cases, a well-designed and well-conducted epidemiologic study that shows no association between known exposures and toxicity can be used to directly project an RfD (as has been done in the case of fluoride).

b. Critical data

In the simplest terms, an experimental exposure level is selected from the critical study that represents the highest level tested in which "no adverse effect" was demonstrated. This "no-observed-adverse-effect level" (NOAEL) is the key datum gleaned from the study of the dose-response relationship and, traditionally, is the primary basis for the scientific evaluation of the risk posed to humans by systemic toxicants. This approach is based on the assumption that if the critical toxic effect is prevented, then all toxic effects are prevented.

More formally, the NOAEL is defined in this discussion as the highest experimental dose of a chemical at which there is no statistically or biologically significant increase in frequency or severity of an adverse effect between individuals in an exposed group and those in its appropriate control. (See also discussion in the footnote on page A-4). As noted above, there may be sound professional differences of opinion in judging whether or not a particular response is adverse. In addition, the NOAEL is a function of the size of the population under study. Studies with a small number of subjects are less likely to detect low-dose effects than studies using larger numbers of subjects. Also, if the interval between doses in an experiment is large, it is possible that the experimentally determined NOAEL is lower than that which would be observed in a study using intervening doses.

c. Critical end point

A chemical may elicit more than one toxic effect (end point), even in one test animal, or in tests of the same or different duration (acute, subchronic, and chronic exposure studies). In general, NOAELs for these effects will differ. The critical end point used in the dose-response assessment is the one at the lowest NOAEL.

3. Reference Dose (RfD)

In response to many of the problems associated with ADIs and SFs, which were outlined in Section II, the concept of the "reference dose (RfD)" and "uncertainty factor (UF)" is recommended. The RfD is a benchmark dose operationally derived from the NOAEL by consistent application of generally order of magnitude uncertainty factors (UFs) that reflect various types of data used to estimate RfDs (for example, a valid chronic human NOAEL normally is divided by an UF of 10) and an additional modifying factor (MF), which is based on a professional judgment of the entire data base of the chemical.* See Table A-1.

The RfD is determined by use of the following equation:

$$RfD = NOAEL/(UF \times MF) \qquad (2)$$

which is the functional equivalent of Eq. (1). In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. The RfD is appropriately expressed in units of mg/kg-bw/day.

The RfD is useful as a reference point for gauging the potential effects of other doses. Usually, doses that are less than the RfD are not likely to be associated with any health risks, and are therefore less likely to be of regulatory concern. However, as the frequency of exposures exceeding the RfD increases, and as the size of the excess increases, the probability increases that adverse effects may be observed in a human population. Nonetheless, a clear conclusion cannot be categorically drawn that all doses below the RfD are "acceptable" and that all doses in excess of the RfD are "unacceptable."

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*"Uncertainty factor" is the new description applied to the term "safety factor" (see Page A-4). This new name is more descriptive in that these factors represent scientific uncertainties, and avoids the risk management connotation of "safety." The "modifying factor" can range from greater than zero to 10, and reflects qualitative professional judgements regarding scientific uncertainties not covered under the standard UF, such as the completeness of the overall data base and the number of animals in the study.

TABLE A-1.
GUIDELINES FOR THE USE OF UNCERTAINTY FACTORS IN DERIVING REFERENCE DOSE (RfD)

Standard Uncertainty Factors (UFs)

Use a 10-fold factor when extrapolating from valid experimental results from studies using prolonged exposure to average healthy humans. This factor is intended to account for the variation in sensitivity among the members of the human population. [10H]

Use an additional 10-fold factor when extrapolating from valid results of long-term studies on experimental animals when results of studies of human exposure are not available or are inadequate. This factor is intended to account for the uncertainty in extrapolating animal data to the case of humans. [10A]

Use an additional 10-fold factor when extrapolating from less than chronic results on experimental animals when there are no useful long-term human data. This factor is intended to account for the uncertainty in extrapolating from less than chronic NOAELs to chronic NOAELs. [10S]

Use an additional 10-fold factor when deriving a RfD from a LOAEL, instead of a NOAEL. This factor is intended to account for the uncertainty in extrapolating from LOAELs to NOAELs. [10L]

Modifying Factor (MF)

Use professional judgment to determine another uncertainty factor (MF) which is greater than zero and less than or equal to 10. The magnitude of the MF depends upon the professional assessment of scientific uncertainties of the study and database not explicitly treated above; e.g., the completeness of the overall data base and the number of species tested. The default value for the MF is 1.

SOURCE: Adapted from Dourson, M.L.; and Stara, J.F. (1983) Regulatory Toxicology and Pharmacology 3:224-238.

(This is a consequence of the inability of either the traditional or the RfD approach to completely address the question of dose-response extrapolation.)

The Agency is attempting to standardize its approach to determining RfDs. The RfD Work Group has developed a systematic approach to summarizing its evaluations, conclusions, and reservations regarding RfDs in a "cover sheet" of a few pages in length. The cover sheet includes a statement on the confidence the evaluators have in the stability of the RfD: high, medium, or low. High confidence indicates that the RfD is unlikely to change in the future because there is consistency among the toxic responses observed in different sexes, species, study designs, or in dose-response relationships, or the reasons for differences, if any, are well understood. Often, high confidence is given to RfDs that are based on human data for the exposure route of concern, because in such cases the problems of interspecies extrapolation are avoided. Low confidence indicates that the RfD may be especially vulnerable to change if additional chronic toxicity data are published on the chemical, because the data supporting the estimation of the RfD are of limited quality and/or quantity.

C. Exposure Assessment

The third step in the risk assessment process focuses on exposure issues. For a full discussion of exposure assessment, the reader is referred to EPA's recently published guidelines on the subject (51 *Federal Register* 34042-34054, Sept. 24, 1986). There is no substantive difference in the conceptual approach to exposure assessment in the case of systemic toxicants and of carcinogens.

In brief, the exposure assessment includes consideration of the populations exposed and the magnitude, frequency, duration and routes of exposure, as well as evaluation of the nature of the exposed populations.

D. Risk Characterization

Risk characterization is the final step in the risk assessment process and the first step in the risk management process. Its purpose is to present to the risk manager a synopsis and synthesis of all the data that contribute to a conclusion on the risk, including:

- The qualitative ("weight-of-evidence") conclusions about the likelihood that the chemical may pose a hazard to human health.
- A discussion of dose-response and how this information, through the use of particular uncertainty and modifying factors, was used to determine the RfD.
- Data such as the shapes and slopes of the dose-response curves for the various toxic end points, toxicodynamics (absorption and metabolism), structure-activity correlations, and the nature and severity of the observed effect. These data should be clearly discussed by the risk assessor, since they may influence the final decision of the risk manager (see below).
- The estimates of exposure, the nature of the exposure, and the number and types of people exposed, together with a discussion of the uncertainties involved.
- A discussion of the sources of uncertainty, major assumptions, areas of scientific judgment, and, to the extent possible, estimates of the uncertainties embodied in the assessment.

In the risk characterization process, comparison is made between the RfD and the estimated (calculated or measured) exposure dose (EED), which should consider exposure by all sources and routes of exposure. The risk assessment should contain a discussion of the assumptions underlying the estimation of the RfD (nature of the critical end point, nature of other toxic end points, degree of confidence in the data base, etc.), and the degree of conservatism in its derivation. The assumptions used to derive the EED should also be discussed. If the EED is less than the RfD, the need for regulatory concern is likely to be small.

An alternative measure that may be useful to some risk managers is the "margin of exposure (MOE)" (see footnote on p. A-3), which is the magnitude by which the NOAEL of the critical toxic effect exceeds the estimated exposure dose (EED), where both are expressed in the same units:

$$\text{MOE} = \text{NOAEL (experimental dose)} / \text{EED (human dose)} \qquad (3)$$

In parallel to the statements above on EED and RfD, the risk assessment should contain a discussion of the assumptions underlying the estimates of the RfD and the degree of possible conservatism of the UF and MF. It can be noted that when the MOE is equal to or greater than UF x MF, the need for regulatory concern is likely to be small.

Section VI contains an example of the use of the concepts of NOAEL, UF, MF, RfD, and MOE.

IV. APPLICATION IN RISK MANAGEMENT

Once the risk characterization is completed, the focus turns to risk management. In reaching decisions, the risk manager must consider a number of risk factors, nonrisk factors, and regulatory options that influence the final judgment. It is generally useful to the risk manager to have information regarding the contribution to the RfD from various environmental media. Such information can provide insights that are helpful in choosing among available control options. However, in cases in which site-specific criteria are being considered, local exposures through various media can often be determined more accurately than exposure estimates based upon generic approaches. In such cases, the exposure assessor's role is particularly important. For instance, at a given site, consumption of fish may clearly dominate the local exposure routes, while, on a national basis, fish consumption may play a minor role compared to ingestion of treated crops.

RfDs should be apportioned by route of exposure. Where specific exposure analysis can be made, such apportionment is readily performed. If exposure information is not available, assumptions must be made concerning the relative contributions from different routes of exposure. At present, different EPA offices use assumptions that differ to some degree. These assumptions are being reviewed by an Agency risk assessment group.

As illustrated in Figure A-1, the risk manager utilizes the results of risk characterization, other technological factors, and nontechnical social and economic considerations in reaching a regulatory decision. Some of these factors include efficiency, timeliness, equity, administrative simplicity, consistency, public acceptability, technological feasibility, and legislative mandate.

Because of the way these risk management factors may impact different cases, consistent--but not necessarily identical--risk management decisions must be made on a case-by-case basis. For example, the Clean Water Act calls for decisions with "an ample margin of safety"; the Federal Insecticide, Fungicide and Rodenticide Act (FIFRA) calls for "an ample margin of safety," taking benefits into account; and the Safe Drinking Water Act (SDWA) calls for standards that protect the public "to the extent feasible." Consequently, it is entirely possible and appropriate that a chemical with a specific RfD may be regulated under different statutes and situations through the use of different "regulatory doses (RgDs)".

Expressed in general terms, after carefully considering the various risk and nonrisk factors, regulatory options, and statutory mandates in a given case (i), the risk manager decides upon the appropriate statutory alternatives to arrive at an "ample" or "adequate" margin of exposure [MOE(i)], thereby establishing the regulatory dose, RgD(i) (e.g., a tolerance under FIFRA or a maximum contaminant level under SDWA), applicable to that case:

$$RgD(i) = NOAEL / MOE(i) \qquad (4)$$

Note that, for the same chemical (with a single RfD), the risk manager(s) can develop different regulatory doses for different situations that may involve different exposures, available control options, alternative chemicals, benefits, and statutory mandates. Also note that comparing the RfD to a particular RgD(i) is equivalent to comparing the MOE(i) with the UF x MF:

$$RfD/RgD(i) = MOE(i)/UF \times MF \qquad (5)$$

In assessing the significance of a case in which the RgD is greater (or less) than the RfD, the risk manager should carefully consider the case-specific data laid out by the risk assessors, as discussed in in Section III. D. 4. In some cases this may require additional explanation and insight from the risk assessor. In any event, the risk manager has the responsibility to clearly articulate the reasoning leading to the final RgD decision.

V. OTHER DIRECTIONS

While the Agency is in the process of systematizing the approach outlined in this Appendix, risk assessment research for systemic toxicity is also being conducted along entirely separate lines. For example, the Office of Air Quality Planning and Standards is using probabilistic risk assessment procedures for criteria pollutants. This procedure characterizes the population at risk, and the likelihood of various effects occurring, through the use of available scientific literature and elicitation of expert judgment concerning dose-response relationships. The dose-response information is combined with exposure analysis modeling to generate population risk estimates for alternative standards. These procedures present the decisionmaker with ranges of risk estimates, and explicitly consider the uncertainties associated with both the toxicity and exposure information. The Office of Policy, Planning, and Evaluation is investigating similar procedures in order to balance health risk and cost. In addition, scientists in the Office of Research and Development have initiated a series of studies that should lead to future improvements in risk estimation. First, they are investigating the use of extrapolation models as well as the statistical variability of the NOAEL and underlying UFs as means of estimating RfDs. Second, they are exploring procedures for less-than-lifetime health risk assessment. Finally, they are working on ranking the severity of toxic effects as a way to further refine EPA's health risk assessments. While these procedures are promising, they cannot be expected at this time to serve as a foundation of a generalized health risk assessment for systemic toxicity in the Agency.

VI. HYPOTHETICAL, SIMPLIFIED EXAMPLE OF DETERMINING AND USING RfD

Suppose the Agency had a sound 90-day subchronic gavage study in rats with the following data:

A. Experimental Results

Dose (mg/kg-day)	Observation	Effect Level
0	Control - no adverse effects observed	--
1	No statistical or biological significant differences between treated and control animals	NOEL
5	2% decrease* in body weight gain (not considered to be of biological significance)	NOAEL
	Increased ratio of liver weight to body weight	
	Histopathology indistinguishable from controls	
	Elevated liver enzyme levels	
25	20% decrease* in body weight gain	LOAEL
	Increased* ratio of liver weight to body weight	
	Enlarged, fatty liver with vacuole formation	
	Increased* liver enzyme levels	

* = Statistically significant compared to controls.

B. Analysis

1. Determination of the Reference Dose (RfD)

a. From the NOAEL

UF = 10H x 10A x 10S = 1000

MF = 0.8, a subjective adjustment based on the fact that the experiment involved an astonishing 250 animals per dose group.

Therefore UF x MF = 800, so that

RfD = NOAEL/(UF x MF) = 5 mg/kg-day / 800 = 0.006 mg/kg-day

b. From the LOAEL (i.e., if a NOAEL is not available)

If 25 mg/kg-day had been the lowest dose tested,

UF = 10H x 10A x 10S x 10L = 10,000

MF = 0.8

Therefore UF x MF = 8,000, so that

RfD = LOAEL/(UF x MF) = 25 (mg/kg-day) / 8000 = .003 mg/kg-day)

2. Risk Characterization Considerations

Suppose the estimated exposure dose (EED) for humans exposed to the chemical under the proposed use pattern were .01 mg/kg-day; i.e.,

EED > RfD

Viewed alternatively, the MOE is:

MOE = NOAEL/EED = 5 mg/kg-day / 0.01 mg/kg-day = 500

Because the EED exceeds the RfD (and the MOE is less than the UF x MF), the risk manager will need to look carefully at the data set, the assumptions for both the RfD and the exposure estimates, and the comments of the risk assessors. In addition, the risk manager will need to weigh the benefits associated with the case, and other nonrisk factors, in reaching a decision on the regulatory dose (RgD).

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