Risk-Cost Assessment Methodology for Toxic Pollutants from Fossil Fuel Power Plants

J. G. Bolten, P. F. Morrison, K. A. Solomon

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PREFACE

Risk assessment of toxic pollutants has received increasing attention in recent years. This report describes the development and application of a general methodology for assessing the control costs and chronic health risks of toxic pollutant emissions from coal-fired electric power plants. The approach emphasizes surface water discharges and pollution, but it incorporates emissions to air, water, soil, and groundwater and transfers of pollutants between these media. Although it was developed for the utility industry, the methodology could reasonably be applied to toxic emissions from most point sources, so long as the emissions are reasonably continuous and may lead to chronic health risks.

This report provides a basic overview of the methodology, discusses its various components in detail, and describes its application to a simplified case study. It is the first presentation of results in a continuing study sponsored by the Energy Analysis and Environment Division of the Electric Power Research Institute. Additional work and further refinements of the methodology will be discussed in future documents. The material in this report should be of interest to federal, state, and local policymakers, as well as members of industry, concerned with risk assessment and pollution control regulation.

SUMMARY

In recent years the public has become increasingly aware of the potential problems posed by toxic substances in the environment. These materials can be emitted by many different types of industrial facilities, including coal-fired power plants. Toxic emissions from such sources will be dispersed throughout the surrounding region and may be transferred from one medium to another or taken up by local organisms and passed through food webs. The regional population can be exposed to these toxic substances through a variety of pathways, including inhalation, dermal absorption, and consumption of drinking water, aquatic organisms, or animal and vegetable products. Continued exposure to even low levels of these pollutants may lead to development of chronic health problems, most notably various types of cancer.

The growing concern about these problems has led to the passage of pollution control legislation at the state and federal level. In particular, the Clean Water Act of 1977 specifies 65 toxic pollutants or groups of pollutants whose discharge into surface waters will be regulated by the Environmental Protection Agency. In the long run, such regulations will have to be based on assessment of the relative costs and risks associated with the emission and control of toxic substances.

The electric utility industry, and coal-fired power plants in particular, will be subject to guidelines or restrictions on toxic emissions to all media (air, surface water, and soil or groundwater). To compare the relative costs and risks associated with different levels of regulation for a toxic pollutant, we have developed a risk-cost methodology. Although this methodology emphasizes discharges to surface waters, because of intermedia transfers it incorporates emissions to all media and the subsequent transfers of pollutant between them. It determines the chronic health risks associated with these emissions and facilitates their comparison with the direct costs of controlling the emissions.

The methodology follows toxic emissions from a coal-fired power plant through the different media to determine environmental concentrations in the region surrounding the power plant and more distant areas, if appropriate. To do this, we apply a composite multimedia modeling approach that uses independent models for each medium and couples their inputs and outputs to provide the necessary interactions between models. The resulting environmental concentrations are used to calculate exposure rates from

- Inhalation of atmospheric pollutants.
- Dermal contact with pollutants in the atmosphere or in surface and groundwater.
- Ingestion of domestic water supplies, aquatic organisms, or animal and vegetable products.

These calculations incorporate the effects of bioaccumulation or bioconcentration in transfers through regional food webs. The exposure rates are combined with regional population data and dose-response models to calculate the incidence of chronic health effects in the area surrounding the power plant.

The calculation of chronic health risks can be difficult. Generally, the results of a risk analysis of this type will have significant uncertainty. This approach facilitates investigating the sources and propagation of uncertainty through the calculations. In addition to applying conventional techniques to study how risk estimates vary with model parameter values, the user can observe how different models may change the results at various stages of the analysis. This flexibility should also make the risk assessment framework more cost-effective in the long run, because the analysis can emphasize areas that are most important to a problem.

The determination of emission control costs can be as difficult as the assessment of chronic health risks. The electric utility industry has had little experience with the control of toxic substances in gaseous or liquid effluents. Technologies that have been used in other

industries may not be cost-effective at coal-fired power plants, because utility wastes have larger volumes and lower toxic pollutant concentrations than other industries. Consequently, information about both the cost and effectiveness of different types of controls has been limited. Moreover, because coal-fired power plants differ widely in how they use and manage water, control schemes and costs will frequently be plant-specific.

We have illustrated the use of our risk-cost methodology by applying it to a case study of arsenic discharges from a hypothetical power plant located near Richland, Washington. This study emphasized direct discharges from the plant into the Columbia River, and calculated how these discharges would affect the incidence of skin cancer in the regional population. We limited our exposure calculations to ingestion of drinking water, but investigated how our results varied with changes in parameter values as well as dose-response models.

Although the basic approach to risk assessment can be described, it may be difficult to apply this procedure to specific cases. Potential problem areas include:

- Application and coupling of suitable environmental models.
- Obtaining data about emissions, environmental behavior, and health effects of toxic pollutants.
- Selection and estimation of appropriate dose-response models.
- Estimation of pollutant emission control costs and effectiveness.

As a consequence, the exposure calculations, in particular, must be highly uncertain, even for such direct exposure pathways as consumption of drinking water. Moreover, calculation of health effects associated with these exposure rates will also be extremely uncertain until research provides more information and a basis for selection of particular dose-response models.

The arsenic concentrations and net risk values found in the hypothetical case study indicate that the arsenic exposure and risk associated with the plant emissions are small compared to background levels. However, the sensitivity analysis found that the net risk

estimates can be extremely sensitive to the values of certain parameters and the choice of dose-response model. Moreover, the choice of dose-response model will also affect the sensitivity of risk estimates to variations in other parameters. This demonstrates the importance of using site-specific data, whenever possible, in an actual risk assessment.

Risk assessment always demands a thorough understanding of the problem. The analyst must have reasonable data and knowledge of the basic characteristics of the power plant, regional environment, and toxic pollutant. This study represents an initial attempt to develop a useful framework for the risk assessment process. Future work in this area will be directed toward expanding and improving the basic methodology, making it more accessible to the potential user, and applying it to additional, more realistic case studies.

ACKNOWLEDGMENTS

This study could not have been completed without the assistance of many people. It would be impossible to mention them all. Although we will acknowledge the contributions of certain individuals and organizations, others undoubtedly also deserve our gratitude.

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Although we were heavily dependent on the contributions of other people, we must take sole responsibility for any errors that may remain in this volume.

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1. INTRODUCTION

Legislative and administrative actions during the last five years have reflected a growing national concern over the effects of toxic pollutant emissions. The 1977 amendments to the Federal Water Pollution Control Act (FWPCA) were in part designed to strengthen the federal basis for controlling these emissions to surface water bodies. Establishment of an effective long-term regulatory program, however, will require that water quality and effluent standards be based on assessment of the costs and benefits associated with the emission of toxic pollutants.

The electric power industry will be subject to the toxic pollutant regulations developed under the Clean Water Act and other legislation, including the Resource Conservation and Recovery Act of 1976. With the expected increased use of coal, relative to other fuels, for power generation, toxic emissions from power plants have become the subject of growing concern, within both the industry and government agencies at the federal and state level. These organizations have emphasized the need for an approach to determining the health risks and control costs of toxic emissions from coal-fired power plants.

LEGISLATIVE BACKGROUND

The Water Quality Act (WQA) of 1965 was the first federal regulatory program mandating regulation of a point source based on a prediction of how it affected the quality of ambient surface waters. Although the WQA permitted enforcement of water quality standards before a violation occurred, it had several enforcement problems. It allowed the federal government to initiate enforcement procedures only when pollution in one state endangered the health and welfare of persons in another state or damaged the interstate marketing of shellfish or fish. When the pollution was confined within one state, the federal government could intervene only when the state expressly requested it to do so.

The Federal Water Pollution Control Act Amendments of 1972 (Public Law 92-500), further amended by the Clean Water Act of 1977 (Public Law 95-217), were drafted with the intent of correcting some of the enforcement problems of the 1965 WQA.

The goal of the FWPCA were to "restore and maintain the chemical, physical, and biological integrity of the Nation's waters." This goal was to be achieved by requiring dischargers to comply with federally established effluent treatment levels and limitations by specific dates. Dischargers were to achieve by July 1, 1977, effluent limitations that represented the application of the "best practicable control technology currently available" (BPT). Not later than July 1, 1983, dischargers must meet effluent requirements that represent the "best available technology economically achievable" (BAT). Industries that discharge into a publicly owned treatment works (POTW) must meet certain "pretreatment standards" by these dates. In addition, new discharge sources are subject to national standards of performance that reflect the greatest degree of effluent reduction achievable through application of the best available demonstrated control technology, processes, operating methods, and other alternatives.

The FWPCA also requires the Environmental Protection Agency (EPA) to establish effluent standards or prohibitions controlling toxic pollutants. Toxic pollutants are defined as those pollutants, or combinations of pollutants, which after discharge and upon exposure to any organism, either directly or indirectly, will "on the basis of information available to the Administrator" cause death, disease, or other abnormalities in the organism or its offspring. The EPA must issue a list of toxic pollutants and then must establish standards for those pollutants. In determining effluent standards for those toxic pollutants, the EPA must consider the pollutants' toxicity, persistence, and degradability, as well as the presence of organisms in any affected waters.

The 1972 act recognizes the importance of water quality standards. The EPA is required to establish and periodically review water quality criteria for several classes of water use. These criteria can be used to establish enforceable water quality standards for interstate and intrastate waters.

To implement these various effluent limitations and water quality standards, the act requires major point source dischargers to obtain permits under the National Pollutant Discharge Elimination System (NPDES). These permits impose certain requirements on the discharger, who must apply for the permit with data describing his pollutant emissions. Each permit should contain conditions requiring the discharger to meet all effluent limitations and treatment technology requirements (including schedules of compliance), water quality standards, new source performance standards, and toxic effluent standards. Facilities discharging into POTWs do not require permits but must meet all appropriate pretreatment standards.

The Clean Water Act (CWA) of 1977 amended the FWPCA in several significant ways, but did not change the basic goals of the act. It made important changes to the 1972 legislation with regard to toxic substances. The EPA had not been able to implement the provisions of the FWPCA that related to toxic pollutants. Consequently, the Natural Resources Defense Council filed suit against the EPA to force regulation of 65 specified toxic pollutants. In the resulting Consent Decree, EPA agreed to develop and issue BAT effluent limitations guidelines, pretreatment standards, and new source performance standards for 21 major industry groups, including the steam-electric utility industry, for the 65 pollutants, or groups of pollutants, by December 31, 1979. These pollutants are shown in Table 1.1.

The 1977 act codified this strategy and incorporated the list of 65 toxic pollutants directly. In addition, it did the following:

- Set July 1, 1984, as the deadline for compliance with the BAT effluent limitations for these pollutants.
- Required compliance within three years of promulgation of effluent limitations for other toxic pollutants added to the list.
- Provided that the EPA may add to, or remove from, the list of toxic pollutants as deemed necessary, on the basis of listed toxicity factors.

Table 1.1

TOXIC POLLUTANTS LISTED IN THE CLEAN WATER ACT

Acenaphthene	Cyanides	Naphthalene
Acrolein	DDT{b}	Nickel{a}
Acrylonitrile	Dichlorobenzenes	Nitrobenzene
Aldrin/dieldrin	Dichlorobenzidine	Nitrophenols
Antimony{a}	Dichloroethylenes	Nitrosamines
Arsenic{a}	2,3-dichlorophenol	Pentachlorophenol
Asbestos	Dichloropropane (-pene)	Phenol
Benzene	2,3-dimethylphenol	Phthalate esters
Benzidine	Dinitrotoluene	Polychlorinated
Beryllium{a}	Diphenylhydrazine	biphenyls
Cadmium{a}	Endosulfan{b}	Polynuclear aromatic
Carbon tetrachloride	Endrin{b}	hydrocarbons
Chlordane	Ethylbenzene	Selenium{a}
Chlorinated benzenes	Fluoranthene	Silver{a}
Chlorinated ethanes	Haloethers	2,3,7,8-Tetrachloro-
Chloroalkyl ethers	Halomethanes	dibenzo-p-dioxin
Chlorinated	Heptachlor{b}	Tetrachloroethylene
naphthalene	Hexachlorobutadiene	Thallium{a}
Chlorinated phenols	Hexachlorocyclohexane	Toluene
Chloroform	Hexachlorocyclopentadiene	Toxaphene
2-chlorophenol	Isophorone	Trichloroethylene
Chromium{a}	Lead{a}	Vinyl chloride
Copper{a}	Mercury{a}	Zinc{a}

{a} Element and compounds.

{b} Material and metabolites.

- Required control of all toxic pollutants using BAT regulations, unless the EPA issues more stringent standards.
- Set July 1, 1980, as the deadline for issuance of BAT regulations for the original list of 65 toxic pollutants.
- Required compliance with toxic pollutant effluent standards within one year of promulgation, with a potential three year extension if compliance is technologically infeasible for a category of sources.
- Permitted the EPA to exclude a pollutant from regulation if it is either not present in effluent, is present only in trace amounts, or is present only because of its presence in intake water.

To implement these and other requirements of the Clean Water Act, the EPA has used the list of 65 toxic pollutants to generate an expanded and more specific list of 129 priority pollutants (EPA, 1980a). On November 28, 1980, they published final water criteria and documents for the 65 toxic pollutants (EPA, 1980b).

RISK-COST-EFFECTIVENESS METHODOLOGY

The FWPCA directs the EPA to consider the costs and benefits of control technologies in making decisions about control of toxic substances. Although these requirements have been variously interpreted by the courts, the Administrator is required to balance total costs and effluent reduction benefits in determining BPT, and to consider costs as one factor in interpreting BAT for an industrial category and pollutant. Moreover, it is clear that state and federal laws, regulations, and court decisions relating to toxic emission standards will have to consider the relevant risks, costs, and effectiveness of controlling these emissions.

One consequence of this growing reliance on risk and cost estimates is the need for accurate and reliable methods of risk assessment, not only for the electric utility industry, but for all industries. Accordingly, the primary objective of our research has been to develop a general methodology for assessing the costs and risks associated with the control of toxic substances emitted by coal-fired power plants. We have also tried to demonstrate the use of this methodology by applying it to the control of one toxic pollutant at a hypothetical power plant.

Although our risk methodology has emphasized the contamination of surface water bodies, as regulated under the Clean Water Act, it is designed to follow and include toxic emissions of all types to all environmental media. By doing so, it should form the basis of an integrated risk assessment methodology for emission of toxic pollutants from point or area sources. Emitted pollutants are traced through the environment to determine the exposure rates and chronic health effects in an exposed population.

The risk methodology consists of a general framework into which specific models can be inserted. The choice of models at any stage of the analysis will depend on the resources, needs, interests, and abilities of the user, as well as the specific characteristics of the problem. The framework should be able to deal with a wide variety of pollutants and plant site conditions. In addition to this flexibility of application, the framework permits the user to compare the results obtained from alternative models. When combined with the results of uncertainty analysis applied to the model parameters and other inputs, this should provide valuable insight into the application and limitations of particular models and the relative contributions of these models to overall uncertainty in the calculations.

ORGANIZATION OF THE REPORT

In this report we describe our risk-cost-effectiveness methodology and apply it to a simplified case study. Chapter 2 describes the overall methodological framework and how it can be applied to a risk assessment problem. This description also includes a discussion of how uncertainty can affect the results of an analysis and of alternative approaches to dealing with this uncertainty. Chapters 3 through 6 consider the various stages of the risk analysis in more detail. Chapter 3 describes the general area of pollutant emissions and control options for toxic materials discharged by coal-fired power plants. In Chap. 4, we discuss the general subject of environmental transport and fate. This discussion also considers the basic requirements for models to determine pollutant transfers between media and concentrations in the atmosphere, soil, groundwater, and surface waters surrounding a power plant. Chapter 5 summarizes pollutant uptake, concentration, and accumulation by plants and animals, and describes how we model the many potential exposure pathways leading to man. Chapter 6 explains the theory and practice behind the determination of chronic health effects in humans. This explanation includes a discussion of alternative dose-response models and their application, pharmacokinetic considerations, and the uncertainty associated with alternative models and their parameters.

Chapter 7 discusses the problem of defining and calculating control costs for toxic pollutant emissions. Representative cost calculations for the case study are presented in Chap. 8, which describes in detail how the methodology was applied to a hypothetical power plant, and presents the results of the basic calculations. Our sensitivity analysis of the case study is explained in Chap. 9, where we also discuss our conclusions and their implications for the general area of risk analysis.

Several appendixes are included to add supporting material for the main text. Appendixes A and B amplify the discussion of health effects in Chap. 6. Appendix A considers the solution and parameterization of the two-component, linear pharmacokinetic model, and App. B discusses more complex physiological pharmacokinetic models. Appendix C discusses how the selected environmental transport models should be coupled and applied to an integrated risk analysis. In App. D we describe in some detail the various potential discharge streams for toxic emissions from coal-fired power plants. Alternative control technologies and procedures for these emissions are presented and described in App. E. Appendix F contains the mathematical description and equations for the exposure calculations discussed in Chap. 5. Finally, App. G presents a description of the computer program that we developed for this study to calculate exposure rates and chronic health effects in the human population around a power plant. This appendix also includes a description of the primary program variables, inputs and input formats, the types of output generated by the program, an example of the inputs and outputs associated with a typical analysis, and a listing of the computer code.

2. DESCRIPTION OF THE METHODOLOGY

The basic objective of this study is to develop a methodology to assess the risks and costs associated with controlling the emissions of toxic pollutants from coal-fired power plants. This type of framework is needed if we want to balance the risks, costs, and benefits of regulating the emissions of such toxic substances. Such risk-cost-benefit assessment falls under the general envelope of cost-benefit analysis.

COST-BENEFIT ANALYSIS

Cost-benefit analysis is the systematic evaluation of all benefits and costs (both tangible and intangible) that will accrue to society under a set of alternative policies. Because this technique has been widely described in the literature (see, for example, Mishan, 1976), we will not discuss it in detail. In principle, the process consists of the following eight steps:

1. Establish project goals or objectives.
2. Identify alternative policies.
3. Formulate analysis assumptions.
4. Determine impacts of alternatives (present and future).
5. Assign values to impacts.
6. Compare costs and benefits of alternatives.
7. Choose preferred alternative using some decision rule.
8. Test sensitivity of costs, benefits, and choice to analysis uncertainties.

Although the procedure appears clear in principle, applying it to a specific problem can be difficult and can often produce controversial results. Five primary problems of implementing cost-benefit analysis are:

- Specifying or defining all benefits and costs.
- Predicting the outcomes of alternative policies.
- Quantifying benefits and costs in common units.
- Incorporating distributional effects of alternatives.
- Discounting costs and benefits.

Many of these problems arise because policies may have impacts (both costs and benefits) that are intangible, delayed, highly uncertain, or even unknown. Benefits such as improvement in environmental quality or reduction in health risks may inherently be difficult to quantify, particularly in monetary terms. Some costs, including regulatory enforcement costs and indirect economic effects, may also be hard to determine. Both costs and benefits may be distributed unevenly across population and economic groups or geographical regions. Traditionally, cost-benefit analysis does not consider these distributional effects.

Alternative policies will have impacts that vary over time. Typically, one compares these temporal distributions of costs and benefits by using a discount rate to reduce them to present values. Algebraically, the present value of a future stream of costs or benefits is given by

$$\text{Present Value} = \sum_i X_i / (1 + r)^i$$

where X_i = cost (benefit) in year i (dollars),

r = annual discount rate,

i = index for years from start,

n = duration of time stream of costs (benefits) (years).

The present value of a stream of benefits may be extremely sensitive to the choice of discount rate. Accordingly, this choice has long been a major source of controversy in cost-benefit analysis and no definitive solution has been found. Discount rate selection and other problems associated with cost-benefit analysis are discussed in Stokey and Zeckhauser (1978), Mishan (1976), Pigou (1932), and Mikesell (1977).

COST-EFFECTIVENESS ANALYSIS

Cost-effectiveness analysis is similar to cost-benefit analysis, except that costs and benefits can be measured in different units without a common metric. This approach can be applied most easily when either (1) every alternative is constrained to have the same cost, or (2) alternatives are chosen to have the same benefits (effectiveness). In some instances where these conditions do not hold, we can compare alternatives by determining the ratio of effectiveness to cost for each policy--a measure of benefits per unit cost. Because costs and benefits are measured in different units, however, we cannot be sure for any policy that total benefits exceed total costs, i.e., that the undertaking is justified.

RISK-COST-BENEFIT ANALYSIS

Risk-cost-benefit analysis can be used to assess alternative actions that cause changes in risks to human health. This type of analysis identifies the health effects of a policy separately and compares them with the other costs and benefits. As in cost-effectiveness analysis, although the risks, costs, and benefits are expressed quantitatively, they need not be defined in common units.

In our approach, we have specified benefits to be solely the change in risk to human health. We have not attempted to include other benefits of toxic pollutant control--benefits such as reduced damage to animal, aquatic, and vegetable species in the environment.¹ Human risk has been defined as the total number of cases of each health effect in the regional population. Because background concentrations of most toxic pollutants will not be negligible, the net risk associated with any alternative is the difference between total risk with the alternative and total risk from background sources only.

¹The methodology could be extended to include these benefits in the future. It would require identifying affected species and determining how much they have been affected. This damage could be evaluated either in monetary terms or left in its own natural units for comparison between alternatives.

An analyst may or may not choose to use discounting techniques on the human risk calculations. In the dose-response equations that we have used, risk is defined as total lifetime incidence of the health effect. There will be a substantial time delay between initial exposure to the toxic material and incidence of the effect. This time delay will depend on the pollutant and health effect, among other things, but will probably have a significant random component. Although it would be possible to specify a distribution of incidence times, and use this for discounting, the results would have little meaning unless the health effects were converted to some common monetary basis, which we do not propose.

For this analysis we have defined costs to include only initial capital investment and operation and maintenance expenses of alternative control schemes. We have not therefore included any costs to the government for administration, monitoring, or enforcement of control regulations. These costs should not influence decisions made by an electric utility, although they should be important to a government agency in its decisions about regulation levels. Finally, we have not included the indirect economic costs or intangible costs associated with toxic pollutant control. To incorporate these costs accurately would require an extensive analysis beyond the scope of this study.

The electric utility industry commonly uses the concept of levelized cost or revenue requirements to compare alternative investment schemes. This approach is a method of discounting that converts a stream of costs or revenue requirements to an equivalent constant annual requirement over the life of the project. The Electric Power Research Institute (EPRI, 1979) has described this procedure and supplied a set of recommended values for the variables needed to implement it. We have used these assumptions in our cost calculations, as described in Chaps. 7 and 8.

To compare the alternative control policies in an analysis, one must define a base case. This should be the "current" situation, with respect to pollutant control levels or equipment and plant operation. For a new power plant, the base case could be defined either as the situation without the plant or as a nominal plant without specific

pollutant control systems. Often, the base case can be regarded as the case with the least regulation of emissions. We can define the control costs for the base case to be zero, and specify the costs of alternatives to be the incremental costs from this situation. The same definitions can be applied to the determination of risks. We can calculate the risk associated with the base case and the alternatives, and compare them accordingly. When the base case is a situation without the power plant, its risk will be that associated with the background pollutant levels in the environment.

Once the costs and risks of the alternatives have been determined, they must be presented in a manner that facilitates deciding among them. This can be done by plotting the cost versus risk for each alternative on a common graph, as shown in Fig. 2.1. If risk is defined in terms of two or more health effects, they can be combined into one risk index as described below. In the figure, each point represents the relative costs and risks of one alternative. The envelope curve connects the set of "efficient" alternatives, although points on the curve between alternatives may not be achievable. Constraints on cost or risk can be

Fig. 2.1 — Risks and costs for alternative cases

included as horizontal or vertical lines on the graph, also as shown. Using this method of comparing costs and risks, it should be possible to select a preferred alternative.

OVERVIEW OF METHODOLOGY

We have tried to develop a risk-cost-benefit analysis framework that is as general and flexible as possible, so that it can be applied to a wide range of toxic emission problems. For any given problem, the user of this methodology must, for each alternative configuration or case:

1. Specify plant pollutant emissions.
2. Estimate pollution control costs.
3. Assess the health risk to the surrounding population.

The general risk assessment framework required for step 3 consists of five basic stages, as described below. We will briefly discuss each of the above steps in this chapter, and consider them in more detail later.

Plant Pollutant Emissions

Coal-fired power plants (and other fossil fuel plants) can emit pollutants into the air, surface water, and soil or groundwater. Although this study emphasizes surface water emissions, because of intermedia transfers our risk methodology must include all of the emissions. As a consequence, one must be able either to measure or calculate the appropriate rates for each type of discharge. These emission rates must be specified for each alternative plant configuration or pollution control scheme.

Emissions can be classified by the medium into which they are discharged. Atmospheric emissions include both stack gases and emissions from coal storage piles and ash disposal operations. Because they may behave differently, gaseous and particulate discharges should be considered separately. Depending on plant water management systems, many sources can contribute to surface water pollution. The primary sources include:

- Ash handling and transport systems.
- Cooling systems.
- Water treatment processes.
- Flue gas desulfurization (FGD) systems.
- Metal cleaning operations.
- Coal-pile runoff.

Contamination of the soil and groundwater around the plant will be caused primarily by leaching from coal piles, ash and sludge disposal sites, and treatment ponds. Source emissions are discussed in more detail in Chap. 3 and App. D.

Ideally, emission rates should be based on measured data. The behavior of toxic pollutants within power plants is not well understood and depends critically on such factors as (1) the pollutant and its sources within the plant, (2) plant age, processes, and design, (3) pollution control technology and systems, and (4) maintenance history and procedures. Trying to estimate emission characteristics and rates may add unnecessary uncertainty to the calculations if measured data can be obtained. On the other hand, if monitoring programs are infeasible, source emission rates can be based on a combination of measurements and calculated values.

Pollution Control Costs

For each alternative plant configuration or pollution control system, the user must estimate the direct control costs. As discussed earlier, these costs should include initial capital investment as well as operation and maintenance expenses. Costs for each alternative should be calculated relative to the base case configuration, because the analysis considers incremental costs and risks of pollution control levels.

In general, the direct pollution control costs will depend strongly on the specific design of the power plant, its water management systems, and its operating procedures. Consequently, it will normally not be possible to determine the costs associated with a particular control

process without referring to a specific plant design. Where two or more pollutants will be controlled by one alternative system, the user should not attempt to allocate control costs between pollutants. Any allocation scheme would have to be arbitrary and would not reflect any natural division. Instead the user should calculate the total risks associated with emissions of both pollutants for each alternative and compare these to total control costs.

We have used a particular standard technique for calculating and expressing costs in our case study (see Chaps. 7 and 8). That standard, used by EPRI, has been described in EPRI (1979). It is not necessary that a user follow this same procedure, however, so long as all costs are defined in a consistent manner between alternatives.

Health Risk Assessment

We can consider the framework for risk assessment to consist of five distinct stages: (1) source generation, (2) environmental transport, (3) exposure calculation, (4) population description, and (5) dose-response modeling. To assess the risk associated with emission of a toxic pollutant from a specific source, we must use models, measured data, or a combination of both at each stage of the analysis. We can describe the overall process by referring to Figs. 2.2 and 2.3, which present a schematic outline of the risk assessment process.²

Figure 2.2 traces the toxic pollutant from the input coal (arrow entering at the left of the figure) through the source generation and environmental transport stages.³ The pollutant passes through the coal

²In these figures, rectangles indicate processes' activities. Plant emissions or wastes are shown by slanted rectangles, and hexagons indicate pollutant concentrations either in the environmental media or parts of the exposure network. Circles are used to show pollution control points, where use of some treatment process or control technology could reduce the amount of pollutant reaching the general population. The arrows indicate the general flow of pollutants through the environment and the risk assessment process.

³In this analysis we do not consider the risks associated with mining, preparing, or transporting coal to the power plant. Expanding the problem boundaries to include these processes would have made the analysis too complex. Moreover, regulation of steam electric power plants under the Clean Water Act does not cover these aspects of power generation.

Fig. 2.2 — Risk assessment framework — part 1

Fig. 2.3 — Risk assessment framework — part 2

storage pile and the various processes within a power plant to be discharged into the environment. These discharges include emissions into the air, surface water, and soil or groundwater. Discharges into the different media are dispersed, transformed, and transferred between media. The figure indicates these general processes and the interactions between media that must be considered in determining the distribution of pollutant concentrations in the region surrounding the source.

Figure 2.3 begins with these environmental concentrations and shows the stages of exposure calculation, population description, and dose-response modeling. Pollutant concentrations in the environmental media are passed through food webs and other exposure pathways (both direct and indirect) to generate exposure rates by type of intake for the regional population. These exposure rates are combined with a regional population model or other information and with dose-response models for the pollutant to calculate the overall risk associated with the original emission levels.

The methodology has been designed to determine chronic health risks that, as discussed, are most likely for the regional population. In limiting the assessment to chronic health risks, we have been able to restrict our study to long-term or average emission and exposure rates. This permits the use of steady-state models in the environmental transport and exposure calculations, and considerably simplifies the calculation procedure.

Source Generation. Source generation includes determining the primary emission sources from a power plant and calculating or measuring the emission rates for all pollutant species of interest. We discussed plant emissions earlier, because, although we include source generation as a part of the risk assessment methodology, it would normally be a separate calculation or measurement program outside the model framework. Emission rates, particularly for direct discharges to water, depend strongly on plant design and operation, and cannot easily be calculated using models. Under most circumstances, toxic pollutant concentrations and discharge volumes would have to be based on measured data.

Emission rates must be specified in a manner that is compatible with the input requirements of the environmental transport models selected for the analysis. This compatibility includes not only measurement units, but also temporal and spatial resolution. However, the availability of suitable emission information may be a constraint on the choice of models to be used in the analysis. Because we are dealing with chronic health effects, we can normally restrict ourselves to considering long-term average emission levels. Only when health data indicate that short-term exposure variations are important to chronic effects should it be necessary to consider such time periods. Even with this limitation, however, it may be extremely difficult to determine accurate rates for a given source.

Environmental Transport. After release from their source, toxic materials follow a complex path through the environment before reaching man. Processes that affect these pollutants include (1) transport, (2) physical and chemical transformation, (3) degradation and decay, (4) transfer between media, and (5) biological uptake and transport through the food web. To follow the passage of a pollutant through the environment, we must be able to model the appropriate processes in each of the media, as well as the transfers between media. We can approach this problem of multimedia environmental modeling in two distinct ways.

The first approach is to develop a fully coupled multimedia model. Such a model contains submodels for each of the media (air, soil, groundwater, and surface water). These submodels are fully integrated and each can interact with all other submodels. Unfortunately, this type of model can quickly become extremely large and difficult to use, unless the various submodels are simplified. When this is done, the submodels become less accurate, and the results of the overall model become less realistic. In many cases these results may be adequate for the analysis, but frequently it is necessary to have more accurate calculations of pollutant concentrations in the environment.

The second approach is to utilize a composite multimedia model. In this model we select appropriate models for each medium and couple their inputs and outputs externally, using either small computer programs or hand calculations. This allows us to use more realistic models, if

necessary, while retaining the option of employing the simplified models if they are suitable.⁴ Because models for each pathway can be replaced, the overall methodology is more flexible and can be adapted to a variety of sites and pollutants. In the long run, this may make the approach less expensive and more applicable to the problem. The ability to substitute alternative models also provides an opportunity to compare intermediate outputs between models and to assess the relative sensitivity of our results to model choice. This would be useful in assessing and reducing the overall uncertainty in a risk analysis.

We have chosen to use the composite multimedia approach. In doing so, we must address the primary problem associated with this approach--that of adequately coupling the models to account for intermedia transfers. We achieve this coupling by vectoring the output file of one pathway model to the input file of the next pathway model. Feedback between models can be incorporated by using external calculations and applying the concept of primary and secondary pollutant pathways.

Primary pathways begin at the emission source. They represent local problems having acute environmental effects from higher contaminant concentrations. For secondary pathways, the contaminant has first been transported by some other medium--the primary pathway. These pathways represent more regional problems with chronic environmental effects from lower contaminant levels. Concentrations are lower because the pollutant has been dispersed, diluted, and perhaps degraded in the primary pathway.

The primary and secondary pathways that should be considered for power plant discharges are discussed in App. C. Briefly, for surface water pollution, the primary pathway includes direct discharges into the surface water by the power plant. In most regions, these are likely to be far more important pollutant sources than secondary pathways such as deposition of atmospheric pollutants or contributions from contaminated groundwater. For groundwater pollution, the primary pathways include leaching from coal piles and ash or sludge ponds. Again, pollutant

⁴Although we are concerned with chronic health effects and long-term pollutant concentrations, the individual models need not necessarily be steady-state. Time-dependent models may be used if their outputs can be averaged over the appropriate time periods.

contributions from these sources will dominate contributions from surface water sources or infiltration through regional soils.

In using the composite multimedia approach, we first analyze the primary pathways for plant emissions using local models. These results are then used as the secondary source inputs for appropriate regional models. Only when feedback of unexpected significance occurs between secondary and primary pathway models should it be necessary to rerun any of the secondary models.

Chapter 4 discusses environmental transport analysis in more detail and recommends a series of models that may be used for the various primary and secondary pathways. The interactions between these models are considered in detail in App. C.

Exposure Calculation. The population surrounding a power plant can be exposed to toxic emissions by three general routes: inhalation, ingestion, and dermal absorption. Not all of these intake modes will be important for any given pollutant. For many pollutants, only direct inhalation of atmospheric particulates or gases may be significant. For others, ingestion of polluted waters or aquatic organisms may be the primary route of exposure. For most toxic pollutants, dermal absorption can generally be neglected except in unusual circumstances. Nevertheless, the methodology can consider any of these exposure pathways that may be appropriate.

To calculate exposure, we first partition the area surrounding the emission source (power plant) into population subregions. These subregions form the basis for all exposure calculations. All members of a population group within a subregion are assumed to have the same exposure rates. Accordingly, mean pollutant concentrations and deposition rates need to be determined by subregion for many of the exposure calculations. The calculations and concentrations may be based either on particular pollutant species or a suitably defined combination of species. If exposure rates are determined by species, these can later be combined within the dose-response calculations.

In calculating inhalation exposure rates, we must differentiate between gaseous and particulate pollutants. Atmospheric transport models should be able to calculate concentrations for each form, although one form or the other may be negligible for a particular

pollutant. Gaseous and particulate pollutants will also behave differently within the human body. Absorption rates within the lungs may be different, for example, and a fraction of the particulates inhaled will actually be ingested. Pollutant behavior within the lungs is complex and highly variable. As with all exposure pathways, we must understand how the pollutant behaves before we can hope to make reasonable exposure calculations.

To clarify and simplify the calculations, we have separated ingestion exposure into five categories: (1) atmospheric particulates, (2) drinking water, (3) aquatic organisms, (4) animal products, and (5) vegetable products. We calculate exposure rates for each relevant category, using regional data where possible, basing the calculations on our definition of subregions. For atmospheric particulate ingestion, exposure rates come directly from atmospheric concentrations. Drinking water exposure is based on pollutant concentrations in the relevant water supplies for each subregion, including the effects of water treatment. We assume, however, that exposure rates for ingestion of aquatic organisms and animal and vegetable products are uniform (by population group) across the entire region.⁵ Concentrations within the food products are calculated by subregion, but these concentrations are averaged over the region, assuming uniform distribution of products. Where necessary, background exposure from ingestion of products produced outside the region is incorporated in the analysis. For many pollutants, consumption of drinking water and aquatic organisms may be the primary exposure routes. These two routes have generally been the only exposure pathways considered by the EPA in specifying water quality criteria for the priority pollutants (EPA, 1980c).

Dermal exposure comes from contact with atmospheric particulates, domestic water supplies, and recreational water. These calculations must be based on assumptions about contact times and exposed body area, using the appropriate pollutant concentrations in the atmosphere, domestic water supplies, and water at recreational locations in the region.

⁵Known variations in ingestion rates over a region could either be studied with sensitivity analysis or by creating a special population group existing only in the relevant subregions.

Chapter 5 discusses these exposure calculations in more detail, including information requirements, procedures, and appropriate units. We have also produced a computer program that calculates exposure rates from input pollutant concentrations in the various media and other appropriate information about the plant region. This program and a description of its use are given in App. C.

Population Description and Dose-Response Modeling. The risk assessment methodology should determine the net risk to regional population from chronic health conditions associated with a toxic pollutant. In this study, net risk is defined as the change in the total regional incidence of health effects associated with the change in pollutant emissions between alternative cases. Calculating net risk thus requires determining emission rates, environmental concentrations, and exposure rates for the "background" case and the situation of interest. The exposure rates, converted to equivalent doses, can then be used with dose-response models and population data to calculate total and net risk.

Exposure rates are determined as a function of subregion and subgroup within the population. Population subgroups may be based on age, sex, health status, or other significant variables. The definition of subgroups, however, should be based on known variations in exposure rate (or pollutant dose) or response to exposure. In other words, division by subgroup may not be worthwhile unless the exposure or health effects data are sufficient to distinguish between the subgroups.

By partitioning the population by subregion and subgroup we can incorporate two significant variables in the risk calculations. However, in reality both exposure rates and population characteristics will vary over time. Exposure rates will vary as the power plant changes its (1) capacity factor, (2) pollution control procedures, (3) fuel source, or (4) operating conditions. Changes in the regional environment may also cause exposure rates to vary. Population characteristics will vary over the life of a power plant (or other pollutant source) because of normal demographic processes, including urban development and migration, aging, birth, death, and immigration or emigration.

Although we would like to incorporate these variations in the risk calculations, this can be very difficult. Exposure rates can be predicted over time, but most risk analyses use mean values and assume that regional populations are static in space and time. These assumptions are necessary because most normal dose-response equations are based on lifetime, constant-dose experiments. When variable doses or population variations are important, the dose-response equations no longer hold in their standard forms.

In our initial analysis, we have used the constant exposure and static population assumptions as well, using mean values averaged over the life of the power plant. This approach may be reasonable for simple exposure variations, but it is less useful when a changing population is subject to different plant and background dose levels for varying periods of time. As a consequence, it will be necessary in the future to develop the equations and procedures required to incorporate basic population models and variable exposure rates into the calculations.

An additional complication arises when a toxic pollutant can cause more than one type of chronic health problem. In this case, the risk associated with an alternative becomes a vector containing the total cases of each health effect. Normally these effects will not be equally important, e.g., skin cancer and lung cancer. It may sometimes be desirable to convert the risk vector into a single index, for example, when doing simplified calculations to screen out undesirable alternatives early in an analysis. This conversion can be done by creating a weighted risk index of the form

$$N_r = \sum_i y_i n_i$$

where N_r = weighted risk index,

y_i = weight for health effect i ,

n_i = incidence of health effect i .

The weights for this process can be based on an appropriate measure, such as treatment costs or reduction in life expectancy. To insure that

the weights do not drive the analysis, however, one should compare alternative risks using a number of different weighting schemes. For more information, refer to Solomon and Abraham (1979) who have described this concept and applied it to the problem of occupational risk.

The basic assumptions and principles behind the use of dose-response models to calculate chronic health effects are discussed in Chap. 6. There we describe how the primary models have been developed and how they can be applied to a risk analysis problem. In our case study, discussed in Chap. 8, we use four of these models to calculate the risk of skin cancer associated with discharges of arsenic by a coal-fired power plant.

APPLICATION, ASSUMPTIONS, AND LIMITATIONS

The potential user should recognize that risk analysis is not a precise science that can develop exact risk estimates. Instead, risk analysis is an exercise that can (1) roughly approximate the relative risks of alternative designs, (2) determine the relative sensitivities of calculated risks to uncertainties in input data, and (3) identify weaknesses in the original assumptions. Our risk assessment methodology is based on a particular set of assumptions and is subject to limitations. If some aspects of a toxic emission problem fall outside these limitations, these aspects may require separate independent analysis.

This risk assessment framework should not be used without a thorough understanding of the various models and their application. This is true, however, of any risk analysis. One cannot construct a foolproof package of models that can be properly used by someone unfamiliar with the basic problem or the general situation. The user must also have access to adequate information about (1) the operation and characteristics of the power plant and its waste streams; (2) regional geography, geology, hydrology, and meteorology; (3) regional population; (4) the behavior and chronic health effects of the pollutant; and (5) regional production and consumption of animal, vegetable, and aquatic organisms (if relevant). No toxic risk assessment could be done without this information, although the level of detail required will differ from one case to another.

One must also remember that other sources of toxic pollutants may be important in many regions, and that these sources must be included. For example, priority pollutants may show up in pesticides or fertilizers used in agriculture, or in the discharges from municipal, commercial, or other industrial sources. Normally these contributions to regional background levels would be independent of the power plant, but they would not be properly considered if only national averages or other typical values were used in the analysis. Thus it is important that as much site-specific information as possible be used in the risk assessment.

Certain other basic assumptions or limitations should be discussed. These assumptions either affect the application of the methodology or the content of this report.

Health Effects

All toxic pollutants may have both acute and chronic effects. Acute effects are caused by short-term exposure to high concentrations. For surface water pollution, these concentrations are probably much higher than power plant operation should produce in the surrounding environment. As a result, our methodology has been designed to assess the risk associated with chronic health effects in the exposed population. These effects are caused by prolonged exposure to lower pollutant concentrations, although short-term exposure to higher concentrations may also contribute to their incidence. We do not limit the health effects solely to carcinogenesis but include other chronic conditions or diseases. Thus, although we are more concerned with prolonged exposure rates or total accumulated doses, depending on the pollutant, we must not ignore the possible contributions to chronic health effects from high exposure rates over a relatively short period.

Time Frame

Because we are dealing with chronic health risks, we have limited our analysis to long-term conditions. In other words, emission rates are assumed to be annual average values based on more-or-less continuous operation and discharge of pollutants. Although emissions and

environmental conditions may vary significantly in the short term (from hourly to daily), if the resulting concentrations are below acute levels, these variations can be averaged out over the course of a year.⁶ For variations with monthly or seasonal periods, it may be necessary to calculate corresponding exposure levels if dose-response models and health data are sufficiently detailed. In general, however, we would expect that long-term average values, reflecting some normal operating level for the power plant and typical environmental conditions, should determine the character of the chronic risk faced by the surrounding population.⁷

By restricting our analysis to long-term or average circumstances, we can select environmental transport and exposure models that assume steady-state conditions. Although time-varying models may be used, they are not required. Changes in long-term operating or emission rates may generate transient phenomena in the environment. For atmospheric and surface water transport, these transients should not last. For soil and groundwater transport, however, because processes occur more slowly and time delays become important, it may be necessary to consider transient conditions in more detail to properly incorporate them into the analysis. We have structured the methodology to do this, but have not incorporated these calculations in our case study (see Chap. 8).

Environmental Pathways

This study has primarily been directed toward regulation of toxic emissions discharged into surface waters. Because pollutants may be transferred between air, water, and soil, however, the risk assessment models must be able to incorporate emissions to all media, secondary

⁶If plant emissions were somehow correlated with environmental conditions, such as wind direction or surface water flow rates, this would have to be considered in the environmental transport analysis.

⁷Time-weighted averaging of exposure levels may not always be appropriate. Toxicologists have found that in some cases duration of exposure and concentration of toxicant are not inherently interchangeable, particularly if concentrations vary over large ranges. Whether these variations would have to be included in a particular risk analysis would depend on the pollutant characteristics, emission variations, environmental concentrations, and dose-response model availability.

transfers between media, and the subsequent exposure pathways, not just those directly relating to surface water bodies. Our discussion of power plant emissions and control technologies, however, concentrates on direct discharges of liquid wastes into surface waters. Processes and control technologies for plant emissions into the other media have not been considered in detail.

Toxic Pollutants

The federal government regulates toxic emissions to surface water bodies primarily under the provisions of the Clean Water Act. As discussed in Chap. 1, this act lists 65 toxic substances or classes of substances subject to regulation. The EPA has developed from this information a list of 129 specific elements and chemicals that it considers to be priority toxic pollutants. The pollutants in this list differ widely with respect to their (1) chemical and physical properties, (2) behavior in the environment, and (3) health effects and behavior in humans. Moreover, our understanding of how these substances behave in power plants, the environment, and the human body varies substantially.

Our approach and models have been designed to be sufficiently general and flexible to be applied to almost any of the priority pollutants, or to other toxic materials not included in the list. The primary restriction in an analysis will be data and an understanding of how the material behaves in the environment and human body. For most of the priority list, emission and environmental data are scarce, at best, and quantitative information about chronic health effects does not exist. Before beginning an extensive analysis, one should determine that sufficient information is available to at least attempt a quantitative assessment. If health effects data are inadequate to estimate dose-response relationships, it may be necessary to do environmental transport and exposure calculations only, comparing alternatives on the basis of regional concentrations or exposure rates.

Control Technology--Cost and Effectiveness

Waste treatment and control technologies for the toxic pollutants have not been widely applied commercially in most industries. As a result, cost and effectiveness data are severely limited, even for the most common toxic materials. Only a few studies have considered the general problem of controlling utility toxic emissions (Colley et al., 1978; EPA, 1980a; Teknekron, 1976; Rice and Strauss, 1977; UWAG, 1979), and these studies have only limited quantitative information.

Utility liquid effluents differ significantly from those of most other industries. The direct discharges have larger volumes and lower concentrations of toxic substances. Many treatment processes have not been used at such low concentrations, so neither their cost nor effectiveness can be verified without more direct utility experience (Colley et al., 1978).

There is little consistency in the design of utility water management and treatment systems. The volume and composition of waste streams are determined by plant processes and design, raw materials, environmental conditions, water recirculation and reuse, control processes and equipment for stack gases, and operating and maintenance procedures, among other things. With this variability in characteristics, control costs and effectiveness for toxic pollutants will depend primarily on the specific plant characteristics and pollutant being removed. As a consequence, in these matters, we have been limited in this report to indicating which control or treatment technologies should be considered and where relevant cost information can be found.

UNCERTAINTY ANALYSIS

Quantitative risk assessment has long been plagued by the problem of uncertainty. An analysis may have little meaning and questionable value in setting regulations, if its results have an uncertainty of several orders of magnitude. Unfortunately, this may frequently be the case when determining the chronic health effects of toxic pollutants. Even if the overall uncertainty in an analysis cannot be reduced, it is incumbent on the analyst to obtain some measure of it before completing

his work. If he can also determine the primary causes of the uncertainty in his results, then it may be possible in the future to improve the analysis in these areas.

The basic cause of uncertainty in an analysis is lack of knowledge about the problem and the environment. More specifically, this lack of knowledge can be separated into four general sources of uncertainty. The first is uncertainty caused by the true stochastic characteristics of the problem. This type of uncertainty is the motivation behind probabilistic models for describing these characteristics. The second type of uncertainty comes from variations between alternative models and submodels that could be used in an analysis. These variations arise from necessary assumptions and simplifications and may reflect incomplete understanding of the physical processes involved. The third type of uncertainty arises from lack of knowledge about the correct values of parameters and inputs required for the models. Finally, the fourth source of uncertainty is the potential lack of completeness in the overall analysis. A factor whose existence is not known, or is overlooked by the analyst, will not be included in the analysis.

We must deal with the different sources of uncertainty in different ways. Treatment of the first source--the stochastic character of nature--is explicit in the risk estimation process. It is the motivation for using probabilistic models to describe certain characteristics and occurrences. Accordingly, we will not consider it further here. Similarly, the last source of uncertainty, incompleteness due to ignorance or error, cannot always be estimated or quantified. This type of uncertainty may potentially have the largest impact on risks, but we can only deal with it by (1) validating all individual models when possible, (2) being more thorough in the analysis, (3) using independent review, and (4) improving basic research into all aspects of the problem. Although we cannot deal further with these two sources of uncertainty, we can attempt to deal with the two remaining sources.

Uncertainty can arise from errors in measurements used to estimate the values of input data and parameters required by the various models. Because measurements of different parameters may or may not be independent, uncertainties in parameter values may be additive, multiplicative, or averaging. As each model operates, uncertainty in

the data and parameter values will propagate through the analysis. Our understanding of the nature of this process and its results is limited by our theoretical understanding of how to deal with uncertainty as well as by our lack of knowledge of the uncertainty associated with each input and model parameter.

Many methods have been developed and applied to deal with the propagation of uncertainty in model inputs and parameters. In addressing this source of uncertainty, we will do three things. First, by example, we will demonstrate the importance of performing a strong uncertainty propagation analysis. We do this by illustrating the wide range in risk estimates that can be obtained from a given set of data using four different dose-response models. Second, we will discuss a set of theoretical alternatives for assessing uncertainty propagation. In considering these theoretical alternatives, we base some of our discussion on the results of Cox and Baybutt (1981). We array the primary advantages and disadvantages of each method and select those we judge to be the most appropriate methods for application to toxic risk assessment. Third, we identify the steps necessary to apply this method to a case study.

One can also use our framework to assess how alternative models contribute to the overall uncertainty in results. At the various stages of the analysis--emissions, environmental transport, exposure, and dose-response--alternative models may be used in the assessment. Modeling errors cannot always be approximated, however. The normal method of judging model accuracy is by experimentation and model verification. Some models, such as environmental transport models, may have been extensively verified by empirical evidence. Thus we know something about the errors or uncertainty associated with these models. Dose-response extrapolation models, however, have generally not been verified. Therefore, we have no prior knowledge about the adequacy of these models, and model selection must normally be a subjective undertaking connected with an unknowable degree of uncertainty. In this case, we can only investigate the sensitivity of results to model choice. The problems associated with these models are discussed more completely in Chap. 6.

In our framework, source emission rates are specified by the user, and can be modified to reflect uncertainty or variability in plant materials and operating levels. In the environmental transport analysis, the user selects the most appropriate models that meet the requirements of the problem, given the constraints on the analysis. By incorporating both simple and complex models for a medium, one can determine how these models interact with other environmental models and affect intermediate outputs (media concentrations and transfer rates) and the overall health risks associated with the study. In addition to providing information about how the various models affect the risk estimates, this procedure may also allow the user to better apply his resources in future analyses to reduce the overall uncertainty.

Why Assess Uncertainty Propagation?

We can demonstrate the need for analysis of uncertainty propagation with an example from the field of toxic risk assessment. This example illustrates that even if one assumes an identical data base, the choice of model used to estimate lifetime cases of cancer influences significantly the size of the estimate. Taken from Schneiderman (1980), the data in Table 2.1 demonstrate the range of predicted risk that results from the choice of several different dose-response models fitted to the same laboratory test data. In this case the data relate to a National Academy of Science study and report on saccharin.

Table 2.1
RISK ESTIMATES FOR DIFFERENT DOSE-RESPONSE MODELS

Model	Lifetime Cancer Risk (cases/million population)
One-hit	1200
Two-stage	5
Multihit	0.001
Probit	450

We compound these divergent values when we combine them with uncertainty in input data and other stages of a risk analysis. While the extreme variation indicated in the table is not necessarily typical, it does alert us to the fact that even choice of model can dramatically influence the results of a study.

Theoretical Basis

There are a number of theoretical approaches to quantifying uncertainty associated with input and parameter values. Cox and Baybutt (1981) address five alternative methods for quantifying uncertainty; we assess their proposals and address three more. The advantages and disadvantages of each method are displayed in Table 2.2.

The Analytic Approach. The analytic approach can be applied only when an explicit formula describing an output function in terms of all input functions is given. Then specific statistics of the output function, such as mean value and standard deviation, could be described as functions of the mean and standard deviation of the input values. The uncertainty is given in closed analytic form.

Although this approach clearly has advantages when the process in question can be described analytically, its application is highly limited otherwise. In general, knowledge of the actual input distributions, and not merely their means and variances, is required. In practice, considerable computation may be required when applying the analytic approach. Toxic risk assessment may be too complex to permit an analytic description of calculations.

On the positive side, this method details output uncertainty upon which an importance ranking can be based. The method is also precise, although certainly quite unruly for more than very modest calculations.

Monte Carlo Simulation. In the Monte Carlo approach the output function is simulated directly by assigning to each of the input functions a set of joint probabilities. One takes a large number of independent samples from these distributions forming the joint probability--and calculates the corresponding outputs. The laws of probability assure that as the sample size increases, this spectrum of outputs provides a good representation of the true output distribution.

Table 2.2

ADVANTAGES AND DISADVANTAGES OF UNCERTAINTY ANALYSIS METHODS

Method	Advantages	Disadvantages
Analytic	<ul style="list-style-type: none"> o Ranks uncertainty contributors o Precise 	<ul style="list-style-type: none"> o Limited application o Expensive o May be too complex
Monte Carlo	<ul style="list-style-type: none"> o Widely applicable o Easy to develop confidence intervals 	<ul style="list-style-type: none"> o Sensitive to input assumptions o Cannot rank uncertainty contributors o Long computation times o May be too complex
Response surfaces	<ul style="list-style-type: none"> o Economical o Widely applicable o Ranks uncertainty contributors 	<ul style="list-style-type: none"> o Hard to assess accuracy
Differential sensitivity	<ul style="list-style-type: none"> o Widely applicable o Accurate o Ranks uncertainty contributors 	<ul style="list-style-type: none"> o May be difficult to compute
Confidence intervals	<ul style="list-style-type: none"> o Measures uncertainty caused by statistical variability in data 	<ul style="list-style-type: none"> o Not always applicable o Does not rank uncertainty contributors
Extreme value	<ul style="list-style-type: none"> o Gives range of uncertainty o Economical 	<ul style="list-style-type: none"> o Pointwise information o Range too broad
Linear propagation	<ul style="list-style-type: none"> o Accurate o Applied in modularized fashion 	<ul style="list-style-type: none"> o Need to model processes quantitatively o Difficult to estimate component uncertainty
Simple sensitivity	<ul style="list-style-type: none"> o Simple o Inexpensive 	<ul style="list-style-type: none"> o Cannot bound uncertainty

Results are generally presented as a histogram, developed by grouping each of the outputs into suitable categories. From this, or directly from the output table, summary statistics such as the mean and variance, as well as confidence intervals, can normally be calculated.

The principal advantage of the Monte Carlo method is that it can be applied to many different types of problems. There are no restrictions on the form of the joint input distribution or on the nature of the relationship between input and output. A second advantage is that confidence intervals for calculated quantities can easily be developed.

This method has three major disadvantages. First, it requires a large number of runs, often several thousand. Second, it may often be difficult to assess the sensitivity of results to the assumed input distributions. Changing the distribution of even one input often requires rerunning the entire calculation. Third, Monte Carlo results do not tell us which variables are the most important contributors to output uncertainty. Because uncertainty analyses are often performed in order to find effective ways to reduce output uncertainty, this is a major disadvantage. Thus, this approach does not apply to our case study.

Response Surface Approach. Response surface methods of uncertainty analysis were developed to overcome the disadvantages of the Monte Carlo approach. In the Monte Carlo approach the output function is complicated; in the response surface approach this complicated output function is replaced by an analytic approximation termed a response surface. Typically, the analytic output function is taken as a linear combination of certain simple functions, the coefficients being determined by least square fitting. Once a response surface has been fit, it may be analyzed using the analytic or Monte Carlo techniques.

The principal advantage of this approach is economy. For even very complicated functions, defined by long running computer codes with a large number of inputs, response surfaces can be fit using only a few tens of runs. The major disadvantage is the difficulty in estimating the accuracy with which the response surface approximates the original output function. This approach may be applicable to our case study and generally to risk analysis of toxic emissions.

Differential Sensitivity Approach. The sensitivity approach involves solving a set of linear equations obtained by differentiating the general output function. Once these sensitivity functions have been calculated, they must be combined with measures of actual input uncertainty to obtain an estimate of output uncertainty.

According to Cox and Baybutt (1981), differential sensitivity approaches appear to have an advantage over response surface approaches, because the sensitivity functions obtained are based explicitly on the equations linking input to output. Response surfaces, by contrast, treat the relation between input and output as a black box.

The differential sensitivity approaches have two primary disadvantages. First, in problems of practical interest, considerable analytic and code development work may often be required to set up and solve the equations for sensitivity coefficients. Second, in practice it may be necessary to evaluate sensitivity functions for several sets of input parameter values, if large variations in the inputs are to be accommodated. This leads to increased computation time.

Confidence Interval Approach. Statistical confidence bound approaches are based on evaluation of confidence intervals. They are applicable in situations where the system model is well defined in an analytic sense and when sufficient statistical data are available to estimate input parameter values. Boolean algebraic approaches such as fault trees based on pass/fail component data are typical examples of the confidence interval approach.

The advantage of this classical approach is that it provides an estimate of output uncertainty based only on the statistical variability of input data. Its disadvantages, however, do not permit the application of the confidence interval approach to our case study or similar risk analyses. First, the computations in this type of study are often not well defined mathematically--a fundamental requirement of this approach. Second, the approach fails to provide an importance ranking of input uncertainties.

Extreme Value Approach. In this approach, the analyst selects minimum, best, and maximum values for each primary input parameter, and makes three calculations. First, all parameters are given their best estimate values in sequential calculations. The second and third calculations are made in similar fashion using consistently the most pessimistic and optimistic values for all parameters. This approach obviously gives a broad range of calculated values, from totally pessimistic to totally optimistic.

To its advantage, this approach is quite simple--only three sets of calculations need to be made. On the other hand, the primary disadvantage is overwhelming. The range between purely pessimistic and purely optimistic cases may be so broad that it is uninteresting. Similarly, this range may provide no useful information for any set of calculations that is more than just modest.

Linear Propagation of Errors. The linear propagation of errors (LPE) method is based upon first-order Taylor series expansion of the calculated quantity in terms of the random input parameters. This approach permits one to calculate variance from the sensitivity coefficients and covariance structure of the input parameters.

A strength of the LPE method--used in some nuclear reactor core physics calculations of hot-spot temperatures in fuel elements--is that it may be applied in a modularized fashion. As such, it is quite amenable to multisystem risk calculations. The applicability of the LPE method is of course limited by the extent to which the uncertainties in transport and other models can be bounded. Moreover, for complex calculations, this approach may specify an unrealistically broad confidence band.

Simple Sensitivity Analysis. All of the methodologies we have discussed thus far are analytic and deterministic. That is to say, they provide a recipe of tasks that operate on a set of input data to achieve an output that has the form (more or less) of some mean value with a distribution around it. Each of these approaches thus has a shortcoming in the sense that as the number of sequential operations with uncertain data increases, the progressive outputs develop a wider and wider distribution about the mean for any fixed level of confidence. This implies that for a very complex set of operational calculations (as in our case study), we are likely to find an order of magnitude or greater of range in the output value for perhaps only a 90 or 95 percent confidence level. This problem is especially pronounced when using the extreme value approach. In addition, many of these analytic approaches are cost-intensive in terms of human resource and computational time.

What is the alternative? Given the limited resources that have been allocated to the uncertainty propagation problem in this first phase of our study, the most cost-effective method of assessing uncertainty propagation is through a simple sensitivity analysis.

Our sensitivity analysis consists basically of rank ordering each of the input parameters as a function of sensitivity on the final output, selecting a range of "reasonable" variation of these parameters, and assessing these variations one at a time. The process is then continued for sets of two parameters at a time.

While this approach fails to bound the final output, it does distinguish between the more sensitive data and the less sensitive data and tends to approximate this bound. The sensitivity analysis also tends to operate much better than most analytic approaches in an environment of restricted resources.

The Best Approach

Summarizing in part from Cox and Baybutt (1981) and interjecting additional thought relative to our problem at hand, we have concluded that the response surface, differential sensitivity, linear propagation of errors, and simple sensitivity analysis approaches are the only four methods both simple enough and sufficiently general to apply to our case study or similar complex risk analysis. Of these four approaches, only the last--the sensitivity analysis--has been applied in the first phase of our study. At this point, we anticipate that our follow-up efforts will apply at least one of the other three methods as well as more precisely detail the simple sensitivity analysis. Chapter 9 discusses the details of our analysis and considers how the sensitivity approach could be expanded in the future.⁸

⁸We recognize that sensitivity analysis is not strictly uncertainty analysis as it has frequently been defined. As we discuss in Chap. 9, however, uncertainty analysis includes an extensive range of approaches. The most rigid of these approaches attempt to develop probability distributions for the results of an analysis. Less rigid approaches try to approximate these distributions with statistical parameters such as means and variances. The least rigid approach involves developing multiple point estimates of parameters (best, low, and high estimates) and using these to bound the distribution of results. Consequently, our sensitivity analysis can be considered a somewhat informal class of uncertainty analysis.

3. POLLUTION EMISSIONS AND CONTROL OPTIONS

POWER PLANT PROCESSES

Many steam electric power plants produce electricity by burning fossil fuels--either coal, oil, or natural gas. In a coal-fired power plant, the generation process begins when coal is burned in a furnace surrounding the boiler, a large multitubed heat exchanger. As the coal burns, feedwater flowing through the boiler tubes is converted to superheated, pressurized steam. This steam passes through a turbine that drives a generator or alternator. The turbine exhaust steam passes through a condenser, where enough heat is removed to cool and condense the steam back to water. Finally, this water returns to the boiler for reuse in the cycle. Although the actual generation process is more complex than this, because of other stages that increase the overall efficiency of the plant, we do not need to discuss these modifications here. Detailed descriptions of the steam generation process can be found in Teknekron (1976) and Babcock and Wilcox (1978).

A typical fossil fuel power plant site consists generally of the following components:

- Power generation complex (boiler, turbine-generator, plant control and administration facilities).
- Air quality control equipment.
- Water treatment facilities.
- Fuel storage and handling facilities (rail siding, conveyor systems, reserve storage pile, live storage pile in a coal-fired plant).
- Waste heat dispersal (cooling towers).
- Waste handling system (ash ponds, sludge ponds, other waste ponds, handling systems).

Other facilities and equipment may be found at particular plants. The above list represents primarily those components of a plant that can generate emissions of toxic or other pollutants.

Power plants that use coal as their primary energy source can emit toxic pollutants into the air, surface water, or soil. The main emission sources are shown in Fig. 3.1, a schematic diagram of the primary processes and material flows from a typical coal-fired steam electric plant. Emissions will vary in characteristics and magnitude between plants, depending on (1) plant design and construction, (2) raw materials, and (3) operating and maintenance procedures. We will use Fig. 3.1 to discuss plant processes and emission sources in more detail later.

The combustion of coal yields an ash residue of inorganic minerals and uncombusted organic compounds. This residue may represent from 3 to 30 percent of the original coal weight, although the range for most U.S. coals is from 6 to 20 percent. The combustion ash can be roughly separated into three types: (1) bottom ash, (2) economizer ash, and (3) fly ash. Bottom ash generally appears as fused particles (slag) or loose, dry material that drops out of the gas stream in the furnace and accumulates in the furnace bottom. Economizer ash consists of the slightly less dense material that collects in the economizer hoppers beyond the boiler flue gas exit. Fly ash is the remaining material that has been entrained in the combustion gas. It may either be collected in the flue gas pollution control equipment or emitted from the stack. For reasons of simplification, we will lump economizer ash with bottom ash in all further discussion.

The partitioning of total ash between bottom ash and fly ash depends on the (1) coal type, (2) boiler furnace design, and (3) boiler furnace bottom (EPA, 1980a; Hart and DeLaney, 1978; Colley et al., 1978). Different coals melt at different temperatures and in different ways. The ash fusion temperature determines, in part, how much ash will accumulate in the furnace. If this temperature is low, the melting point of the ash may be exceeded during combustion, increasing the production of bottom ash.

Three basic types of boiler furnaces are used: the spreader stoker, cyclone, and pulverized coal. Spreader stokers produce about two-thirds fly ash but are not used in large power plants. Cyclone furnaces produce the least fly ash, generating between 80 and 85 percent

SOURCE: Colley et al. (1978).

Fig. 3.1 -- Processes, flows, and emissions for a coal-fired power plant

bottom ash. They are used more frequently, but generally in smaller power plants. Pulverized coal furnaces produce only from 20 to 35 percent bottom ash, but are by far the most common type of furnace. They account for 90 percent of the coal combustion in current power plants (EPA, 1980a).

Pulverized coal furnaces may have either wet or dry bottoms. In dry bottom processes, the ash drops through a grate at the bottom of the furnace and collects in water filled hoppers. In wet bottom processes, the ash is collected in a fused or molten state. The process used depends on the fusion temperature of the coal--low fusion temperatures require wet bottom furnaces, which produce more bottom ash than dry bottom processes.

Table 3.1 shows the general partitioning between bottom ash and fly ash as a function of the combination of furnace and bottom design. The handling and disposal of fly ash and bottom ash will be discussed later, when we describe emissions by direct discharge to surface waters.

Table 3.1
DISTRIBUTION BETWEEN BOTTOM ASH AND FLY ASH

Type of Firing	Type of Bottom	Typical Percentage	
		Bottom Ash	Fly Ash
Pulverized coal	Wet	35	65
Pulverized coal	Dry	15	85
Cyclone	--	90	10
Spreader stoker	--	35	65

SOURCE: EPA (1980a).

TOXIC POLLUTANT BEHAVIOR IN POWER PLANTS

The Clean Water Act specified 65 classes of toxic pollutants that must be regulated by the EPA. The EPA modified these classes to generate a list of 129 priority pollutants to be controlled. Unfortunately, only limited data are available to characterize these pollutants in power plant wastes. Many of the priority pollutants

are not specifically used in power plants, but appear only because of their presence in the fuel or water supply of the plant. Of the priority pollutants, only about 40 have been found or reported in the liquid discharges of steam electric power plants, and only 32 reported by two or more plants (EPA, 1980a). Priority pollutants appearing most often in liquid waste streams include antimony, arsenic, asbestos, beryllium, cadmium, chromium, copper, EDTA, lead, mercury, nickel, phenol, selenium, silver, thallium, vanadium, and zinc.

The most common priority pollutants are trace elements. These elements, or any priority pollutant, may enter a coal-fired power plant in several ways. Primary sources include (1) raw coal, (2) intake water, (3) chemical additives, and (4) corrosion of plant equipment. Of these sources, the most important is normally the raw coal supply. Table 3.2 summarizes data on typical trace element concentrations in U.S. coals.

Table 3.2
TRACE ELEMENT CONCENTRATIONS IN RAW COAL
(ppm)

Element	Minimum	Maximum	Typical
Antimony	0.2	9	1.3
Arsenic	0.5	106	5-14
Beryllium	0.2	31	1.6-2
Cadmium	0.1	65	2.5
Chromium	0.0	610	14-20
Copper	1.8	185	12-15
Lead	4.0	218	9-35
Mercury	0.01	5	0.2-2
Nickel	0.4	104	20-21
Selenium	0.4	8	2-3
Silver	0.1	2	--
Thallium	2.0	4	3.5
Vanadium	0.0	1281	33
Zinc	2.0	5350	272-329

SOURCES: Braunstein et al. (1977), Los Alamos (1976), Kuhn et al. (1980), Heit (1977).

Unfortunately, we do not understand all aspects of the behavior of trace elements in power plants. During the combustion process, the constituent elements of the coal are partitioned between bottom ash (including economizer ash), fly ash, and vapor. The fly ash and vaporized elements or compounds leave the boiler furnace in the flue gas stream. The elements in this stream may be repartitioned as the stream cools and passes through the flue gas pollution control equipment. Repartitioning occurs as vapors are adsorbed on the fly ash particles. Because smaller particles have a larger surface area per unit mass, this absorption process may concentrate some elements on these smaller particles (Klein et al., 1975a and 1975b; Smith, 1980).

The air pollution control equipment efficiently removes the larger fly ash particles but may be less efficient for smaller particles and vapors. The collected bottom ash, fly ash, and vapors (primarily SO_2) are transported to treatment or disposal sites. Uncollected fly ash and vapors are emitted through the stack to the atmosphere. Because the control processes may have differential removal efficiencies, pollutant behavior within the plant will determine (1) how much of the pollutant will be emitted, (2) the chemical and physical properties of the emissions, and (3) which environmental medium will receive the emissions (Klein et al., 1975b; Smith, 1980). Pollutants that remain in the bottom ash will enter the environment in one way. Those that vaporize and are selectively adsorbed by small particles will enter in a very different way.

Trace elements may be enriched within coal relative to their normal crustal abundances and are generally associated with the mineral fractions of the coal (Smith, 1980). Studies of their partitioning behavior during combustion have determined three classes of elements: (I) elements concentrating approximately equally in bottom ash and fly ash, (II) elements preferentially concentrated in fly ash, and (III) elements remaining in the vapor phase (Klein et al., 1975b; Coles et al., 1979; Bahor et al., 1981). Of the priority pollutants, only thallium falls into Class I. Class II pollutants include antimony, arsenic, cadmium, copper, lead, selenium, and zinc. These elements are mostly volatilized during combustion and subsequently condense on the

smaller fly ash particles. Consequently, they become relatively enriched in the atmospheric emissions. Only one studied priority pollutant, mercury, belongs to Class III. It vaporizes completely during combustion and remains in the gas phase until discharged. Several other trace elements, notably beryllium, chromium, nickel, and vanadium, appear to fall somewhere between Classes I and II (Klein et al., 1975b; Coles et al., 1979).

Clearly, many priority pollutants may be emitted from power plant stacks as particulates or gases. They may also be collected as bottom ash or fly ash and pass through the plant ash handling systems. From these ash systems, priority pollutants may eventually enter surface water or groundwater bodies. Each pollutant will behave differently within a given power plant, and predicting or measuring emission levels can be particularly difficult. How a pollutant behaves within a given power plant will be influenced particularly by (1) the chemical composition of the coal, (2) coal preparation and cleaning processes, (3) combustion process conditions, and (4) flue gas handling and treatment processes.

Many authors have attempted to generalize on the behavior patterns of particular materials (Klein et al., 1975a and 1975b; Kaakinen et al., 1975; Smith, 1980; Billings et al., 1973; Campbell et al., 1978; Davidson et al., 1974; Block and Dams, 1976; Ondov et al., 1979; Meserole et al., 1979). Most of these studies have dealt with the trace elements. Although organic compounds are known to be emitted from power plant stacks and in direct discharges, much less attention has been given to studying their behavior and effects (Natusch, 1978).

POWER PLANT EMISSIONS

Toxic pollutants can be emitted into the atmosphere, surface water, or soil and groundwater. We can best discuss specific emission sources and their characteristics by grouping them with respect to these three receiving media.

Atmospheric Emissions

Plant emissions to the atmosphere come from three sources: (1) stack gases and particulate fly ash, (2) dust from coal storage and handling facilities, and (3) dust from ash storage, handling, and disposal operations. Each of these sources has its particular characteristics and control options.

The flue gas stream from the combustion chamber includes carbon monoxide, carbon dioxide, water vapor, fly ash particles, nitrogen and sulfur oxides, and trace elements in the vapor phase. As it moves from the boiler, the gas cools, and trace elements condense on fly ash particles, as we have discussed above. The discharge of these particles and that of nitrogen and sulfur oxides from power plants are limited by EPA regulations.¹ To meet these limitations, coal-fired power plants must use certain air pollution control methods. The control technologies for particulates and sulfur oxides affect toxic pollutant emissions most. We will briefly discuss alternative methods for controlling these emissions.

Particulate (fly ash) emissions can be controlled using four basic types of systems: (1) electrostatic precipitators, (2) wet scrubbers, (3) fabric filters, and (4) mechanical collectors.

Electrostatic precipitators (ESPs) are the most common type of equipment used by coal-fired power plants. They apply an electrical charge to the particles and collect the charged particles on electrodes. ESPs have many advantages compared with other systems. In particular, they have

- High collection efficiencies (99 to 99.9 percent).

¹These regulations limit sulfur dioxide emissions to 1.20 lb/million Btu heat input, and particulate emissions to 0.10 lb/million Btu heat input for plants started after August 17, 1971. For plants started after September 18, 1978, sulfur dioxide emission limits are the same, but at least 90 percent reduction in potential emissions is required. These more recent plants must also meet more stringent particulate emission standards of 0.03 lb/million Btu and 99 percent emission reduction (CFR, 1981).

- Low pressure drops and power requirements.
- Relatively low costs and maintenance requirements (Dvorak et al., 1978).

Unfortunately, ESP collection efficiencies decrease somewhat as particle sizes decrease. Consequently, pollutants that selectively concentrate on the smallest particles (Class II) may not be removed effectively. Moreover, because of problems with fly ash resistivity, more ESPs are being used upstream of the air preheater, where flue gas temperatures may be at or above 600°F (Dvorak et al., 1978). At these temperatures, many toxic pollutants may still be in the vapor phase and will not be removed at all.

Wet scrubbers use inertial impaction, interception, and diffusion processes to remove particles with water droplets (Dvorak et al., 1977). Power plants use Venturi and moving-bed scrubbers most often, although pressure drops across these scrubbers are relatively high. Venturi scrubbers create a high-velocity flow of stack gas and inject the absorbing liquid into this stream. They have a nominal efficiency of 99 percent. Moving-bed units pass flue gases upward through a bed of plastic or glass spheres, through which the scrubbing liquid is passing downward. These scrubbers have collection efficiencies ranging between 98.7 and 99.9 percent (Dvorak et al., 1978).

Fabric filters collect fly ash by passing the flue gases through fine fabric bags. Efficiency increases as particles collect on the bags, but so does the pressure drop across the filter. These filters must be cleaned periodically and cannot be used where either temperatures or humidity are high. They are generally more expensive than ESPs but can achieve high collection efficiencies (above 99 percent) for small diameter particulates. In addition, they can operate effectively on low-sulfur coals, which produce a fly ash that can be difficult to precipitate in ESPs. For these reasons, although power plants have not had extensive experience with fabric filters, they have become a competitive alternative to ESPs in new plants.

Mechanical collectors use centrifugal forces to remove fly ash particles. They are inefficient (less than 90 percent) for small particles (less than 10 microns) and are not often used in modern power plants.

Power plants that cannot obtain or use low-sulfur coal must incorporate flue gas desulfurization systems to remove sulfur dioxide from stack gases. Many processes have been developed to do this. These processes use wet scrubbing with alkaline slurries to absorb and react with the sulfur dioxide in exiting gases. They may be used separately or combined with other stack gas treatment equipment in a power plant, depending on the characteristics of plant design, raw materials, and other factors. We will discuss these systems in more detail later.

Particulate control equipment in a power plant removes most of the fly ash particle mass but leaves the smallest particles. If the plant uses high-sulfur coal and must have sulfur dioxide controls, these fly ash particles will pass to the flue gas desulfurization system. This scrubber will remove most of the remaining particulates, including the trace elements, from the flue gas stream. The remaining discharges of toxic pollutants to the atmosphere should be small, compared to the original emissions from the combustion chamber, but may still be significant.

It is particularly difficult to determine the characteristics of stack emissions. These depend on specific chemical and physical properties of the coal and the nature of power plant processes. Toxic pollutant behavior within the power plant and pollution control system is complex and varied. Consequently, it is not generally possible to calculate atmospheric emission rates accurately or reliably. We have chosen to use calculated emissions in our case study only because we are investigating a hypothetical plant. In a real analysis, the user must have measured values of stack emissions whenever possible, to avoid the introduction of potentially serious errors.

Compared to stack emissions, gaseous and particulate emissions from coal storage and handling operations and ash handling and disposal systems should be small. These emissions should generally consist of relatively large, wind-blown dust particles. As such, they depend on

meteorological conditions and their intensity and character will vary frequently. Little work has been done on describing these emissions, and data on dust concentrations are difficult to obtain (Dvorak et al., 1977). Moreover, they can and normally should be controlled by watering, compaction, enclosure, and various handling techniques. For these reasons, we do not consider these emissions to be important outside the immediate area of the plant, although our environmental models have been selected to include them, if necessary.

Discharges to Surface Water

As shown in Fig. 3.1, many processes and operations within coal-fired power plants can lead to direct discharges of liquid wastes into surface water bodies. The volume and character of these wastes will vary greatly between plants. Referring to the figure, the potential waste generating processes or systems include the:

1. Condenser cooling system.
2. Ash handling system.
3. Boiler blowdown.
4. Metal cleaning.
5. Air pollution control (FGD) system.
6. Coal-pile and plant drainage.
7. Boiler makeup water treatment.
8. Miscellaneous waste streams.

We discuss each of these streams in more detail in App. D and in the case-study description of Chap. 8.

Not all of these systems produce waste streams at all times. Only five (condenser cooling, ash handling, boiler blowdown, air pollution control, and boiler makeup water treatment) generate wastes more or less continuously. Their volume will thus depend on plant operating levels. The remaining streams (metal cleaning, coal-pile and plant drainage, and miscellaneous systems) generate wastes intermittently. Their frequency and volume will depend in part on maintenance procedures, weather conditions, or other factors not related to plant operating levels.

No two power plants have the same water circulation or treatment systems. The various waste streams may be (1) combined for treatment or disposal in different ways, (2) recycled to other systems, or (3) nonexistent in some plant designs. Waste and water management schemes within a plant can reduce discharges and the need for end-of-pipe treatment. For example, (1) some cleaning wastes may be incinerated in the boiler furnace, (2) cooling tower blowdown may be treated and used as makeup water, (3) various streams may be used for ash transport, and (4) metal cleaning wastes can be ponded with plant ash and sludge.

Not all waste streams produce significant amounts of priority pollutants, and most priority pollutants have not been found consistently in any power plant waste stream (EPA, 1980a; Colley et al., 1978). The characteristics of plant waste streams can also vary not only between plants but over time within a plant. Causes of these variations include (1) coal composition, (2) makeup water quality, (3) plant maintenance procedures, (4) environmental conditions, and (5) plant operating levels. Calculations of plant emissions should ideally take these variations into account.

According to Hart and DeLaney (1978), the two waste streams with the greatest hazard potential are those associated with coal ash handling (fly ash and bottom ash) and flue gas scrubbing. As discussed earlier, the characteristics and volumes of bottom ash and fly ash depend on coal type and composition, plant combustion processes, and ash collection systems. Coal ash typically contains high concentrations of trace elements, including those classified as priority pollutants (Bahor et al., 1981). Ash handling and disposal systems may be either wet or dry. Wet systems may generate important direct discharges. In these systems the collected ash is mixed with water for sluicing to dewatering ponds. The overflow from these ponds may contain pollutants picked up from the ash. Air pollution control systems to remove sulfur oxides (FGD scrubbers) generate large volumes of sludge which can contain significant quantities of trace elements. Although these systems should be designed with high water recirculation rates, they may also produce liquid discharge streams containing some of these priority pollutants.

Other waste streams that are potentially hazardous include metal cleaning wastes and coal-pile drainage (Hart and DeLaney, 1978). Metal cleaning includes both chemical cleaning of boiler tubes and nonchemical cleaning of air preheaters and boiler fireside surfaces. The chemical cleaning process wastes are potentially more hazardous and are regulated separately from the nonchemical processes (UWAG, 1982). Runoff from precipitation can contain materials leached from the coal in storage piles. The volume and composition of this runoff depend on coal composition and type, coal-pile design, and meteorological conditions. Both metal cleaning wastes and coal-pile runoff are intermittent waste streams that can be collected, mixed with other wastes, and treated at the plant site.

Table 3.3 summarizes some of the primary priority pollutants that may be found in these liquid waste streams. Data are limited for many of the discharge streams, particularly for the toxic pollutants.

Groundwater Emissions

Plant emissions into the soil and groundwater occur as leaching from three sources: (1) coal storage piles, (2) ash and sludge disposal ponds and landfills, and (3) plant drainage and accidental spills. Of these sources, the third is much less important than the first two and will not be considered further in this discussion. Water and contaminants leaching into the ground are deposited in the soil over time and may move down through unsaturated sections of the soil until they reach groundwater levels. Priority pollutants that reach groundwater may be transported through the aquifers and may also subsequently be transferred into surface water bodies.

The first potential source of groundwater emissions is infiltration from coal storage piles. As discussed in App. D, runoff and leaching from storage piles can include many of the priority pollutants. Chemical reactions within a coal pile are complex and not well understood (Coward and Horton, 1980; Cox et al., 1979; Hart and DeLaney, 1978). The composition of coal-pile leachate will depend on a variety of conditions, including (1) rainfall characteristics, (2) coal properties, (3) coal preparation and cleanliness, (4) storage-pile

Table 3.3

TOXIC POLLUTANTS POTENTIALLY FOUND IN DIRECT DISCHARGE WASTE STREAMS

	Pollutant																	
Waste Stream	Sb	As	AB	Be	Cd	Cr	Cu	CN	ED	Pb	Hg	Ni	PH	Se	Ag	Th	Va	Zn
Condenser cooling																		
Once-through	x	x			x	x	x			x	x	x	x	x	x	x		x
Tower blowdown	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x		x
Ash handling	x	x		x	x	x	x	x		x	x	x	x	x	x	x	x	x
Metal cleaning{a}			x			x	x		x	x	x							x
Boiler tubes		x			x	x	x		x	x	x	x	x	x	x		x	x
Boiler fireside						x	x					x						x
Air preheater						x	x					x						x
Boiler blowdown						x	x		x			x	x					x
Water treatment{a}		x				x	x		x	x	x	x	x		x			x
Clarification						x	x			x		x	x					x
Ion exchange					x	x	x			x	x	x	x					x
Evaporation						x	x	x			x	x	x					x
Coal-pile runoff		x		x	x	x	x			x	x	x	x	x				x
FGD system	x	x	x	x	x	x	x			x	x	x		x	x		x	x
Other	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x	x

SOURCES: Knight et al. (1980); EPA (1980a); Hart and DeLaney (1978); Colley et al. (1978); Bahor et al. (1981).

NOTES: AB--Asbestos; ED--EDTA; PH--Phenol.

{a} Waste stream within category not specified.

dimensions and design, and (5) pollution and dust controls.

Pollution controls for leaching may include lining or treating the ground beneath the storage pile to minimize its permeability.

Runoff and infiltration water in general may have characteristics and effects on local soil similar to those of acid mine drainage. The amount of priority pollutants leached from a coal pile will depend on the acidity of the infiltrating water as well as the composition and characteristics of the coal (Coward and Horton, 1980). Leaching of heavy metals from coals increases dramatically as the leachate becomes more acid. Leaching is also enhanced by the presence of complexing agents (organic materials) in the water. Thus coal piles that generate leachate with these characteristics will have a greater problem with potential contamination.

Waste disposal or treatment ponds are the primary source of groundwater pollution at power plants. These ponds may be used either as interim storage and treatment facilities for wastes or as ultimate disposal locations. Under past and current practice, coal-fired power plants frequently use one or more ponds, either in series or parallel operation, to treat or dispose of (1) fly ash, (2) bottom ash, (3) FGD sludge, and (4) other wastes generated within the plant. These other wastes may include sludges and discharges from wastewater treatment processes, makeup water treatment, and metal cleaning operations.

Selection of specific treatment and disposal options depends on plant and site characteristics. Collected ash, FGD sludge, and other wastes may be sent to separate or combined ponds. In these ponds, solids settle out or stabilize and the supernatant overflow can be further treated, discharged, or returned to the plant for reuse. If the ponds are used only for interim treatment, the settled waste is periodically removed for transport to a landfill site for ultimate disposal. If the ponds also serve as disposal sites, they may be either reclaimed for development or abandoned at the end of their useful life. Typically, required land areas for pond disposal of ash and FGD sludge range from 0.1 to 0.7 acres per megawatt of generating capacity, over a 35 year plant life (CH2M, 1980; Dvorak et al., 1978).

Both vertical and lateral seepage may occur at ponds, particularly if the wastes are deposited as slurries. The properties of leachates have been reported by various groups (Bahor et al., 1979; Knight et al., 1980; Theis et al., 1978; Dudas, 1981; Theis and Marley, 1975a and 1975b; Theis and Richter, 1979), but the general problem is not yet well understood. The volume and chemical characteristics of leachate will depend on the physical and chemical properties of the effluents, site characteristics, and the specific methods used for treatment and disposal. Ponds may be either lined or unlined. Unlined ponds are cheaper to produce, but lining will be necessary if the soil below the pond is relatively permeable. Otherwise, wastes saturated with water will percolate contaminants through the pond bottom. Liners not only reduce permeability and pollutant mobility, but they also conserve water for subsequent reuse.

Ponds should be lined with an impervious material to limit seepage. Lining can vary from compacting and treating the existing bottom soil to depositing a layer of clay, concrete, asphalt, plastic, or rubber. Problems associated with these types of liners include high cost, selective permeability, and susceptibility to oxidation, cracking, and puncturing under stress. Utility experience with lining materials and procedures is still limited, especially with respect to long-term properties and durability of liners.

Landfills are used for ultimate disposal of utility wastes that have been stabilized or dewatered to a solid material that can be handled with conventional earth-moving equipment. These wastes include collected ash, FGD sludge, and other solids from treatment processes, including ponding. Leaching from disposal landfills occurs when precipitation or runoff infiltrates the material. The characteristics of this pollution depend on conditions similar to those discussed above of disposal ponds, namely: (1) waste properties, (2) site and soil characteristics, and (3) landfill design, including lining. Leaching can be controlled by properly designing the landfill to minimize infiltration into the compacted material and to reduce the permeability of the soil layer underlying the landfill.

POLLUTION CONTROL OPTIONS

Power plants can choose either one or a combination of two basic water management philosophies--once-through or recirculation. The design of plant water systems is usually based on a combination of considerations, including (1) plant processes, (2) raw materials, (3) makeup water quality and availability, (4) economics, and (5) discharge regulations.

In once-through systems plant water is not recirculated or reused. Makeup water requirements are roughly equal to discharge rates (including water lost in sludges and solid wastes) plus losses to evaporation. Discharge rates are high, but pollutant concentrations in the effluent are normally low. Because makeup water requirements and discharge rates are high, water conditioning may be large, and wastewater treatment, if necessary, is difficult and expensive.

Recirculating systems reuse fresh water within the plant, thus reducing fresh water makeup needs. Recirculation also reduces the amount of discharge accordingly, and increases pollutant concentrations in both the effluent and plant water systems. Consequently, scaling, corrosion, and biofouling are more serious problems. To control these processes, plant operators must treat the recirculating water or discharge some fraction of it in blowdown streams, or use a combination of both approaches. Discharge rates are lower and effluent pollutant concentrations higher than for once-through systems. Wastewater treatment is correspondingly less difficult and less expensive.

Wastewater handling and treatment systems in power plants must be designed specifically to work with individual plant (1) processes, (2) water systems, (3) waste streams, and (4) pollution characteristics. Although a number of wastewater management and treatment alternatives are available for the priority pollutants, their utility at power plants is uncertain for three reasons. First, power plant wastes are frequently quite different in character from those of the industries where treatment processes have been used. Second, utilities have little or no practical experience with most of the treatment alternatives. Finally, data on treatment effectiveness and cost for most of the priority pollutants either do not exist or are severely limited.

Toxic pollution from coal-fired plants can be reduced in three general ways. First, plant processes and equipment can be redesigned and modified to reduce the initial production of pollutant species. Because such redesign must consider all relevant plant processes and raw materials, it may be a complex and costly process. Second, plant materials and processes may be altered to minimize the production of offending pollutants in only the controlled effluent streams. This process should involve less significant changes in plant operations, depending on the specific circumstances. Third, wastewater treatment processes can be incorporated to remove pollutants before effluents are discharged. Because these systems can normally be considered individually, plant operators can determine the relative costs, efficiencies, and benefits of each specific technique independently. This simplifies decisions about which techniques to use in controlling emissions.

Numerous technologies have been suggested for treatment of utility wastes to control toxic effluents (CH2M, 1980; Teknekron, 1976; EPA, 1980a; Colley et al., 1978; Rice and Strauss, 1977). Discussion of these technologies has involved identifying and assessing control options that are commercially available or currently in use in some industry, not necessarily the utility industry. Unfortunately little information is available about the use of these processes within power plants, particularly with regard to:

- Priority pollutant control capabilities.
- Process interference.
- Secondary pollutant emission problems.
- Capital and operating costs.

Appendix E describes the most important of these alternative control technologies, and Table 3.4 summarizes technologies that might be used for the various utility waste streams. In considering these applications, however, we must note that the specific waste management and control procedures adopted by a power plant will depend on a variety of conditions and constraints specific to that plant. No general wastewater management scheme can be applied to any significant fraction of the utility industry.

Few of the control technologies will work equally well on a majority of waste streams or priority pollutants. For many pollutants, particularly organic chemicals, very little control information is available. In Table 3.5 we have indicated which priority pollutants might be removed from some waste streams in significant amounts by technologies suggested for the utility industry. Some of these technologies can be effectively applied only to a limited number of waste streams and then with highly variable results. In the table, organic pollutants have been grouped together because of limited information about their presence in, and removal from, most utility waste streams.

Table 3.4

POTENTIAL POLLUTION CONTROL TECHNOLOGIES FOR UTILITY WASTE STREAMS

	Technology									
Waste	R/R	C/DBF	E/SP	D/L	INC	P-C	SAC	DAH	C/F	
Cooling water										
Once-through							x			
Blowdown	A					x	x			
Sidestream						x				
Water treatment										
Clarifier			x	x						
Ion exchange										
regenerant{a}		x	x						x	
Filter backwash			x							
Lime softening										
blowdown			x							
Reverse osmosis										
brine			x							
Evaporator brine			x							
Fly ash handling										
Once-through	x		x			x		x{b}		
Blowdown		x	x			x			x	
Sidestream		x				x			x	
Bottom ash handling										
Once-through	x{c}		x	x		x		x{d}		
Blowdown	A	x	x	x					x	
Sidestream						x				
Boiler blowdown	x		x			x				
Metal cleaning										
Waterside	x{e}		x		x	x			x	
Fireside	x{e}	x	x						x	
Floor/lab drains			x							
Coal-pile runoff	x{e}	x	x					x{f}		
FGD system										
Overflow	A						x			
Purge liquors			x		x					
Prescrubber										
blowdown		x	x		x				x	
Cooling water	A		x							
Miscellaneous	x		x							

SOURCES: CH2M (1980); Teknekron (1976); EPA (1981); Colley et al. (1978); Rice and Strauss (1977).

NOTES: R/R--Reuse or recirculate; C/DBF--Clarification and deep bed filtration; E/SP--Evaporation or settling pond; D/L--Dewater and landfill;

L-SS--Lime-soda softening; INC--Incineration in boiler firebox; P-C--Physical-chemical treatment; SAC--Substitution of alternative chemicals or fill materials; DAH--Dry ash handling; C/F--Coagulation and flocculation; A--Ash transport or FGD sludge handling system.

{a} Some regenerant should be treated as metal cleaning wastes.

{b} Pneumatic fly ash handling systems can be substituted for wet handling systems.

{c} Recirculation of bottom ash handling water requires using a system of dewatering bins and settling tanks to dewater the wet-collected ash.

{d} Dry bottom ash handling systems are used only in small stoker-fired boilers.

{e} Water will normally require some treatment before being used elsewhere in the plant.

{f} Runoff composition is highly variable, but aeration and lime-neutralization treatment are frequently necessary.

Table 3.5

PRIORITY POLLUTANTS POTENTIALLY CONTROLLED BY SELECTED TECHNOLOGIES

Class of Pollutant	Technology				
	L-SS	E/SP	P-C	C/F	C/DBF
Antimony	x	x	x	x	x
Arsenic	x	x	x	x	x
Asbestos		x			x
Beryllium		x	x		x
Cadmium	x	x	x	x	x
Chromium	x	x	x	x	x
Copper	x	x	x	x	x
Lead	x	x	x	x	x
Mercury	x	x			x
Nickel	x	x	x	x	x
Selenium		x			x
Silver	x	x	x		x
Thallium	x	x			x
Zinc	x	x	x	x	x
Organic Chemicals{a}		x	x	x	x

SOURCES: Colley et al. (1978), EPA (1981).

NOTES: X--Demonstrated control; L-SS--Lime-soda softening; E/SP--Evaporation or settling pond; P-C--Physical-chemical treatment; C/F--Coagulation and flocculation; C/DBF--Clarification and deep bed filtration.

{a} Some organic chemicals can be controlled.

No specific cost-volume relationships have been developed for these suggested treatment processes for the utility industry. Treatment system designs are highly plant-specific, depending on plant combustion processes and materials as well as plant water management and use systems. Moreover, retrofit of many treatment technologies can be far more expensive and variable in cost than inclusion of these technologies in initial design and construction. Finally, because utility wastes differ significantly from those of other industries, and utility experience with many of these technologies is limited, general cost and effectiveness information has not been developed for most processes.

4. ENVIRONMENTAL TRANSPORT, TRANSFORMATION, AND FATE MODELS¹

GENERAL METHODOLOGY

Coal-fired power plants and other sources can release toxic chemicals into the environment through emissions into all media: air, soil, surface water, and groundwater. Once in the environment, these chemicals undergo complex processes of (1) transport, (2) degradation and decay, (3) transformation, (4) intermedia transfer, (5) adsorption, and (6) biological uptake. The interactions of these various pathways and their linkage to humans were discussed in Chap. 2 and shown in Figs. 2.2 and 2.3.

The environmental transport problem can be best described in terms of environmental pathways. These pathways may be either primary or secondary. Primary pathways begin at the emission source. They are associated with primarily local and acute environmental effects and produce high contaminant concentration levels. Secondary pathways, on the other hand, are those for which the contaminant is first transported in another medium before reaching the medium of interest. These secondary pathways are generally associated with more regional and chronic environmental effects, and produce low contaminant concentrations (because of dispersion and dilution occurring in the primary pathway). Typical sequential primary and secondary pathways for chemicals released through stacks, coal piles and landfills, ash sludge ponds, and direct liquid discharges to surface water are shown in Fig. 4.1.

Because complex phenomena are involved in contaminant migration and fate processes in air, water, and soil, it is difficult to assess the levels of chemical concentrations in the environment. Underestimating concentrations may cause unacceptable damage to humans and ecological systems. Overestimating concentrations and potential risks may cause unnecessary requirements for additional waste treatment to be imposed on the electric power industry. Multimedia mathematical models can

¹Most of this chapter has been summarized from Onishi et al. (1982).

SOURCE: Onishi et al. (1982).

Fig. 4.1 — Sequential primary and secondary pathways of chemicals from a coal-fired power plant

integrate such complex transport and fate mechanisms into a single framework so that potential concentrations and exposures can be more accurately evaluated.

A multimedia environmental exposure assessment methodology can be based either on a fully coupled multimedia model or a composite multimedia modeling approach. The fully coupled approach uses a model whose submodels for each pathway are integrated parts of the overall multimedia model. With this approach, each submodel interacts with other submodels. Unfortunately, the various pathway submodels generally become oversimplified to achieve understandable input data streams, reasonable computer core size, and reasonable run times. Consequently, these models frequently do not produce realistic estimates of toxic chemical concentrations in the environment. They may be useful as a preliminary screening tool for a risk assessment, but they are not well suited for more realistic evaluations of acute or chronic risks.

The composite multimedia methodology can be assembled by selecting and loosely combining appropriate models for various pathways. These individual models are coupled into a composite methodology by transferring the output file from one pathway model to the input file of the next pathway model. Specific feedback between the various media pathways, which is the primary justification for the integrated approach, can be handled by the analysis of a problem in terms of its primary and secondary pathways for each medium and the appropriate interactions of the models and their results.

With the composite approach, we can select more realistic and appropriate individual pathway models for an analysis. Because only the necessary models will be used in a given problem, and because the pathway models are replaceable, the overall methodology will be more flexible. The model structure can be tailored to each problem and analysis resources allocated as necessary to optimize the overall effort. In addition, this approach provides a framework that can accommodate future model development as it occurs.

In this study we have selected the composite modeling approach. We can describe this approach by referring to Fig. 4.1. For a typical coal-

fired power plant, we first analyze all primary pathways for each medium in a local model area near the source. The outputs from these primary analyses are fed forward as inputs to more regional models. Combining the results of the regional models with those of the more local or primary models produces the environmental concentrations and transfer rates required as a function of space and time. Occasionally it may be necessary to repeat a secondary pathway model to incorporate any significant feedback from another secondary model that was not accurately included in the initial analysis.

We can also illustrate the composite multimedia approach using an example. Consider the surface water pathway for a coal-fired power plant that uses a system of ponds for combined ash and sludge disposal. The primary emission sources for surface water contamination will be the pond overflows and other direct plant discharges. On the regional scale, several secondary pathways may be important. These include (1) deposition of atmospheric discharges to surface water, (2) overland runoff of atmospheric deposition, and (3) groundwater infiltration to surface water of pollutant leached from the disposal ponds. Each of these secondary pathways transfers to the surface water pollutant that has been emitted into and transported through at least one other environmental medium.

The secondary pathways or intermedia transfers may be either natural or caused by man. Natural transfers include:

- Wet and dry deposition from the atmosphere to the soil and surface water bodies.
- Resuspension from land to the atmosphere.
- Volatilization from surface waters to the atmosphere.
- Overland runoff into surface waters.
- Infiltration from the soil into groundwater.
- Deposition from the groundwater into the soil.
- Infiltration from surface water to groundwater.
- Seepage from groundwater to surface water.

The primary transfers implemented by man include:

- Return flows to surface water from groundwater used for irrigation.
- Return flows to surface water from groundwater used for commercial, industrial, or municipal purposes.
- Deposition in soil from surface water or groundwater used for irrigation.

When these various secondary pathways become important, they will influence the selection of models, their use, and the coupling of information between the models.

Selection of Environmental Models

To use this risk assessment methodology, one must select environmental models for each relevant medium. This selection process should consider a number of criteria, to insure that the models chosen are the most appropriate for the situation. These criteria must include:

- Type and magnitude of plant emissions.
- Local and regional environmental characteristics.
- Nature of the toxic materials of concern.
- Required spatial and temporal resolution.
- Environmental realism and fidelity of available models.

To avoid compromising the analysis, the selected models must be reasonably faithful to the environmental processes involved and must be capable of making realistic predictions of concentrations and transfer rates. Within these constraints, however, other criteria can be used to dictate the final selection. These criteria include:

- Resources and level of effort available for the analysis.
- Model data and computational capacity requirements.

- Acceptable level of uncertainty in results.
- Previous experience and abilities of analysts.
- Model availability.

Models should not be selected only because they are the best available. It is equally important that they be compatible with the requirements and constraints of the analysis and analysis personnel. The most physically realistic models may have large data requirements and large numbers of highly uncertain parameters. They may also be much more difficult to use and understand and harder to incorporate into the overall model framework. Under the circumstances, a somewhat less complex model applied with extensive sensitivity analysis may be far more useful in an analysis.

From the above discussion, it is clear that the physical characteristics of the plant, pollutant, and regional environment are particularly important. More specifically, the models selected for each medium should be able to deal with the most significant features of the physical problem, including the (1) primary and secondary sources; (2) physical, chemical, and biological processes; (3) intermedia transfers and interactions; (4) spatial scale; and (5) regional environmental characteristics. Although the spatial and temporal resolution of the models should be compatible, this is not generally possible. Most individual environmental models have not been designed for interaction with other models. Consequently, specific coupling routines and procedures for mating model inputs and outputs will be needed in any case. We discuss this problem in detail in App. C.

Although the methodology may be modified to suit the characteristics of any particular problem, we have selected a series of models that could be used in the environmental analysis. Other models may be suitable for a risk assessment, but these meet the technical requirements and have the flexibility to be applied to most types of plants, pollutants, and environmental conditions. We will briefly describe these models in the succeeding sections as we discuss each of the environmental media in more detail. Our discussion will include general considerations, important media mechanisms and processes,

reasons for selecting various types of models, and brief discussions of the recommended models. For an extensive discussion of the recommended models, the reader should consult Vol. 2 of Onishi et al. (1982), which describes each model in detail, including (1) physical, chemical, and biological mechanisms; (2) mathematical formulation; (3) input data requirements; (4) outputs; (5) applications and limitations; and (6) accuracy and uncertainty.

Model Interactions and Coupling

In the composite multimedia methodology, one must couple the individual pathway models to produce the necessary transfers and interactions between environmental media. Although the general procedures used for coupling models may be obvious, the specific details of data flow, translation, and formatting will differ for every set of models and analysis conditions. We have developed the necessary procedures to couple the environmental models selected in this study. These procedures are described in detail in App. C, but we can summarize the primary considerations here.

The generic problem of coupling one model with another can be broken down into four major areas: (1) data consistency, (2) temporal resolution, (3) unit conversion, and (4) spatial resolution. Each of these areas must be considered in any analysis, although they are closely related, and some may present more difficult challenges than the others. In all cases, we must use procedures that maintain mass and energy balances between models and converted data sets.

Data used as input to more than one model must be consistent and taken from the same source whenever possible. If spatial or temporal scales differ in the separate models, the averaging methods used must conserve mass and energy. These constraints must also be observed when converting the output of one model to form the input or source boundary conditions for another. Temporal conversion may require using specific interpolation or averaging procedures to make the time units of one model's output consistent with another model's input requirements. Similarly, in unit conversion, one must select proper conversion techniques to change units or even combine output streams from the

supplying model. This may be more difficult for spatial resolution, because the models may use different projections; coordinate system types, origins, and rotations; and grid sizes. In all of these cases, however, we must always try to maintain mass and energy balances.

Analysis Procedure and Mathematical Notation

In applying the risk assessment methodology, we partition the region surrounding the power plant into a number of subregions. These subregions are characterized by populations, production rates for animal and vegetable products, and other relevant variables. The specification of subregions will depend on the characteristics of the region and analysis, particularly the population distribution, surface water network, water sources, and agricultural production.

The final outputs of the environmental modeling are pollutant concentrations in the subregions around the plant. These concentrations are used in the exposure modeling to determine population exposure rates by intake route. Although we describe exposure modeling in the next section, we should define here some of the notation that relates to the environmental models. Because we are dealing with steady-state conditions, at least over the time period of interest, pollutant concentrations will not be specified as functions of time. Instead, we define these concentrations as

$C(i,k,r)$ = Concentration of pollutant species i in environmental medium k of population subregion r .

$BC(i,k,r)$ = Background concentration of pollutant species i in environmental medium k of population subregion r .

Pollutant species i can be defined and followed separately in the analysis, if data and computational resources permit. Otherwise, they can be combined initially (before environmental transport) or before health effects are calculated. As we discuss later, we consider five *environmental media*: (1) atmospheric gases, (2) atmospheric particulates, (3) surface soil, (4) surface water, and (5) groundwater. To calculate exposure rates, the pollutant concentrations in each of

these media must be known and specified primarily by subregion, although some exposure calculations may require different averaging or analysis techniques.

The method of obtaining these subregion concentrations depends on the specific exposure pathway. For some pathways, such as inhalation of atmospheric gases and particulates, the necessary concentrations are mean values averaged over the area of the subregion. For other pathways, including ingestion of domestic water, concentrations are defined at the point of withdrawal from the surface or groundwater body. Finally, some additional concentration values are specified not by subregion but rather by point of use (recreational use) or waterway (consumption of aquatic organisms). As a consequence, the results of the environmental modeling will have to be appropriately averaged, converted, or otherwise manipulated to provide the information needed by the exposure program.

ATMOSPHERIC TRANSPORT

General Considerations

Atmospheric models are used in the analysis to calculate the transport and deposition of a pollutant. The primary outputs from these models are atmospheric concentrations of the pollutant and deposition rates, both wet and dry. Atmospheric concentrations determine human exposure through direct inhalation. Deposition rates determine how much of the pollutant emitted to the atmosphere reaches the soil, surface water, and groundwater. Because the overall analysis is directed toward chronic effects, the atmospheric transport models must produce long-term average values of concentrations and deposition rates. Consequently, they will most likely operate with the assumption of steady-state conditions.

In general, the atmospheric models must address the problems of transport, diffusion, and deposition. In addition, acceptable models must also consider the potential types of emissions, scales of motion, and the area of coverage. The following section will discuss these conditions and criteria for model selection in more detail.

Types of Emissions

Two primary types of emissions may contribute toxic substances to the air. These are stack emissions (flue gas and fly ash) and particulates or gases released from coal storage piles and ash landfills. Generally, coal-pile and landfill emissions will be controlled and significant only in the immediate area. We have included them, however, because they might become important sources under some circumstances.

Atmospheric Transport

During transport through the atmosphere, a pollutant will be advected, dispersed, and removed. These processes determine the deposition rates and resulting concentrations over space and time. If the emission is from a stack, the buoyancy of the plume will move the pollutant vertically until stabilization takes place. It is especially important to model this feature in the region near the plant, because the effective stack height, atmospheric stability, and settling velocity will control when the plume will reach the ground. Once the plume intercepts the ground surface, depletion from dry removal processes will begin. The fraction removed by this dry deposition is important, because it will accumulate on the ground until precipitation occurs. When precipitation takes place, wet deposition to surface water or land will occur, and runoff and percolation processes will become important. Moreover, such precipitation can also remove accumulated dry deposition.

For coal- or ash-pile emissions, other effects must be considered. The transport and fate of particulate emissions will depend on coal properties, particularly coal size and moisture content, and wind speed. Although the same advection, dispersion, and removal processes discussed above will apply to these emissions, the particle size distribution becomes more important. Large particles will be deposited near the emission source, but smaller particles (less than 10 microns) may be carried significant distances and subsequently contribute to the overall health risk.

Coverage Area and Scales of Motion

Two scales of motion should be considered: (1) mesoscale--the area within 50 km of the plant, and (2) regional scale--the area from 50 to 2000 km from the plant. Different types of models are used to evaluate the effects of atmospheric processes for these two scales. In mesoscale transport, local winds and terrain are dominant in determining the atmospheric flow characteristics. Here also, variations in plume concentration and deposition must be determined with finer resolution than is appropriate on the regional scale. The regional scale model may be used to deal with the problems of regional distribution of toxic pollutions and acid rain.

Mesoscale transport will also be appropriate for emissions from coal piles and ash handling or disposal facilities. These emissions will be primarily particles suspended from the surface and subsequently deposited downwind. Generally, 90 percent of the suspended particles from coal piles are between 9 and 77 microns in diameter (Blackwood and Wachter, 1978). This size range precludes a major deposition effect beyond 50 km, because gravitational settling and deposition velocities of these particles would remove most of them from the air within 50 km of the source. Although information is very limited, we would suspect that ash handling and disposal dust would have similar characteristics.

Basis for Model Selection

Combining the two scales of motion with the two types of emissions yields three combinations for which models must be selected, as we need not be concerned with regional transport of dust emissions. These three cases are (1) mesoscale transport of stack emissions, (2) regional scale transport of stack emissions, and (3) mesoscale transport of coal and ash emissions.

The models selected for these cases must meet certain criteria. As we have discussed, they must use long-term time averaging. Furthermore, for all cases the models should be able to represent pollutant transport and diffusion through the atmosphere. Observed winds or the compilation of wind data in a joint frequency table of wind speeds and direction

versus stability could also be used. These criteria eliminate the simple deterministic models. Similarly, the models should incorporate both vertical and horizontal diffusion through the use of a Gaussian plume or other technique that represents the vertical and horizontal spread of the plume.

The models for all cases should be able to calculate both wet and dry deposition rates. It is important to be able to separate deposition into wet and dry fractions. Such separation is necessary for estimating the amount of runoff in the overland analysis and the soluble fraction of pollutant deposited in precipitation.

Moreover, for case (1), the model should incorporate plume rise. For tall stacks, maximum dry deposition of gases and particles may occur far from the source. The model must therefore incorporate both stack height and the additional rise due to the plume buoyancy to yield an effective stack height.

Finally, for cases (1) and (2), the models should be able to deal with chemical reactions and transformations. Although this ability will not be required for all pollutants, it may be important for some of them.

Model selection in an analysis should thus be based on these technical criteria, the appropriate case or cases to be studied, and other criteria including (1) model data and computer requirements, (2) user resources and abilities, (3) site and pollutant characteristics, (4) model verification, and (5) allowable uncertainty in the results. Additional information about the alternative types of atmospheric transport and air quality models can be found in Drake et al. (1979). These reports describe in detail the features and characteristics of a large number of alternative models and discuss the use and application of these models to different types of problems.

We based our choice of recommended models on the previously described selection criteria and the three cases of interest. For all models, we assumed that the averaging time was long (greater than one month), so that deposition processes can build up surface pollutant concentrations. This is necessary because the surface water and groundwater models require steady-state concentrations and transfer

rates as inputs. This requirement eliminates the need for models specifically designed for producing hourly, three-hour, and 24-hour air concentration averages.

None of the selected models include terrain adjustments for deposition. For cases where terrain effects are significant, it may be necessary to reevaluate the models used. Based on the above cases, model criteria, and time averaging requirements, no one model was found to be acceptable. Instead, we chose a number of models. These recommended models are presented and described in the next section.

Recommended Models

This section briefly describes the models recommended for the three cases defined above. More complete descriptions of these models can be found in Onishi et al. (1982) and the cited literature.

Regional Scale Model. In the regional study case, we have recommended the Regional Air Pollutant Transport (RAPT) model. It meets the basic criteria set up for model selection and has been well documented (McNaughton and Powell, 1981). This model, a product of developmental research sponsored by the U.S. Department of Energy and the U.S. Environmental Protection Agency, has been used in a number of case studies involving acid rain, but it can also be used to evaluate trace element emissions. Statistical verification of the output with observed data has been performed (McNaughton et al., 1981). When applied to long-term studies from a source, it will provide a basis for separating the deposition into wet and dry fractions as input to the surface and groundwater models. A scaled-down version of this model could be applied to the mesoscale.

The objectives in developing this model were to simulate the transport, transformation, and removal of pollutants from anthropogenic emissions over regional scales. The initial modeling efforts were limited to studies of the fate of sulfur oxides, but the scope of studies has increased to include fine particles and oxides of nitrogen with an emphasis on understanding the acid pollutant deposition problem.

The RAPT model is a Lagrangian model that operates with reference to two fixed rectangular grids nested in the National Meteorological Center (NMC) octagonal northern hemisphere grid (Jenne, 1970). The

grids are an advection grid used for wind data input and transport calculations and a sampling grid used for input of precipitation data and calculations of pollutant concentration and deposition.

Mesoscale--Nonreactive Particles (Stack). The model recommended to represent nonreactive particles emitted from the stack is ANDEP (Vaughn et al., 1975). This model has been used in an environmental impact analysis to calculate wet and dry deposition around coal-fired power plants (Watson, 1981). ANDEP satisfies the criteria established for case (1). Used with an appropriate source term, ANDEP could be altered to estimate the transport, diffusion, and wet and dry deposition of particles suspended from coal piles and ash handling operations.

The current version of ANDEP is a refinement of work reported in Vaughn et al. (1975). The modifications were designed to make the model run more efficiently and to make the output simpler to use. Modules have also been added to account for hot plume rise and mixing height limitation. The model divides the region surrounding a pollutant source into 16 sectors, and calculates air concentrations and wet, dry, and total deposition in each sector using climatological data. The climatological data needed included joint frequency distributions of wind speed and direction by atmospheric stability class, and precipitation amounts by wind direction.

Mesoscale--Reactive and Nonreactive Gases and Particles (Stack). The model recommended to represent wet and dry deposition of both reactive and nonreactive particles and gases is the Source-Transport-Receptor Analysis Model (STRAM) (Hales et al., 1977). STRAM satisfies most of the criteria, except that plume rise is not explicitly incorporated. Instead, an effective stack height is used. A parallel model, SMICK, used in scavenging studies, has been applied to a number of case studies of precipitation scavenging of power plant plumes (Dana et al., 1975; Drewes and Hales, 1981).

STRAM is a user-oriented computer program that provides computed estimates of concentrations and removal rates in airborne, multicomponent, reactive plumes. The model can incorporate multiple sources on a regional scale. This makes it possible to calculate ground-level pollutant concentrations from a superposition of a number of plumes originating at different geographical locations.

We must mention two potential problems associated with using STRAM. First, the model should have an updated washout subroutine to use hourly precipitation in calculating wet deposition rates. Second, STRAM provides calculated plume distributions at a given instant of time. These distributions correspond to a snapshot of plumes as seen from a satellite. Such distributions would have to be computed for successive increments of time, and a time series of these distributions averaged to obtain the climatological pollutant concentration distributions.

Mesoscale--Nonreactive Particles and Gases (All Sources). The Industrial Source Complex (ISC) model is recommended for situations where gases and particulates may be emitted from coal piles and ash handling operations as well as from a stack. This model has been written to handle both point (stack) and area sources. It satisfies all the criteria, except it must be modified to handle wet deposition.

ISC is an assessment dispersion model, which consists of two submodels, a short-term model (ISCST) and a long-term model (ISCLT). ISCST uses hourly data to calculate three-hour and 24-hour values of air concentration. ISCLT is a sector-averaged model with smoothing. It uses seasonal or annual joint frequency weather tables (wind speed and direction by atmospheric stability class) to calculate seasonal or annual air concentrations. However, the short-term model (ISCST) can be used for annual assessments.

These two models can be applied to either individual or combinations of point, area, and volume sources. The models are designed to calculate air concentration or deposition, but not both at the same time. A complete description of the ISC model can be found in Bowers et al. (1979).

The Need for Multiple Models

Multiple models are needed to compute deposition and concentrations for a coal-fired generating plant because no one model can meet the criteria for all combinations of scale and type of emission. The characteristics of the four recommended models are summarized in Table 4.1.

Table 4.1

CHARACTERISTICS OF RECOMMENDED AIR MODELS

Emissions	Scale	Stack Effect	Horizontal Transport	Diffusion	Wet Deposition	Dry Deposition	Output
RAPI							
SO ₂ , SO _x , and trace contaminants from stacks	Regional, 50 - 2000 km (variable user-specified)	Effective stack height	Gridded winds Hourly time step used	Vertical but no horizontal (assumes variation in horizontal transport over long periods will represent diffusion)	Hourly precipitation used with a removal coefficient SO ₂ is 5 m ⁻¹ and SO ₄ is 1.75 x p.625 where p is the precipitation in inches/hour	Dry deposition velocity Tapes of (1) air concentration (μg/m ³) (2) wet deposition (μg/m ²) (3) dry deposition (μg/m ²) (4) total deposition (μg/m ²) (5) pH At user-specified time interval, i.e., monthly, seasonal, or annual	
ANDRP							
Particles released from stacks	Mesoscale, 1 - 50 km (variable user-specified)	Plume rise - Briggs (1969)	Joint frequency tables of wind speed, direction, and stability Monthly, seasonal, or annual tables used	Gaussian	Scavenging coefficient equal to 1.43 x E _p .75 where p is precipitation in mm/hr and E is the scavenging efficiency Uses climatological values of precipitation vs. wind direction	Dry deposition velocity Printer plot and file of: (1) air concentration (μg/m ³) (2) wet deposition (μg/m ²) (3) dry deposition (μg/m ²) (4) total deposition (μg/m ²)	
STRAM							
Particles, gases, reactive and nonreactive, from stacks	Mesoscale, 1 - 50 km (variable user-specified)	Effective stack height	Gridded winds Hourly time step used	Gaussian	Alterations to program needed to use hourly precipitation with washout coefficient (irreversible scavenging)	Dry deposition velocity Tapes of all air concentrations (μg/m ³) and deposition (μg/m ²) At user-specified time interval, i.e., monthly, seasonal, or annual	
ISC							
Nonreactive trace contaminants from stack coal pile, ash handling, and storage	Mesoscale, 1 - 50 km (variable user-specified)	Plume rise - Briggs (1971, 1975)	Joint frequency tables of speed, direction, and stability Monthly, seasonal, or annual tables used	Gaussian with correction for settling velocity	None	Dry deposition velocity Tapes of all air concentrations (μg/m ³) or deposition (g/m ²)	

SOURCE: Omlshé et al. (1982).

RAPT could be modified to be used for the mesoscale case. However, it would have to be modified to incorporate a settling velocity distribution for large particles, plume rise, representative deposition velocities, and wet removal before it could be used for all criteria, emissions, and time and distance scales. ANDEP can be used only for nonreactive particle deposition on the mesoscale. The program would have to be extensively rewritten before it could consider reactive gases and large particles with significant settling velocities or be used for regions beyond 50 km from the source. STRAM could be used on the regional as well as mesoscale but would have to have a wet removal subroutine. Because many parallel models already have such wet removal in their codes, this would not be difficult. Modifications would also be required to incorporate an area source such as a coal pile and to incorporate gravitational settling for large particles. ISC was developed for use in determining air quality on the mesoscale, but it does include a method for computing dry deposition rates. Major revisions of the code would be necessary to include wet deposition with hourly precipitation, dry deposition representative of the range of particle size needed, and reactive gases. ISC can calculate concentrations and deposition within 1000 m of the power plant. This area can not be covered by the other models.

OVERLAND TRANSPORT

Background and Model Selection

Processes controlling runoff, soil erosion, and contaminant transport on land surfaces are continuous, although the bulk of contaminant migration may take place only through storm events that produce runoff. To model these processes we must use a continuous simulation model. This model must be selected not only to simulate toxic pollutant transport during a storm event, but also to simulate (1) processes that change the moisture content of the soil and (2) events that influence soil and contaminant conditions between storms. Moreover, the model must be able to model the migration of both dissolved and particulate (those absorbed to the soil) phases of the contaminant.

One overland contaminant-transport model meeting these criteria is the Agricultural Runoff Management Model (ARM) (Donigian et al., 1977; Donigian and Davis, 1978). ARM has been extensively tested and used throughout the United States (Donigian et al., 1977; Donigian and Davis, 1978; Donigian and Crawford, 1976a). The documentation for understanding and using the model is excellent. The program is small enough to be handled efficiently, yet it considers many of the important mechanisms related to contaminant migration. These mechanisms include snowmelt, absorption/desorption, degradation, sediment transport, mineralization, nitrification/denitrification, immobilization, and nitrite leaching, among others. ARM is easy to use and inexpensive to run. A more complete description of the model can be found in the earlier references or in Onishi et al. (1982).

Several other models are as sophisticated as ARM and could be used in its place. These models have not been selected for the analysis because they fail to meet all the requirements as well as ARM.

ACTMO (Agricultural Chemical Transport Model) (Frere et al., 1975) is an agricultural chemical transport model based on the USDAHL-74 (Holtan et al., 1975) hydrologic model. It has not been completely validated, however, and documentation of the complete model is sketchy, at best. Application based on available reports would be difficult (Overcash and Davidson, 1980).

Bruce et al. (1975) have developed a parametric model for water-sediment-chemical runoff in single storm events. This model incorporates a number of functions to represent the most important physical processes, but does not consider plant nutrient concentrations (Knisel, 1980).

TEHM (Terrestrial Ecosystem Hydrology Model) (Huff et al., 1977) has been incorporated into the UTM (Unified Transport Model), forming the Wisconsin Hydrologic Transport Model (WHTM) (Patterson et al., 1974). These codes are all part of the UTA (Unified Transport Approach) developed at Oak Ridge National laboratory (ORNL) (Huff et al., 1977; Patterson et al., 1974). WHTM is a lumped parameter simulation code that represents water and trace contaminant movement and storage within a watershed. It includes simulation of the atmospheric and groundwater

environment as well. The model has not been extensively tested or used as much as ARM. Moreover, because this model includes atmospheric and groundwater modeling components, updating or changing it could prove to be difficult and costly.

McElroy et al. (1976) have developed a simple loading function to compute the transport of contaminants attached to sediment. The loading function assumes that the amount of contaminants delivered to the stream is directly proportional to the sediment load. This is a deterministic model, using analytical solutions of algebraic equations, and the calculations can be performed on a hand calculator. However, loading functions do not represent individual processes within the study area and thus provide for poor spatial resolution. They are more useful as "first-cut" estimates in overland modeling (Overcash and Davidson, 1980).

Two new codes recently introduced to the contaminant-transport modeling community are the HSPF (Hydrological Simulation Program-Fortran) (Johanson et al., 1980) and CREAMS (Chemical, Runoff, and Erosion from Agricultural Management Systems) (Knisel, 1980) models. HSPF is a systems model based on ARM, NPS (Nonpoint Source model) (Donigian and Crawford, 1976a), and HSP (Hydrological Simulation Program) (Hydrocomp, 1976, 1977). The HSPF software is not merely a translation of the above models but a new system with a framework accommodating a variety of simulation modules. It is designed to allow the user to update any mechanism or physical phenomena not considered state-of-the-art. CREAMS was developed by over 60 scientists, researchers, and technicians contributing ideas, direction, and data. It is a "physically-based, daily simulation model that estimates runoff, erosion/sediment transport, plant nutrient, and pesticide yield from field-sized areas" (Knisel, 1980).

Because both of these models are new, they may contain errors in computer formulation under certain conditions. Even if the individual components of the models have been checked, the complete code may still contain mistakes. Extensive simulations of either model by various groups have not yet been published, which makes their overall documentation deficient.

Unlike these alternative models, ARM includes all major mechanisms for contaminant transport, has been used extensively, and is well documented. For these reasons, we recommend ARM as the model for simulating the migration of contaminants overland.

For additional information, the user should refer to one or more of the published summaries of existing models. Overcash and Davidson (1980) present an excellent summary of 14 overland contaminant transport models. Brandstetter (1977) presents a comprehensive review of all major runoff models. Brown et al. (1977) present standardized summaries of many urban, nonurban, and receiving water models. A U.S. Environmental Protection Agency document (EPA, 1976) discusses eight terrestrial runoff models considered operational. Finally, a U.S. Forest Service document (USFS, 1977) presents a current summary of nonurban models.

Model Description and Application

This section provides a general description of the ARM model. For more information about the major component programs, input data requirements, output, applications and model accuracy, sensitivity, and limitations and uncertainties associated with the model, see Onishi et al. (1982).

ARM simulates runoff (including snow accumulation and melt), sediment, pesticides, and nutrient contributions to stream channels from both surface and subsurface sources. It is applicable to watersheds with uniform cropping and management practices up to 2 to 5 km², although the limiting area will vary with climatic and topographic characteristics,

ARM is a large, relatively complex computer program comprising 15 major subroutines written in the FORTRAN IV language. It was originally developed and tested on large IBM computers at Stanford University but can be used on relatively small computers. ARM comprises several submodels that simulate the overland migration of contaminants and considers (1) hydrologic processes, (2) sediment loss, (3) pesticide adsorption and desorption, (4) pesticide attenuation, and (5) nutrient runoff and transformation. The model can be used to simulate single

events and short or extended time periods. The actual time period of simulation will depend on the information needed for the analysis, but should be long enough to overcome the effect of initial conditions.

GROUNDWATER TRANSPORT

Background

As discussed earlier, we can define the primary pathways for any media as those pathways that begin at the contaminant source. Similarly, secondary pathways are those for which the contaminant is first transported in another medium before the contaminated stream reaches the secondary medium. For groundwater, primary pathways consist of direct infiltration from (1) ash and sludge disposal ponds, (2) ash and sludge landfills, (3) coal storage piles, and (4) hazardous chemical spills. These primary pathways can act as secondary pathways for surface water and land models. For groundwater secondary pathways consist of infiltration from (1) surface water, (2) fly ash or coal dust deposited from the air, and (3) fly ash, ash, or coal dust transported as runoff.

Analysis of the groundwater pathway requires three modeling technologies: (1) source term, (2) groundwater flow (both unsaturated and saturated), and (3) contaminant transport. Source term models predict the rate that a contaminant enters the primary groundwater flow system from the liquid pathway or from leaching (from a solid phase) via infiltrating waters. Groundwater flow models define the convective flow field and provide estimates of water velocity fields or flow paths and travel times. These serve as inputs to the groundwater contaminant transport models. The transport models calculate the migration of contaminants through the saturated and unsaturated rock and soil matrices.

Contaminant transport is governed by several mechanisms, including (1) convections, (2) dispersion, and (3) retention. In addition to these transport mechanisms, the models should be able to consider chemical or biological breakdown of the contaminant. These activities must be superimposed onto the transport mechanisms, because such breakdown can reduce contaminant concentrations to negligible levels. We will discuss each mechanism in more detail.

Convection is the movement of contaminants with the average velocity of the groundwater. Its description requires the knowledge of the flow pattern within the geohydrologic system. The flow pattern can, in turn, be influenced by temperature and other changes affecting the density of the fluid (e.g., brine concentration).

Dispersion is the combination of molecular diffusion, which can occur even when there is no flow, and hydrodynamic dispersion. Hydrodynamic dispersion is caused by variation in the local velocity of water in the medium. This variation is with respect to its average value as described by the convection. The velocity variation exists at any scale, from microscopic (in the pore) to macroscopic (caused by heterogeneity of the medium) to megascopic (caused by large-scale variations in rock properties, such as fractures). Dispersion causes mixing, as well as longitudinal and transverse spreading (with respect to the flow direction) of the transported elements.

Under conditions of no flow, molecular diffusion is the only mechanism generating movement of elements through the rock or soil mass. However, if temperature gradients exist, thermal diffusion (Soret effect) can increase this migration.

Retention is a general term covering all types of interaction that can take place between the transported contaminants and the rock or soil matrix of the geohydrologic system. Some of these interactions are (1) filtration, (2) molecular diffusion of elements into immobile water such as a dead-end fracture, (3) ion exchange (electrostatic binding of ions on the surface of the solid), (4) chemical reactions between contaminants (other elements transported in the water and the rock matrix), (5) precipitation and dissolution, and (6) flocculation. Some physical mechanisms (such as filtration and diffusion) can be treated separately for each contaminant, because they are independent. Other processes, generally chemical (for example, ion exchange reactions and precipitation) must be modeled simultaneously for all contaminants present in the water, as these mechanisms can interact or compete with each other.

The basic retentive phenomena are often not specifically distinguished in models. Their total effects are instead empirically represented by a constant ratio between the amount of contaminant retained on rocks or soil and the amount in solution. These distribution coefficients (K_d s) are defined either per unit mass of the soil or rock or by unit area of fractures. The use of K_d s implies that each element migrates independently in a unique chemical form and that retention is instantaneous and reversible. Recent improvements have introduced a kinetic factor in the modeling of distribution constants.

Although, using the K_d approach for modeling retention creates some uncertainty, combining a number of different types of mechanisms into one lumped parameter, it allows one to calibrate to relatively gross observations. It avoids introducing into the analysis many separate processes that may not be well understood, and for which observations may not be available. Moreover, if each potential retention mechanism were identified and modeled precisely, the data and computational requirements would be tremendous, especially for chemical speciation and chemical reactions.

To apply the K_d approach, the analyst must have the following data: (1) Eh, pH and the natural geochemical equilibrium of the water in the medium, (2) complete mineralogic description of the rock or soil, and (3) chemical reaction constants, including spatial variations in the medium. Because of the complexity and interaction among the various retentive mechanisms, K_d values must be selected carefully if this simpler approach is used in a modeling effort. When selecting K_d values used in a transport modeling effort, the user must be careful to (1) factor in the changes in geochemical properties that occur along the predicted path of the contaminant migration, (2) account for possible interaction between migrating contaminants, and (3) account for any other unmodeled mechanisms or effects. Clearly, the model must be carefully calibrated and the results investigated with sensitivity analysis on the K_d values.

One might improve this modeling approach for organic chemicals by applying techniques developed for chromatographic separations. In this way, once the migrational characteristics of some chemicals were known,

the behavior of similar chemicals could be predicted more reliably. Although this method might be appropriate for purely chemical or physical retention processes, predictions about biologically influenced retention would be much more uncertain and subject to significant error.

Selection of Groundwater Codes

Any modeling effort requires some simplifying assumptions to bridge the gap between reality and our current knowledge or understanding of the system being modeled and practicability. In most modeling fields, certain basic simplifying assumptions are routinely accepted, whereas others require justification based on data and observations. In the set of equations that describe groundwater flow and transport, much of the modeling effort and associated assumptions have already been made (Bear, 1972). These assumptions are generally accepted in the field of groundwater hydrology. The equations we write to describe an aquifer system will be for a fluid flow continuum in porous media, or for a porous media equivalent flow for a fracture flow dominated system. We must note that it is not sufficient for a mathematical model to be based on a sound set of equations that describe the physical system. The model must also be based on technically sound hydrologic information and reasonable simplifying assumptions.

The mathematical equations that describe the groundwater flow and transport pathway can be solved with a variety of methods. These solution methods for equations in 1, 2, or 3 dimensions include:

- Analytical solutions.
- Finite differences or integrated finite differences.
- Finite elements.
- Method of characteristics (particle in cells).
- Direct simulation of convective and dispersive transport.

Each solution method has a variety of models available in almost every degree of dimensionality. In general, however, the numerical methods (finite difference, finite elements, method of characteristics, and direct simulation) offer the greatest range of applicability. In terms of dimensionality required, one must investigate the specific

needs of the problems to be solved, since unnecessary use of higher dimensionality results only in higher computational costs.

Analysis of the groundwater flow and transport pathway requires modeling of the unsaturated zone and the saturated zone. Flow and transport in the unsaturated zone is typically dominated by vertical movement. As a result, one-dimensional numerical models are adequate for the majority of problems when infiltration over a large area must be modeled. This type of analysis is sufficient to describe infiltration of contaminated waters that may be associated with coal-fired power plants. For these reasons, a one-dimensional, finite-difference model, UNSAT (Gupta et al., 1978; Gee and Simmons, 1979; Simmons and Gee, 1981), was selected for use in modeling unsaturated groundwater flow.

A real groundwater system is, of course, a three-dimensional system, including precipitation percolating from the surface through unsaturated soil into the uppermost saturated aquifer. In some cases along rivers or streams or at the base of lakes and ponds, the aquifers are discharging into or being recharged from these surface water bodies. These conditions formulate one type of boundary condition for our mathematical model.

The units we call separate aquifers are really water saturated layers in the soil and rock matrix that make up the earth's crust. These units are generally more permeable than the geologic units directly below them and sometimes above them. As a result, the water in an aquifer tends to flow in a horizontal direction along the bedding plane of the more permeable geologic formation, because the resistance to flow is less. The less permeable (aquitard) or impermeable (aquiclude) layers below and sometimes above the aquifer materials retard or completely block the vertical flow.

These less permeable layers are designated as the base and top of the aquifer unit. When a more permeable layer exists below the upper aquifer unit's base, another aquifer may exist. When the aquitard material between aquifers is somewhat permeable, water can transfer between aquifer units, depending on the water potential or pressure in the units. Many currently used groundwater models make a simplifying assumption that transforms this three-dimensional system to a layered two-dimensional system with interaquifer transfer via a potential-driven

leakage term. The mathematical model that uses this set of simplifying assumptions is the multiaquifer formulation of the Boussinesq equation. For these reasons the multilayer two-dimensional finite-difference VTT (Variable Thickness Transient) code (Kipp et al., 1976; Reisenauer, 1979; Bond et al., 1981) has been selected to simulate the saturated groundwater flow pathway.

Consistent with the selection of the two-dimensional VTT code for saturated groundwater flow and the one-dimensional UNSAT code for unsaturated groundwater flow, we have selected the two-dimensional MMT (Multicomponent Mass Transport) transport code (Ahlstrom et al., 1977) and the one-dimensional MMT1D transport code (Washburn et al., 1980). Both of these codes use a direct simulation approach to model transport in the groundwater system. Other alternative models for groundwater flow and contaminant transport have been summarized and tabulated in Nelson (1977).

The selected models are described in detail in the original references and in Onishi et al. (1982). The discussion in Onishi et al. (1982) includes data requirements, outputs, applications, limitations, advantages, and disadvantages.

SURFACE WATER TRANSPORT

Background

Because this investigation emphasizes surface water pollution and its control, the surface water model may be the most important. Surface waters will receive toxic emissions from the power plant both directly and indirectly. These paths include (1) direct discharges (primary source), (2) deposition from the air, (3) transport by runoff after deposition on land, and (4) seepage from groundwater. Although these last three paths (secondary sources) may be less important than direct discharges in most cases, at plants with limited or zero discharge, they may be significant. Moreover, these secondary sources can contribute to the measured background pollutant levels at the plant intake, and thus must be investigated.

The surface water transport model should provide as output the pollutant concentrations as a function of location and time in the surface water network and infiltration rates into adjacent groundwater aquifers. Although we are concerned with long-term effects, monthly or seasonal average concentrations might be appropriate in some cases, particularly when the health effects data warrant this type of resolution or flow variations are large and frequent. Acceptable surface water models could produce either steady-state or time-dependent output. Because the boundary conditions for the surface water model will be constant over the period, time-dependent models should reach near steady-state conditions fairly quickly.

After pollutants have entered a fresh water body, numerous physical and chemical mechanisms will affect their transport and fate. Initially, toxic substances will be dispersed by currents and local turbulence. Pollutants in dissolved form may be adsorbed from solution onto sediments or removed by aquatic organisms. Pollutants associated with particulate matter may settle out and accumulate in bottom sediments. These processes reduce the concentrations in the surface water, but create deposits that become long-term pollutant sources through desorption or resuspension processes. Sediments may continue to be deposited or may be scoured and transported downstream during subsequent periods.

To assess the migration, accumulation, and fate of toxic pollutants in surface water bodies, mathematical models must include the primary contaminant transport, transformation, and fate mechanisms. In particular, the models should consider:

- Convection, dispersion, and diffusion of dissolved and particulate contaminants.
- Chemical decay and degradation from volatilization, photolysis, oxidation, and hydrolysis.
- Biological decay and degradation.
- Adsorption/desorption reactions with sediments.

- Deposition/scouring of sediments.
- Sediment transport.
- Contaminant release and contributions from outside the point and nonpoint sources (secondary sources).

The type of model selected may depend also on the type of water body being considered, as well as the basic assumptions behind the analysis. Surface water bodies in this study are limited to fresh water rivers and lakes (both shallow and stratified) and do not include oceans or estuaries. For some pollutants and water bodies, it may not be necessary to use a model that incorporates all of the above mechanisms. In many of the less complex models, the removal mechanisms may be combined into single removal or equilibrium coefficients.

Many different water quality and sediment transport models are available. These models vary in level of sophistication from simple analytic solutions of mass-balance equations to numerical solution models for multidimensional equations involving sediment interactions and transport of dissolved and particulate pollutants. The fundamental equation of pollutant transport is the continuity equation describing concentration changes over time as a function of advection, dispersion, decay, and sources or sinks of the substance. These equations may be solved in one, two, or three dimensions, depending on the model. Increasing the number of dimensions improves the ability of a model to deal with complex hydrodynamic systems, but at the cost of substantially higher data, resource, and computational demands. For relatively simple rivers, one-dimensional models may be adequate for risk analysis, although they should be able to deal with the important physical and chemical processes that affect pollutant concentration.

Selection of Surface Water Models

Many existing surface water models can deal with sediment and toxic chemical transport (Hoffman, 1977; Onishi et al., 1980b). Of these models, we have selected three programs that should cover the spectrum of problem geometries and necessary levels of sophistication.

The EXAMS (Exposure Analysis Modeling System) code is the simplest of the three approaches to contaminant transport modeling. The program was developed to predict the transport and transformation of potentially harmful chemicals in fresh water, is thoroughly documented (Smith et al., 1977), and has been widely distributed by the EPA. EXAMS is a steady-state chemical toxicant model that uses an n-compartment description of the aquatic ecosystem under consideration. The program does not use a rigorous representation of water, sediment, and chemical transport. Instead, the transport of water, sediment, and contaminant by convection and diffusion between compartments is modeled by user-specified pathways and rates.

The emphasis of the EXAMS code is on the modeling of chemical behavior in the water column and bottom sediments. Among the mechanisms considered in the model are (1) speciation of chemicals; (2) adsorption of dissolved contaminants by sediment and organic material; (3) desorption from sediments and organic material into the water column; (4) chemical degradation through volatilization, photolysis, hydrolysis, and oxidation; and (5) biological degradation by bacteria.

The EXAMS code allows a wide range of flexibility in laying out the compartments used in the model. Consequently, it can be applied to a large variety of surface water geometries, including streams, ponds, and stratified lakes. Because it uses simplified representations of many physical mechanisms, EXAMS should be used primarily for long time periods (i.e., years) where problem dynamics are not important.

For more complex situations, we have selected two models. TODAM (Time-Dependent, One-Dimensional Degradation and Migration model), an unsteady one-dimensional sediment and contaminant transport model, is designed for river and estuary applications (Onishi et al., 1981). TODAM is a simplified version of SERATRA (Sediment and Radionuclide Transport), an unsteady two-dimensional (laterally averaged) sediment and contaminant transport model (Onishi et al., 1980a). Of these models, SERATRA is the most physically explicit. These two models and the EXAMS program are discussed in more detail in Onishi et al. (1982).

TODAM simulates the transport of the sediment and contaminants (radionuclides, heavy metals, pesticides, and other toxic chemicals) in rivers and estuaries. It consists of three submodels coupled to include the effects of sediment-contaminant interactions such as adsorption/desorption, and transport, deposition, and resuspension of contaminated sediments. These models not only calculate distributions of sediment and contaminant concentrations in water but also predict river bed conditions.

The SERATRA code is an unsteady, two-dimensional sediment-contaminant transport model that uses the finite element computation method. The model has general convection-diffusion equations with decay and sink/source terms having appropriate boundary conditions. Similar to TODAM, SERATRA consists of three submodels coupled to include the mechanisms of sediment-contaminant interaction. The submodels describe (1) sediment transport, (2) dissolved contaminant transport, and (3) particulate contaminant transport.

SERATRA can be applied to nontidal rivers, river-run reservoirs, and possibly impoundments. Without modification, it can model shallow water bodies over large longitudinal distances. Although it has not been applied to lakes, we believe that the vertical two-dimensional computational procedure is well suited for lake application.

5. EXPOSURE MECHANISMS AND PATHWAYS

INTRODUCTION

In this chapter we will discuss the procedures to be used in determining the rates at which local populations are exposed to toxic pollutants. We describe the exposure pathways and rate calculations in general terms and relate these to the environmental transport analysis discussed previously. The specific equations for all exposure pathway calculations are presented in App. F.

Our objective is to calculate total exposure by intake route for each population group in each subregion around the power plant. This calculation involves summing the exposure from each separate pathway contributing to the three intake modes: inhalation, ingestion, and dermal contact. We may also want to consider in this summation monthly, seasonal, annual, or longer-term changes in exposure rate. Such changes may, in general, be caused by (1) environmental conditions, (2) normal demographic processes, (3) the evolution of plant emissions and pollutant concentrations over the life of the population, and (4) changes in human uptake as a function of age.

The change in size and age of the population through the plant life may be important in an analysis. Incorporating such changes is exceedingly complex and must generally be handled with simplifying assumptions. In our analysis, we will also use a measured or predicted population distribution by age and location at some representative time during the life of the power plant and assume that this remains static throughout the exposure period. In future work we hope to be able to relax this assumption.

Because we are dealing with steady-state models, our exposure rates will be long-term (monthly, seasonal, or annual) averages that must be summed over the life of the power plant or the individual, whichever is most appropriate. We must also consider the additional pollutant that individuals receive from pathways not affected by the power plant. Some of this exposure will be automatically incorporated into the calculations by specification of background concentrations in the

different environmental media. The remainder is not considered specifically in the environmental transport analysis but must be added during the exposure calculations. Such sources include foods and beverages not originating in the area and consumption of other products not included in the analysis. It may be difficult to estimate these consumption rates, because few people have studied the problem, and then only for a limited number of pollutants (Heit, 1977; Murthy et al., 1971; Mahaffey et al., 1975). However, for calculations of incremental risk, this exposure will remain constant between cases.

Overall risk will be a function of the distribution of population with respect to age, sex, health, and other factors. For most pollutants, virtually nothing is known of the variations in risk that are associated with these factors. The detail to be used in any given risk analysis must be consistent throughout the analysis, to avoid unnecessary cost and time. Accordingly, if health effects are not well understood, particularly for variations in age, sex, or other variables, then it may not be necessary to consider these variations in the population model.

Exposure rates are calculated as a function of location around the plant. The regional population is divided into subregions, and we have assumed that the subregional population will be uniformly exposed to all sources for the subregion. This assumption simplifies the calculations but raises potential problems. For example, many people will not necessarily work in the same area in which they live and will be exposed to different air pollution levels. Moreover, the dietary habits of people vary considerably, and some elements of the population in a subregion will consume far higher levels of contaminated food or water than others. We can list more examples, but these serve to demonstrate the point. Techniques can be devised to overcome the inherent limitations of this uniform exposure assumption, and these should be used whenever they seem appropriate.

Exposure by subregion is calculated differently for the various alternative pathways. Direct inhalation, ingestion, and dermal contact with air pollutants can be calculated using the appropriate regional air pollution concentrations. Ingestion and dermal exposure to treated surface water and groundwater can be calculated knowing the relative

consumption of surface and groundwater in each subregion. Dermal exposure to untreated surface water will come from recreational activities. Here we assume that the entire regional population uses all potential locations uniformly and equally, unless specific data allow the user to allocate recreational activity more accurately.

Food products produced within a subregion may be distributed to other areas for consumption and will normally be supplemented by food produced outside the region. For food products produced within a region, we calculate the appropriate pollutant concentrations by subregion. We then combine these concentrations using the production rates in each subregion to calculate overall mean concentrations for the region. These average concentrations can be applied to the entire regional population, assuming uniform spatial distribution of consumption.

Regional production and consumption of a product will be adequate to serve the needs of some fraction of total regional population. If this fraction is greater than 1.0, based on mean consumption rates, we assume that the entire population derives its total consumption of the product from regional sources. If the fraction is less than 1.0, all production will be used locally to meet the appropriate fraction of regional consumption. For crops or animal products that are shipped outside the area, we can assume that regional production is part of the total U.S. production and scale consumption rates accordingly.

POPULATION AND SUBREGION SPECIFICATION

As we discussed in the previous chapter, the region surrounding the power plant must be partitioned into subregions, based on the constraints of the analysis and available information. The specification of subregions will depend on the characteristics of the region and analysis, particularly the population distribution, water consumption, and agricultural production. The same subdivisions should be used for population as for other exposure calculations.

Population must be specified for exposure calculations through each pathway. The specification will be a function of the distribution of total population in time and space around the site, as well as the particular subgroups of overall population that can be considered.

Initially, we suggest subdividing total population into four basic groups, denoted by the subscript m. These groups are

- Infants (0 to 1 year).
- Children (1 to 11 years).
- Teenagers (11 to 17 years).
- Adults (17 years and older).

No distinction has been made here between sexes in these age groups. Sex separation or partitioning on the basis of health characteristics, income, race, or other variables should be made where either exposure rates or health effects vary in a known manner and may be important to the analysis. Otherwise, such population subdivisions are generally unwarranted and only complicate the analysis.

The distribution of population subject to exposure can be found from atlases, census data, environmental or safety reports, and regional sources including colleges and universities. Divisions should be made to minimize either the data collection or analysis effort, consistent with the available health effects information, and considering the primary media and exposure pathways. In environmental impact studies and safety analysis reports for nuclear facilities, population subregions are defined by concentric circles with uniformly increasing radii, segmented with diameters at 22.5 degree intervals. Although arbitrary, this definition can help standardize and simplify the environmental transport and exposure analysis in many cases.

Census data should be used to determine regional (subregional) population distributions, but they may not always be available, properly disaggregated, or appropriate for a specific case. Census data may not give the population distribution by age group with the necessary spatial resolution, for example. Moreover, census information will not indicate travel and work patterns or the presence of particularly susceptible population groups, such as the children in schools, the elderly, hospital patients, and others. In some cases the user may have to make simplifying assumptions to specify this information. However, whenever

possible, local government agencies or educational institutions should be consulted for appropriate information.

Analysis boundaries used in the exposure calculations should be based on the results of preliminary environmental transport calculations. If air and water pollutant concentrations fall off quickly with distance from the power plant, a smaller regional area can be used. If, however, concentrations remain relatively constant, the exposed area must be increased. Preliminary calculations for various exposure routes may be necessary to define the exposed group, if normal population distributions and geographical factors do not automatically define a reasonable limit.

CALCULATION PROCEDURES

As we discussed in the previous section, the passage of pollutants through the various transport models will generate concentrations of pollutant species in space and time in the various media. The media used in the exposure and transport analysis are

- Atmospheric gases.
- Atmospheric particulates.
- Surface soil.
- Surface water.
- Groundwater.

Concentrations in these media will include both background levels and contributions from the power plant emissions to all media. Pollutant species reaching the exposed population may not correspond to species emitted by the plant. These transformations may be more important in some cases than others, but should be considered within the environmental transport models. The exposure calculations are based on the final concentrations of pollutant species found in the environment. Under normal circumstances, an analysis would probably consider only one aggregated pollutant species, because environmental behavior and health effects of individual species are generally not well understood.

No concentrations have been specified for subsurface soil. The soil, runoff, and groundwater models may calculate these concentrations during the analysis. However, the results do not directly affect exposure rates and are therefore omitted from the analysis.

EXPOSURE PATHWAYS

We have assumed that exposure rates are a function of pollutant species, population group, and population subregion. Total exposure can be classified in terms of how the pollutant enters the body. The three alternative mechanisms are inhalation, ingestion, and dermal contact. We can calculate exposure rates for all important pathways and collect them into these three general classes. We then determine chronic health effects of each pollutant in terms of the specific effects associated with each mode of intake.

Pollutants entering the body will be assimilated, excreted, transformed, and distributed throughout the body. In general, some fraction of the pollutant will be retained within the body and may be concentrated in various tissues and organs. Each toxic material will behave differently, depending on (1) its physical and chemical nature, (2) the exposure intake mode and pathway, and (3) the general health characteristics of the individual. Because understanding of the processes and behavior of most toxic materials is extremely limited, we cannot generalize the pharmacokinetic analysis in the methodology. Ideally, one would prefer to use pharmacokinetic parameters to calculate absorbed pollutant doses for each exposure pathway, and to determine the body burden for those pollutants that accumulate. Unfortunately, for most of the toxic pollutants, the pharmacokinetic behavior and necessary parameters are not known.

The more common means of calculating risk from the various exposure paths is to separate the intake modes. Epidemiological studies and animal experiments normally consider only one type of exposure pathway. The dose-response relationships derived from this information therefore relate health effects to either inhalation, ingestion, or dermal contact exposure. Each intake mode has its commonly accepted units and means of expression. Inhalation exposure is expressed not in terms of the total

amount of pollutant inhaled, but rather as an average inhaled concentration over the life of the individual. Consequently, exposure might be given in units of mg/m^3 of air. Ingestion exposure is typically expressed as an average pollutant concentration in total food consumption. It may thus be expressed in units of mg/kg , averaged over the entire diet. Dermal contact is less well understood and probably much less important for most toxic materials of interest. Some exceptions, such as pesticides and similar organic compounds, may be important. Here exposure rates may be calculated as a pollutant concentration in the contact medium, averaged for continuous, whole-body exposure and expressed in such units as mg/l .

By developing the exposure calculations in this manner, we isolate exposure determination from the calculation of health effects. This provides some flexibility in the procedure, for the dose-response calculations can consider the varying pharmacokinetics of different pollutants, without having to accept the assumptions that would be necessary for a uniform or more integrated exposure rate procedure.

In our approach, we have assumed that exposure rates are constant over the period of calculation, be it monthly, seasonal, or annual. For many pathways, such as inhalation and ingestion of food and water, this is reasonable. For others, such as dermal contact through water recreation, it is clearly not accurate. However, in the absence of specific information about how true exposure rates and health effects vary between assumptions, we chose this simplifying alternative.

Although not every pathway will be important in an assessment, we have incorporated procedures in our methodology to calculate exposure rates for the following specific routes:

- Inhalation of gases and particulates.
- Dermal exposure to atmospheric gases and particulates.
- Dermal exposure to untreated surface water.
- Dermal exposure to treated surface water.
- Dermal exposure to treated groundwater.
- Ingestion of atmospheric particulates.

- Ingestion of treated surface water.
- Ingestion of groundwater.
- Ingestion of aquatic organisms.
- Ingestion of vegetation.
- Ingestion of animal products.

In some analyses, pathways other than those considered above may be important. Such exposure routes should be analyzed whenever necessary, using site-specific data. No specific criteria have been developed to indicate when a potential pathway must be considered. One alternative might be to include any pathway that contributes at least 10 percent of the total exposure from other pathways under normal conditions.

In the following sections we will discuss the general procedures and assumptions used for each pathway. The specific equations included in the analysis are presented in App. F.

Inhalation

Toxic materials may be present in the atmosphere either in a gaseous state or as particulates. How these materials behave in the environment and the human body depends in part on their physical state. Inhaled gases and particulates do not pass completely into the lungs and remain in the body. Some pollutant will be immediately exhaled, and some particulates will collect in the mouth, nasal passages, and throat, to be subsequently ingested. Moghissi et al. (1980) propose that (1) 25 percent of particulates will be exhaled, (2) 50 percent will be deposited in the upper respiratory tract, and (3) 25 percent will be deposited in the lower respiratory tract. These fractions are only rough approximations, because the particle size distribution will strongly affect the absorption of inhaled particulates and the proportion ultimately ingested. Whenever possible, pollutant-specific data should be used instead of the above default values.

In general, inhalation rates vary primarily with age and workload around a set of mean values, such as those assumed in WPPSS (1973b) and Moghissi, et al. (1980). Those studies cite mean inhalation rates (in m³/day) of 3.9 for infants, 10.1 for children, 21.9 for teenagers, and 21.9 for adults, with a weighted mean of 20.0. These rates are not necessary for the inhalation exposure calculations but must be used in determining exposure from ingested particulates.

As discussed earlier, we do not attempt to calculate absorbed doses of pollutants at this stage but rather express inhalation exposure rates as a total pollutant concentration in the atmosphere. This concentration is the sum of gaseous and particulate fractions and depends on the population subregion and chemical species, but not the population group. We realize that ideally one would like to incorporate pharmacokinetic parameters and specific inhalation rates to determine absorbed doses for different segments of the population. Although this approach may be adopted in future work, the current limited understanding of health effects and apparent variability between different pollutants would make it too difficult to implement in the normal analysis.

The pollutant concentrations in air come directly from the atmospheric transport model calculations. These concentrations should be averaged over the time period and subregion of concern. In calculating health effects, the user should determine the average exposure rates over the life of the power plant, if emissions and concentrations change in the long run. The exposure calculations assume that populations remain within their subregions at all time, and that no dilution or concentration of the pollutant occurs within buildings. These assumptions are necessary to simplify the analysis. Although some studies have used complex schemes to incorporate these variations (Johnson and Paul, 1981), these require extensive data collection and analysis that is probably not warranted in a generalized risk assessment methodology. Moreover, these types of variations can be considered specifically in sensitivity analysis to determine if additional work is necessary.

Dermal Contact Pathways

Dermal Exposure Rates. Dermal exposure can arise from contact with either atmospheric pollutants or contaminated surface and groundwater. As described earlier, we have expressed exposure rates in terms of mean pollutant concentrations in the appropriate medium, air or water. The concentrations are adjusted to account for fractional body area and duration of exposure.

The specific exposure rate for atmospheric pollutants is given by the product of the atmospheric pollutant concentration, the exposure time, and the fraction of body area exposed. This rate is then averaged over the year. Because it depends on atmospheric gas and particulate concentrations, the exposure rate for atmospheric contact varies by subregion. Although it would also vary with population group, in most cases the data required to implement this level of analysis would not be available. If necessary, as discussed above, sensitivity analysis could be used to investigate how such variation would affect the results.

For dermal contact with surface and groundwater, the exposure equations can be divided into recreational exposure to untreated surface water and domestic exposure to treated surface and groundwater. Exposure rates for recreational activity are a function of the length of the exposure period, pollutant concentrations in the recreational water, and the fraction of body area exposed. This last factor will depend on the type of activity. To calculate the appropriate pollutant concentrations, the user must determine the locations of water recreation, some measure of the relative usage of these locations, and the pollutant concentrations at each site (from the surface water analysis). We assume that all regional inhabitants participate equally and distribute their activities between all locations. Sensitivity analysis can be used to vary these assumptions to determine how exposure might change for light or heavy users at the various locations.

Exposure from dermal contact with domestic water supplies depends also on the exposure time, pollutant concentration, and type of exposure (fraction of body area in contact with water). Pollutant concentration in a subregion depends on the domestic water source (surface or ground) and the type of treatment processes used. We assume that the water

supplies for a subregion will be mixed and that the entire population of that subregion is exposed to the mixed supply. Variations on these assumptions can be investigated easily with sensitivity analysis, if important.

Domestic Water Treatment. Because domestic water treatment processes can remove significant fractions of dissolved and suspended pollutants, we must specifically incorporate these processes in the analysis. Many processes can be used to treat drinking water after extraction from either surface or underground sources. The removal efficiencies for these processes can vary significantly, as can the extent of treatment. Surface water normally receives more extensive processing than groundwater, which frequently receives very little treatment. Common water treatment processes include coagulation, filtration, chlorination, and lime softening. Removal of toxic elements from raw drinking water supplies will depend strongly on a number of conditions, most notably:

- Chemical characteristics of the pollutant species.
- Physical form of the pollutant (dissolved or suspended) in the water.
- Concentration of the toxic pollutant in the water supply.
- Drinking water source characteristics.
- Details of the water treatment processes used (chemicals, pH, temperature).

Sorg (1978), Sorg and Logsdon (1978), Sorg et al. (1978), and Sorg (1979) discuss the various treatment processes as they might be applied to different chemicals, particularly toxic elements considered in this analysis. In applying a risk assessment procedure, the user must either determine treatment efficiencies using these types of references, or find site-specific data for his particular pollutant and treatment situation. The exposure methodology permits the user to specify a number of treatment types and their efficiencies for each particular pollutant species considered.

Ingestion Pathways

Consumption Rates. When calculating exposure through ingestion pathways, we must know appropriate consumption rates for the regional population. In general, site-specific data should be used whenever possible. If these are not available, the analysis must be based on nominal, age-specific rates developed by other sources. Moghissi et al. (1980) list recommended default consumption rates by population group for animal and vegetable products, aquatic organisms, and drinking water. These rates, drawn from NRC (1977), are presented in Table 5.1.

Alternative consumption rates have been measured and reported in numerous other studies (WPPSS, 1973b; USDOA, 1980; Mahaffey et al., 1975). The rates used should be appropriate to the area under study and the particular circumstances of the analysis. For comparison with the data in Table 5.1, Table 5.2 presents consumption and other rates for the eastern Washington area, the region used for our case study.

These tables make it clear that consumption rates as defined by different sources can differ significantly, emphasizing the importance of using regional information whenever possible.

Table 5.1
MEAN AND MAXIMUM CONSUMPTION RATES BY POPULATION GROUP

Mean (Maximum) Consumption Rates				
Pathway	Infant	Child	Teenager	Adult
Drinking water (l/yr){a}	(330)	260(510)	260(510)	370(730)
Fruits, vegetables, grain (kg/yr)	--	200(520)	240(630)	190(520)
Milk (l/yr)	(330)	170(330)	200(400)	110(310)
Meat & poultry (kg/yr)	--	37(41)	59(65)	95(110)
Leafy vegetables (kg/yr)	--	--(26)	--(42)	--(64)
Fish (kg/yr)	--	2.2(6.9)	5.2(16)	6.9(21)
Seafood (kg/yr)	--	0.33(1.7)	0.75(3.8)	1.0(5)

SOURCE: NRC (1977).

{a} Includes soft drinks, beer, and similar beverages.

Table 5.2

MEAN AND MAXIMUM CONSUMPTION RATES FOR WESTERN WASHINGTON

Pathway	Mean	Maximum
Drinking water (l/day){a}	1.2	2.0
Fish (local) (kg/yr)	0.056	40.0
Recreation (hr/yr)		
-shore	17.0	500.0
-swimming	10.0	100.0
-boating	5.0	100.0
Milk (l/yr)	137.0	274.0
Eggs (kg/yr)	15.0	30.0
Meat (kg/yr)		
-beef	20.0	40.0
-pork	20.0	40.0
Poultry (kg/yr)	9.0	18.0
Leafy vegetables (kg/yr)	15.0	30.0
Grains (kg/yr)	44.2	88.3
Vegetables (kg/yr)	51.0	102.0
Potatoes (kg/yr)	55.0	110.0
Fruit (kg/yr)	147.5	295.0
Melons (kg/yr)	10.0	40.0

SOURCE: WPPSS (1973b).

{a} Includes soft drinks, beer, and other beverages.

Ingestion of Drinking Water. Exposure through domestic water supplies can be either by ingestion or dermal contact. We have previously discussed dermal contact for domestic water and how water treatment processes can affect pollutant concentrations. Exposure through consumption of drinking water can be calculated from pollutant concentrations in the water supply, water treatment efficiencies, and consumption rates for drinking water.

As we have noted, the domestic water supply in any subregion may be composed of either surface water or groundwater or a combination of both. Pollutant concentrations for each subregion source should be taken at the withdrawal point and modified by the removal efficiency appropriate for the type of water treatment that the source undergoes. The final concentration for the subregion is a weighted mean value based on the relative volumes of each source. This procedure assumes that the

entire subregion population consumes a mixture of water supplied to the area by each source. In many cases, we would expect that the entire domestic supply for a subregion would come from one primary source, thus simplifying the analysis.

In recent years, beer, soft drinks, and other beverages have accounted for an increasing fraction of total water consumption. If these products are not locally produced, the analysis should take this into consideration. Water intake rates can be reduced accordingly and the difference included as background pollutant from nonregional sources.

Ingestion of Atmospheric Particulates. A fraction of inhaled particulates will be ingested by each individual, thereby passing through the gastrointestinal system. Total exposure for this pathway can be calculated from an extension of the expression for inhalation exposure, incorporating inhalation rate data into the analysis. We must use inhalation rates for this calculation because ingestion exposures are expressed in terms of quantity ingested, not external concentration of pollutant. At the completion of all ingestion calculations, this ingested quantity is converted to an equivalent concentration in the overall diet.

We calculate ingestion of atmospheric particulates for the population in a subregion from the atmospheric particulate concentration in that subregion, inhalation rates, and a coefficient that specifies the fraction of inhaled particulates that is ingested. This fraction will vary with the pollutant as well as the particle size distribution in the air, so measured values should be used if possible. Lacking the necessary data, a default value of 0.25 can be assumed.

Consumption of Vegetation. Accumulation of trace elements in vegetation depends on the plant species, pollutant, and soil characteristics. Transfer between soil and plant, and transfer within the plant, are complex problems. Pollutants may reach the plant by deposition from the atmosphere, irrigation, use of pesticides, or uptake from groundwater. Pollutant deposited from the air may (1) remain on plant surfaces, (2) be washed off leaves onto the soil, or (3) be partially taken up and incorporated into the plant structure. Pollutants within the surface soil may be taken up by plants, carried off

into surface water, infiltrated into groundwater, or retained. The fraction following each pathway will be affected by many conditions, particularly pollutant and soil characteristics, plant structure (including root behavior), local temperatures, and precipitation.

Within a plant, pollutants behave in different ways. Some elements (arsenic, beryllium, chromium, lead, nickel, and vanadium) accumulate normally in the roots, whereas others (cadmium, copper, and zinc) can freely move to leaves and other edible portions of plants (Dvorak et al., 1978). This variation in behavior makes generalization extremely difficult and forces the analyst to consider the specific characteristics of his pollutant and local environment (including soil and hydrologic conditions) very carefully.

Bioaccumulation or bioconcentration occurs in plants but varies by species and pollutant. It depends on soil properties, plant health, and water availability, among other factors. We can define a concentration ratio between the mean plant and soil concentrations. This ratio can vary with soil concentration, plant characteristics, mineral or element availability, and type of chemical, and gives no information about where the pollutant resides within the plant (edible or nonedible portions).

Total toxic element concentration on plants will be composed of the amount within the edible portions of the plant and the amount deposited (still remaining) on the leaves. These contributions must be determined separately. For purposes of calculation, we assume that these pollutant sources are independent.

Pollutants will accumulate on plant surfaces from wet and dry deposition of atmospheric particulates as well as from irrigation with polluted surface or groundwater. If conditions are reasonably stable, an equilibrium will be reached between deposition and weathering rates taking place over the period that plant surfaces are exposed to the air or to irrigation water. The resulting concentration on plant leaves will depend on the (1) deposition rates, (2) fraction of deposition captured by a plant, (3) surface density of plants, and (4) weathering loss rate.

The atmospheric deposition rate will be the average subregion deposition calculated by the atmospheric transport model used in the analysis. The deposition fluxes for surface water and groundwater

irrigation can be calculated from known or assumed irrigation rates for the region and plant species and the pollutant concentrations in the appropriate type of water. All rates can be expressed in terms of days, to simplify the calculation of exposure times and weathering losses. As with all other calculations, specification of crop areas, irrigation rates, and other variables should be as aggregated as possible--within the requirements of the analysis--to minimize data collection and interpretation efforts. It should be possible to make preliminary simplified calculations of plant concentrations and exposure rates to indicate how much aggregation is acceptable.

The contribution to plant concentration from pollutant taken up from the soil can be calculated from surface soil pollutant concentrations and the bioconcentration factor appropriate to the pollutant and plant. Surface soil concentrations will come from the overland model in the environmental transport analysis or from separate simplified calculations. The needed bioconcentration ratios may be more difficult to obtain. Because they can vary widely, as discussed above, site-specific information should be used. If not available, the user must rely on values of plant-soil concentration ratios given in the literature. These ratios have not been consistently defined and measured, causing published values to vary significantly between sources. One must be particularly careful to understand the measurement techniques and assumptions behind stated values. Table 5.3 illustrates the variation that can be found in the literature.

For each vegetable product, we must determine pollutant concentrations in all appropriate subregions, then calculate a mean concentration for all of the product produced in the region. If total regional production equals or exceeds regional consumption, we assume all consumption is from regional sources. Exposure can then be found from the specific consumption rates for each product and the mean concentration in the product. If regional production does not exceed regional consumption, we must include externally grown produce with an assumed background pollutant concentration.

Certain simplifying assumptions can be made to calculate total exposure to the population through consumption of vegetable products. The primary assumptions are that all supplies of a product grown in a

Table 5.3
PLANT-SOIL CONCENTRATION RATIOS

Element	Concentration Ratio		
	{a}	{b}	{c}
Arsenic	4.2	0.14	0.03
Beryllium	16	0.02	0.03
Cadmium	222	10.7	5.3
Chromium	250	0.02	0.01
Copper	1000	0.47	--
Lead	2	0.45	2.3
Mercury	26	0.02-0.5	0.05
Nickel	331	0.045	0.05
Selenium	4	1.0	1.0
Vanadium	1	0.01	0.008
Zinc	40	0.64	--

{a} Dvorak et al. (1977).
 {b} Dvorak et al. (1978).
 {c} Braunstein et al. (1977).

region are well mixed, consumed within the region (up to total regional consumption), and distributed throughout all subregions. This should produce an upper bound on the net risk estimates in our analysis. Although we have assumed that consumption rates are uniform throughout each defined population group, this can be investigated with sensitivity analysis. It may sometimes be informative to consider individuals or groups with assumed maximum consumption rates, or other variations in consumption patterns, particularly where large amounts of home-grown produce are consumed by individuals near the power plant site.

Total exposure is calculated by summing over all local vegetation consumed directly by humans. In most cases there will be changes in concentration caused by processing of the product by the producer or preparation by the consumer. It would be extremely difficult to incorporate the effects of this processing, so we have assumed it does not reduce pollutant concentration in the vegetable product. This is probably a worst case assumption, as processing would normally reduce the total amount of pollutant reaching the consumer.

The general procedure that should be followed in this type of analysis can be summarized as follows:

1. Determine primary products produced and consumed in the region.
2. Specify distribution of production by subregion.
3. Determine which crops are irrigated with surface water or groundwater and estimate irrigation rates.
4. Specify concentrations of pollutant in irrigation water using results of groundwater and surface water transport models.
5. Calculate total deposition fluxes by crop and subregion using atmospheric deposition (from the air models) and irrigation rates.
6. Calculate surface soil concentration by crop and subregion using the overland model outputs.
7. Calculate pollutant concentrations by crop and subregion.
8. Aggregate each product and distribute uniformly across the region. Compare local production and consumption to calculate exposure rate by crop. Sum exposure rates over products.

If information indicates that crops are consumed by only a subgroup of the overall population, this group should be considered separately from the remainder of the population.

Ingestion of Aquatic Organisms. In some areas, consumption of aquatic organisms may be an important source of exposure. The Environmental Protection Agency has specified water quality criteria based on this pathway, both separately and combined with the consumption of polluted drinking water (EPA, 1980c). Aquatic exposure pathways may include consumption of many different species of fish and shellfish.

Aquatic organisms can accumulate or concentrate toxic materials in many ways, including absorption and adsorption and metabolic processes. These complex processes depend on species characteristics, feeding habits, and environmental variables such as water temperature and salinity (Moghissi et al., 1980). In general, it is not possible to investigate each of the potential uptake mechanisms and the variables that may affect it. Instead, scientists make use of a concentration-

factor approach, similar to that discussed previously for vegetable products. These concentration or bioaccumulation factors, relating the pollutant concentration in the organism to that of the surrounding water, are derived from experimental data for the pollutant of interest or similar materials.

Bioaccumulation factors ideally should be known for each combination of pollutant species and organism. Unfortunately, existing data are limited to a few organisms and pollutants. Unless specific studies have been conducted in the region of interest, the analyst will probably have to use default values for the bioaccumulation ratios. Typical literature values for some pollutants and types of aquatic species are shown in Table 5.4.

The EPA has calculated its water quality criteria using national average consumption rates and bioconcentration factors (EPA, 1980c). Consumption rates can depend strongly on location, however, and national averages may not be particularly useful in a risk analysis for a particular plant. Consequently, regional production and consumption data should be used whenever possible.

Table 5.4
BIOACCUMULATION FACTORS FOR FRESH WATER BIOTA

Element	Fresh Water Fish	Benthos	Invertebrates
Antimony	1	--	10
Arsenic	333	2,000	333
Beryllium	2	--	10
Cadmium	200	--	2,000
Chromium	40/382/4,000	40	20
Copper	200/4,000	200	1,000
Lead	300	--	100
Mercury	1,000	5,185	100,000
Nickel	100	--	100
Selenium	167	5,783	167
Silver	2	--	769
Thallium	10,000/100,000	--	15,000
Zinc	1,000/8,500	875	10,000

SOURCES: Dvorak et al. (1977); Dvorak et al. (1978); Heit (1977).

To calculate exposure, one must determine the primary aquatic species consumed in the area, local production rates (annual catch), and the mean pollutant concentration in the surface waters where the aquatic species are found. It should be possible to make reasonable assumptions about the distribution of organisms (total catch) in regional surface waters. The concentrations calculated by the surface water transport model can then be appropriately averaged to find the necessary concentrations.

Following the general assumptions and procedure used for ingestion of vegetable products, we assume that consumption is distributed uniformly across the region, differing only by subgroup within the population. Again we must compare regional production and consumption rates. When production exceeds consumption, we can assume all consumption is locally produced and determine exposure rates accordingly. Exposure can be calculated by multiplying the surface water concentration by the bioaccumulation factor and the consumption rate appropriate to the population group under consideration. As before, this calculation is done for each important species and the results combined to determine overall exposure rates for aquatic organism ingestion.

If regional consumption exceeds production for a specific organism, we must again consider the pollutant contribution from externally produced supply. Under these circumstances, we assume all local production is consumed locally and add a sufficient external production to make up the difference. This external source will have a given background pollutant concentration that must be estimated.

As before, we ignore the effects of producer or consumer processing on the pollutant concentration in each type of aquatic organism. This is again a conservative assumption, but it may be more valid for aquatic organisms than for vegetable products.

Ingestion of Animal Products. Animals consume toxic elements by inhalation and ingestion of vegetation and drinking water. Some pollutants may concentrate within animals in organs or products that are consumed by humans. The transfer of toxic pollutants through food webs, and their assimilation, metabolism, and excretion within animals has not

been thoroughly studied or well understood. Very little quantitative or qualitative information is available. These processes, however, depend on the characteristics of the toxic material and animal species, as well as environmental conditions.

Most of the effort relating to food web transfers, bioconcentration rates, and exposure has been directed to studying radionuclide behavior in plants and animals. For example, NRC (1977) presents stable element transfer rates for a number of elements, including some priority pollutants. These transfer coefficients give the fractional transfer of radioactive elements from soil to edible parts of plants, from feed to cow and goat milk, and from feed to beef. Without other information, the appropriate values from these references could be used in calculating this exposure rate. NRC (1977) and WPPSS (1973b) also provide information about feed and water consumption by animals. These are shown in Table 5.5. Dvorak et al. (1978) give recommended quality standards for livestock drinking water.

In general terms, the concentration of pollutant in an animal product (including meat, milk, or eggs) can be calculated from the animal's feed and water consumption rates, the transfer functions discussed above, and the pollutant concentration in animal feed and water. Animal production should be specified by subregion. Pollutant concentrations in drinking water can be determined from the results of the surface water transport model. The concentrations in feed can come either from vegetable product concentrations (for grass, corn, or other

Table 5.5
ANIMAL FOOD AND WATER CONSUMPTION RATES

Animal	Feed or Forage (kg/day)	Water (l/day)
Milk cow	50 - 55	60
Beef cattle	50 - 68	50
Goats	6	8
Pigs	4.2	10
Chickens	0.12	0.3

feed crops) calculated previously or from other estimates. The production in all subregions should be combined to determine average pollutant concentrations for all local production.

To determine the exposure rates for ingestion of animal products, we must follow a procedure similar to that used for the vegetation and aquatic organism analysis. In this procedure, we

1. Determine primary animal products produced and consumed in the region and specify the distribution of these products by subregion.
2. Determine the types of feed consumed and mean daily consumption rates of feed and water for each animal species.
3. Estimate pollutant concentrations in feed and water supplies for animals by subregion, using the results of the surface water transport model and concentrations determined for feed crops in the vegetable product exposure calculations.
4. Calculate pollutant concentrations in animal products by subregion.
5. Determine mean pollutant concentration by animal product for the region.
6. Compare local production with local consumption of each product. Distribute consumption of local products uniformly across the regional population, and calculate exposure rates by product. Sum rates over products.

We again make two simplifying assumptions for these calculations. First, no pollutant is removed from the animal food or water or from consumed animal products through processing by the producer or consumer. Second, we compare total regional production with total consumption to determine if external supplies of any product must be included. If total regional production exceeds regional consumption, we assume all consumption comes from regional sources. If regional production does not exceed regional consumption, we must include externally grown products with an assumed background pollutant concentration.

Our assumptions about uniform distribution of products within the region and population groups will not accurately assess pollutant ingestion by individuals or groups with atypical diets. These groups can be considered separately with sensitivity analysis, if necessary for a particular application. Population subgroups may be determined on the basis of any characteristics, not only age or sex.

If the hunting and consumption of game birds or animals is an important activity in a region, it should be considered in this part of the analysis. In general, it is unlikely that such consumption would be a significant contribution to total exposure. Under certain circumstances, however, the pollutant concentrations in hunted species may be extremely high, if they consume polluted feed or forage.

ALTERNATIVE PROCEDURES

The above calculations of ingestion exposure may require collecting large amounts of input data and specifying parameters with incomplete information. The resulting uncertainty in calculated exposure rates could be quite large. It is likely that most of the animal and vegetable products consumed in a region will not generate significant exposure levels. For these reasons, it may be less difficult to make preliminary rough calculations of exposure rates for primary products. The results of the calculations should indicate which products need to be considered in more detail. As an alternative, if resources or data permit, it may be possible to measure the pollutant concentrations in a sample of locally produced foods and to use this information in calculating exposure rates for these products.

6. HEALTH EFFECTS

INTRODUCTION

When the potentially toxic substances present in the emissions of a power plant are dispersed through the environment, people in the area are exposed to the action of these agents. Several modes of exposure are possible, including inhalation of the emitted gases and particulates, ingestion of contaminated drinking water or locally grown foods, and uptake through dermal exposure to agent-containing rain, lake, or ocean water. To assess whether these exposures constitute a significant hazard to the population, the dose of each toxin over time must be evaluated for each geographically and biologically distinct portion of the population and then used to estimate the increased risk over background of a particular toxic response (or set of such responses).

To pass from the dose to the estimated risk, one must depend on statistical or statistical-biological models relating the dose presented to a human or test animal to the increased prevalence of toxic response per unit of exposed population--"dose-response" models. In this chapter, we are primarily concerned with long-term risk rather than acute events and, as a result, we are particularly interested in dose-response models of chemical carcinogenesis.¹ In the sections that follow, we will review the origin, mathematical form, implementation, and limitations of the presently available models.

Most of the response data available for the parameterization of these models have been obtained from animals (e.g., the mouse and rat) exposed to a carcinogen over their lifetimes. However, because

¹Definitions: carcinogen--an agent that transforms a cell from a normal to cancerous state; mutagen--an agent that causes a heritable chromosomal change at the cellular level; teratogen--an agent that causes gross anatomical malformations in newborns; fetotoxin--an agent that causes death of a fetus. Although we emphasize models that deal with carcinogens, the models are not limited only to this type of response and may equally well be used to estimate the prevalence of other chronic diseases such as liver or kidney necrosis (death of cells or tissues), fetotoxicity, and teratogenicity.

utilities are interested in estimating the cancer risk to a human, rather than animal population, methods must be available that allow us to scale from the animal to a human. Accordingly, sections of this chapter will also discuss the calculation of equivalent human doses from animal dosages, including the techniques that take fractional lifetime human exposures and time-varying toxic agent concentrations into account.

We will focus our attention on the problem of extrapolating dose-response relations from the high dose ranges in which most laboratory data are gathered to the low dose range encountered in environmental exposures. The difficulty arises because these dose ranges are generally separated by several orders of magnitude, the laboratory dose levels having been chosen to be very high to guarantee that toxic effects will be observable within a relatively short time (several weeks to two years).

In the case of epidemiological data, such as the increased incidence of cancer in workers exposed to a carcinogen in the work place, a contrasting situation arises in which the dose levels are generally much closer to the environmental range, but the ability of the data to deal with low occurrence events is severely limited by the number of workers in the statistical sample. For example, data obtained for nearly 400 workers exposed to methylene chloride, in which no significant increase of cancers was exhibited, only indicated that the added risk per worker is less than about one in 400. In another case, epidemiological data from the chemical industry were able to unambiguously identify an increased risk of liver angiosarcoma (a cancer arising in the vascular elements of the organ) to workers exposed to vinyl chloride, but only because of the normally low background rate of angiosarcoma and the chemical's strong carcinogenicity. Hence, a particular toxic response may be completely overlooked epidemiologically if the incidence rate is small.

Such rare toxic responses are far more likely to be found when laboratory animals are exposed to high doses of chemical agents. However, the high dosages employed may give a false picture because the high concentrations of agent found within the tissues of the animal may overload activation or detoxification pathways and indicate a dependence

of prevalence (risk) on a dose that is far different than that which actually occurs at environmental dose levels. Because of the potentially large nonlinear effects of this metabolism on risk estimation, and because this metabolism is described by biological experiments independent of animal-response experiments, there is a need to explicitly include metabolism in dose-response analysis.

This is done by estimating the dose or concentration of activated metabolite in the tissue that is the target of the toxic agent, via the formalisms of pharmacokinetics, and rescaling the experimental (unmetabolized agent) dose-response curve to an activated metabolite-response relation. The metabolite-response relation may then be fit by any one of a set of mathematical response models (e.g., of carcinogenesis) and extrapolated into the dose range where risk estimates are to be made. Because several pharmacokinetic formalisms of varying detail and difficulty of use may be employed to accomplish the dose scaling, they will be described in some detail in later sections of this chapter.

Much of the discussion in this chapter deals with methods for estimating risk under the assumption that complete metabolic and toxicologic information, environmental background levels, and dosage dynamics for each emitted pollutant are known. In practice, especially when making first estimates of carcinogenic risk to an exposed population, this information is not available and several simplifications are often made. For example, a major problem is that a large number of different chemicals are released to the environment by a power plant, but metabolite and toxicologic data are available for only a few. For simplification, attention is restricted to only those compounds emitted in the largest quantities, unless simple cellular assays for mutagenicity or animal cancer data indicate very high specific toxicity (i.e., high mutagenicity/carcinogenicity risk per unit mass of material). In another example, it is often difficult to predict and formulate descriptions of the fluctuating dose levels of pollutants. Simplification may be achieved in this case by using time-averaged concentrations. And in still another example, one often assumes that metabolic kinetics are only linearly dependent on metabolite concentration variables to avoid lengthy pharmacokinetic experimental and modeling efforts.

It is important to stress, however, that such simplifying approaches are taken only to deal with incomplete or difficult to obtain data and not because they eliminate factors that only marginally contribute to the estimates of risk. On the contrary, the exclusion of a factor such as the complete pharmacokinetic description of a toxic agent may lead to an error in the risk estimate of several orders of magnitude due to the failure to extrapolate in a proper nonlinear fashion from high to low doses. More accurate estimates of carcinogenic risk would require the gathering of more extensive experimental data and implementation of the more general formulations presented below.

Because of the relatively large number of dose-response models that may be used to describe long-term toxicity, we have included a discussion, following the Pharmacokinetics and Dose-Response section, that provides some guidance to the selection of the most appropriate models.

TOXICITY TESTING

General Considerations of Dose Response

When attention is first focused on the possible toxic effects of an agent, a series of preliminary questions dealing with the mode of action and type of toxic response of the agent must be answered before whole-animal dose-response work begins. These include:

- Is the substance a mutagen, carcinogen, teratogen, fetotoxin, etc.?
- At what approximate dose range are toxic effects observable?
- What tissues or organs are affected?
- Is the substance active or must it be activated by metabolism?

The answers to these questions must be obtained before the long-term and expensive commitment to animal dose-response studies. As a consequence, a large number of short-term in vitro (culture media) and in vivo (whole animal) tests have been devised to approach these issues. Once these tests have been completed, subchronic whole-animal dose-

response studies are carried out for 30, 60, or 90 day periods using doses and following the specific target tissue responses identified in the short-term tests. Some of these short-term tests are quantifiable in the sense that they allow various carcinogens to be ranked in toxicity relative to one another. However, such quantification does not allow absolute added risk estimates to be made for animal or human populations. To provide a background to the available toxicological data, we next briefly summarize the most quantitative short-term tests, keeping the focus primarily on mutagenic or carcinogenic testing.

Short-Term Tests

Most of the short-term tests available today are for mutagenicity. This is because mutations themselves may lead to heritable metabolic diseases, overt anatomical malformations, or other maladies increasing the species' total disease burden. In addition, there is a strong, although not strict, correlation between the carcinogenicity of a compound and its ability to induce mutations (Ashby and Styles, 1978). In this regard, a critical observation is that both cancer- and mutation-caused disease are characterized by the passage of the abnormal behavior of a cell, once established, on to subsequent generations of its daughter cells. This common characteristic hints at an underlying similarity of mechanism that could account for the good mutagen/carcinogen correlation.

Lethal Dose. This is the simplest in vivo test and is used to fix the approximate upper dose level for carcinogenic toxicity testing. Generally, a small number of animals (most commonly rats or mice) are exposed to a wide logarithmically spaced range of doses of a suspected substance and the fraction of animals dying within the test period (anywhere from 24 hours to 14 days) at each dose is noted. From these data, the median lethal dose (LD_{50}) is determined by a standardized probit analysis (Finney, 1971).

This test detects acute toxicity. It is not useful for the assessment of carcinogenicity or mutagenicity because these effects are not observed in such short time periods and because the metabolic responses to such large dosages of agent are potentially quite different from those observed during long exposure to low environmental doses.

Identification of the range where deaths from acute toxicity are likely to occur is useful in intermediate or long-term animal testing, because this knowledge allows the toxicologist to keep dosages low enough to prevent carcinogenic deaths from being masked by unrelated acute deaths.

Short-Term Genetic Assays. There are at least thirty methods for detecting genetic risk (Wodicka et al., 1978). We summarize seven of the best-known ones. The *bacterial reverse mutation assay* (Ames Assay) is the fastest, most sensitive, and most often quoted method for determining mutagenicity and, by implication, carcinogenicity. In this test bacterial cells (usually *Salmonella typhimurium*) that cannot initially synthesize the amino acid histidine are exposed to a potential mutagen, then examined for the presence of (reverse) mutations that reinstate the cell's ability to synthesize the amino acid. The greater the number of cells that regain the histidine synthesizing activity, the greater the mutagenic power of the test substance. Another assay is the *host-mediated assay*, a technique that measures the number of bacterial mutations induced by a toxic substance (like the Ames Assay), except the bacteria are grown in an animal to expose them to actual metabolites. Two assays measure the toxicity of a compound by scoring chromosomal damage. The *micronucleus test* examines red blood cells, taken from animals exposed to a toxic agent, for the presence of chromosomal fragments (micronuclei) that have been created by the action of the agent. *Mammalian cytogenetic tests* sample bone marrow cells or peripheral lymphocytes² from animals dosed with a toxic agent and examine these for structural chromosomal aberrations. Two other assays assess a compound's ability to cause heritable mutations. In the *mouse specific locus test*, mice that are bred true for certain characteristics, such as eye or coat color, are exposed to a suspected toxic agent, mated, and the offspring examined for the presence of altered characteristics. In the *heritable translocation test*, male mice are exposed to a suspected chemical and the offspring examined for sterility. A related test is the *dominant lethal test* in which the exposure effects are measured by counting the fetal loss (or, alternatively, the fraction of offspring exhibiting teratology) following mating.

²Peripheral lymphocytes are a type of white blood cell found in the general circulation of the body and associated with immunity.

In general, all these tests provide information about the site of action of a toxic agent and the sensitivity of response to dose. Collectively they constitute a guide for the selection of the dose range to be used in subchronic and chronic testing.

Intermediate and Long-Term Tests

These tests are employed when the responses of a whole animal to the action of a carcinogen (or other toxic substance) are desired. The tissues investigated most carefully are determined by where the carcinogen normally induces tumors. This approach differs from the short-term tests where special tissues or cells (e.g., sperm or *Salmonella* bacteria) are selected for response before application of the toxic materials.

Most often, the intermediate length (subchronic) tests are 30 or 90 days in duration. The principal goals of these tests are (1) to validate the dose range where, on the basis of short-term tests, toxic effects are expected to occur; (2) to perform much more detailed pathology; and (3) to identify the metabolic pathways involved in long-term exposure. If the dose range has been properly selected, the results will bracket a "no observed effect level" (NOEL). The pathological investigations include post-sacrifice observations of gross organ damage as well as related histopathology. The number of animals (usually mice or rats) used in these intermediate (subchronic) tests is about 40, evenly spread over at least five doses.

To properly evaluate a substance with respect to its ability to cause cancer or other progressively irreversible toxicity, long-term (chronic) tests must be conducted. These extend over most of the lifetime of the test animal (usually mice and rats), generally 24 months, and are conducted in much the same way as are the intermediate tests. A sound study examines at least four nonzero dose levels plus a control and uses a minimum of 50 animals per dose per sex to achieve good statistics on the prevalence of disease--far more animals than in the subchronic tests. The competence of the pathologist in identifying the various tumors that arise in animals (several of which may occur under the action of a single carcinogen) is of extreme importance, since his calls of abnormality underlie all the quantitative data generated.

The dose-response data obtained in these long-term assays are usually dichotomous, these data giving the proportion of animals that exhibit carcinogenicity at a particular dose and the complementary proportion that does not. The approximate degree of carcinogenicity (e.g., the number of tumors in each test animal) is sometimes recorded, but because extremely exhaustive examination would be required to assure an accurate total tumor count, such data are used only qualitatively to indicate the most sensitive target tissues.

DOSE-RESPONSE MODELS

The proportional prevalence data obtained from long-term, whole-animal testing characterizes the high dose range from which they were taken. However, environmental assessment requires that responses be estimated for doses that are far less. To make these estimates, a mathematical model of the dose-response relation is assumed, fit to the experimental data, and then extrapolated into the low-dose range. Several models are presently in use and, in this section, we derive and discuss the best known ones.

These models are frequently used to fit laboratory data where the agent dose (uptake of mass per unit of time) is constant over time and the exposure duration is fixed. In this case, the final form of the mathematical model used for fitting is time-independent. However, in the case of people exposed to toxic pollutants, the pollutant concentration in a given area may change with time, and the length of exposure may be variable depending on a particular subject's residence time in that area. Hence the models we discuss have been modified where possible to account for time dependence.

One-Hit Model

This is the simplest model for carcinogenesis. Although it is a limiting case of the more adaptable multihit and multistage models described next, we include it as a separate case because of the frequent references made to it in both the past and present literature. As will be seen, it is also closely related to one of the confidence bands about the more complicated multistage model.

The one-hit model assumes that a particular, single irreversible event occurs within a cell as the result of the action of a carcinogen. The nature of this event is unspecified but could, for example, be a chemical transformation of a nucleotide in DNA resulting in a point mutation or perhaps breakage of the chromosome. The event (actually a precursor event to the tumor observation) is termed a "hit" and is assumed to occur stochastically in a population of exposed animals, or

$$p_0 = -\lambda(t)p_0, \quad p_0(0) = 1 \quad (6.1)$$

where $p_0(t)$ = probability of finding a hit-free member of the population (a tumor-free individual) at time t ,
 $\lambda(t)$ = transition rate giving the probability per unit time that a hit will occur shifting a member from the tumor-free to the tumor-bearing category.

$\lambda(t)$ is generally assumed to be proportional to the concentration of carcinogen at the target site, i.e.,

$$\lambda(t) = \alpha c(t) \quad (6.2)$$

Integrating Eq. (6.1) for p_0 and subtracting it from 1 gives the probability of finding a responder at time t , P_r , i.e.,

$$P_r = 1 - p_0(t) = 1 - \exp \left[- \int_0^t \lambda(t'') dt'' \right] \quad (6.3)$$

The low-dose limit implies that $\lambda(t)$ is very small and hence

$$P_r \cong \int_0^t \lambda(t'') dt'' = \alpha \int_0^t c(t'') dt'' \quad (6.4)$$

If the metabolism and tissue distribution of the toxic substance are linear in concentration dependence, then $c(t)$ is proportional to the administered dose, $d(t)$.³ Note that once the time of exposure and concentration function are specified, the one-hit model, Eq. (6.4), is a one parameter model.

In laboratory experiments where animals are exposed over a lifetime to a constant concentration of carcinogen, the level of carcinogen within the target tissue often reaches steady-state and may be presumed to be constant and proportional to the dose, d .⁴ In this case Eq. (6.4) may be integrated to give

$$P_r = 1 - \exp(-\theta d) \quad (6.5)$$

where θ = new constant of proportionality implicitly containing the fixed exposure time.

In the low-dose limit, we obtain the linear relation

$$P_r \cong \theta d \quad (6.6)$$

³This relation is explained in more detail in the Pharmacokinetics and Dose-Response discussion below. The definition of the dose d depends on the route of administration. For inhalation, it is approximately the concentration of chemical in the respired gas. For ingestion, it is approximately the time-averaged concentration in the food mass. For dermal exposures, it is approximately the concentration or mass of chemical in the contact medium.

⁴Providing nonlinear metabolism or distribution does not occur.

In summary, Eq. (6.4) is the most general version of the one-hit model but it may require that an additional pharmacokinetic model be known to relate administered dose to target tissue concentration. Equation (6.5) is the version useful for fitting most experimental toxicological data.

Multihit Model

This model begins with the concept of a hit but presumes that several hits may be required before a cell or tissue is transformed to a cancerous entity (Cornfield et al., 1978). The differential stochastic equations that describe this model are

$$\dot{p}_0 = -\lambda(t)p_0 \quad (6.7)$$

$$\dot{p}_n = \lambda(t)p_{n-1} - \lambda(t)p_n \quad 1 \leq n \leq \infty$$

with initial conditions

$$p_0(0) = 1; \quad p_n(0) = 0, \quad n \neq 0$$

where p_n = probability that any particular cell in an individual will contain exactly n hits.

These equations describe a Poisson process except that the transition rate is dependent on time. These equations may be integrated straightforwardly in the order of increasing n and the general solution may be induced to be

$$p_n(t) = \frac{\Lambda^n(t)}{n!} \exp [-\Lambda(t)] \quad , \quad n \geq 0 \quad (6.8)$$

where

$$\Lambda(t) = \int_0^t \lambda(t'') dt'' \quad (6.9)$$

and $\lambda(t)$ is again taken to be given by the expression in Eq. (6.2).

The model assumes that a tumor will develop if any cell has been hit k or more times. Hence the probability of an individual's developing cancer from the carcinogenic agent, P_r , is given by

$$P_r = \sum_{n=k}^{\infty} p_n(t) = \sum_{n=k}^{\infty} \frac{\Lambda^n(t)}{n!} \exp [-\Lambda(t)] \quad (6.10)$$

Cornfield and Mantel (1977) and Rai and Van Ryzin (1979) have shown that the sum on the right side of Eq. (6.10) can be replaced by the integral expression

$$P_r = \int_0^{\Lambda(t)} \frac{x^{k-1} e^{-x}}{(k-1)!} dx \quad (6.11)$$

which may be confirmed by integrating by parts. The factorial in Eq. (6.11) may be replaced by the gamma function to yield an expression evaluable for all real values of k

$$P_r = \int_0^{\Lambda(t)} \frac{x^{k-1} e^{-x}}{\Gamma(k)} dx \quad (6.12)$$

although the hit interpretation of a fractional value for k is

problematical. This expression is termed the GKM (generalized k-hit) model for tumor response in a population and is applicable to the case of variable time of exposure (through the upper limit in Eq. (6.9)) and variable concentration (through Eqs. (6.2) and (6.9)). Note that the multihit model is a two parameter model (α in Eq. (6.2) and k) once the time of exposure and target cell concentration are specified.

In the low-dose limit of Eq. (6.12), $\Lambda(t)$ is always small and x is confined to be near zero. Hence

$$P_r \rightarrow \frac{1}{\Gamma(k)} \int_0^{\Lambda(t)} x^{k-1} dx = \frac{\Lambda^k(t)}{k\Gamma(k)} = \frac{\Lambda^k(t)}{\Gamma(k+1)} \quad (6.13)$$

If the kinetics of toxic substance metabolism and distribution are linear in their concentration dependence and one again considers the lifetime chronic laboratory experiments where dose levels (d) and tissue concentrations are time-independent

$$\lambda(t) \rightarrow \theta'd$$

and Eq. (6.12) becomes

$$P_r = \int_0^{\theta d} \frac{x^{k-1} e^{-x}}{\Gamma(k)} dx \quad (6.14)$$

where $\theta = \theta' t$, t being the fixed time of exposure. In the limit of low dose, Eq. (6.13) shows that

$$P_r \rightarrow \frac{(\theta d)^k}{\Gamma(k+1)} \quad (6.15)$$

k and θ are the model parameters in this case.

When $k = 1$, the multihit (GKM) model reduces to the one-hit model as may be seen by noting that the $k = 1$ case of Eq. (6.15) is identical to Eq. (6.6).

In summary, Eq. (6.12) (along with Eqs. (6.2) and (6.9)) is the most general version of the multihit model although it requires that the time-dependent target cell concentration or dose be specified independently. Equation (6.14) is useful for fitting most experimental toxicological data.

Multistage Model

This model may be viewed as a generalization of the multihit model. The multistage model (Armitage and Doll, 1961) assumes that several hits are required before a neoplastic transformation occurs, but unlike the multihit model, these hits must occur in a particular sequence. This is shown schematically in Fig. 6.1, where a large number of hit permutations are possible but only one is associated with carcinogenesis, i.e., the 123...k sequence.

Fig. 6.1 — The multistage model

Stochastic differential equations may be formulated for this model and are

$$\dot{p}_0 = -p_0 \sum_{i=1}^k \lambda_i \quad (6.16a)$$

$$\dot{p}_1 = \lambda_1 p_0 - p_1 \sum_{j=2}^k \lambda_j \quad (6.16b)$$

$$\dot{p}_{1\dots n} = \lambda_n p_{1\dots n-1} - p_{1\dots n} \sum_{j=n+1}^k \lambda_j \quad 1 < n < k \quad (6.16c)$$

$$\dot{p}_{1\dots k} = \lambda_k p_{1\dots k-1} \quad (6.16d)$$

where $\lambda_i(t)$ = transition probability for i^{th} stage.

Equation (6.16a) describes the rate at which individuals are lost from the initial population because of the hits at each of the k loci involved in carcinogenesis (see Fig. 6.1). However, only one of these ($i = 1$) provides cells for the next stage of carcinogenesis. Equation (6.16b) describes the balance between the production of individuals with one hit at site 1 and the loss of these to the two-hit group $1j$; again only one of these ($j = 2$) provides cells for the next stage. Equation (6.16c) describes all later stages except the last, which is described by Eq. (6.16d). $p_{1\dots k}$ is the probability that an individual exposed to a carcinogen will contract cancer at time t .⁵

The transition probabilities, $\lambda_i(t)$, are assumed in this model to have different values for each stage. Furthermore, they are assumed to have a different form than used in the one- or multihit models. Here

⁵Unlike the multihit model (Eqs. (6.7)), Eqs. (6.16) are a finite set because Eq. (6.16d) makes no provisions for individuals to leave the $(1\dots k)$ category even if further hits were to occur. Thus the summing process used earlier in Eq. (6.10) with the multihit model has already been accounted for in the definition of $p_{1\dots k}$.

$$\lambda_i(t) = a_i + b_i c(t) \quad (6.17)$$

where a_i is the contribution to the transition probability due to non-specific background effects (i.e., hits generated spontaneously or by the action of substances in the environment other than the carcinogen of interest) and $b_i c(t)$ is the contribution due to the action of the toxic agent under investigation. As before, $c(t)$ is the target site concentration. Summing the background effect to the concentration-dependent effect is described as the *additivity assumption* and implies that both the test and background agents have the same site of action, a controversial point (Cornfield, 1977; Cornfield et al., 1978).

Equations (6.16) can be solved formally by integrating them in order of increasing n and using the initial conditions

$$p_0(0) = 1, \quad \text{all else} = 0 \quad (6.18)$$

However, for the general case of a time-dependent $\lambda(t)$, the resulting expression rapidly becomes unwieldy. Armitage and Doll (1961), Whittemore (1977), and others concerned with carcinogenesis, particularly in the environment, have, therefore, focused on the low-dose (small λ_i) version of this model and so we now proceed along similar lines.

Low-Dose Case. In this case, the assumption can be made that the group of individuals characterized by n -hits is not significantly depleted during any time increment by the few undergoing the next hit. Hence, the loss terms on the right-hand sides of Eqs. (6.16) can be dropped. The resulting equations are simple first-order ordinary differential equations and may be integrated successively, along with Eq. (6.18), to give

$$p_{1\dots k} = \int_0^t \lambda_k(t_k) \int_0^{t_k} \lambda_{k-1}(t_{k-1}) \dots \int_0^{t_2} \lambda_1(t_1) dt_1 \dots dt_{k-1} dt_k \quad (6.19)$$

If the expression in Eq. (6.17) is substituted for $\lambda_i(t)$ and an $a_1 \dots a_k$ product is factored out,

$$p_{1\dots k} = \alpha_0 \int_0^t [1 + \alpha_k c(t_k)] \dots \int_0^{t_2} [1 + \alpha_1 c(t_1)] dt_1 dt_2 \dots dt_k \quad (6.20)$$

where $\alpha_0 = a_1 \dots a_k$ and all other $\alpha_i = b_i/a_i$.

Equation (6.20) is the most general form for a variable concentration of toxic substance in the low-dose limit. Note that once this concentration function and the time of exposure (t in Eq. (6.20)) are specified externally, the multistage is a $(k + 2)$ parameter model: α_0 to α_k , and k .

In the case of substances that undergo linear metabolism and distribution and that are tested under lifetime steady-state dosage conditions, the λ_i in Eq. (6.19) become constants and one obtains

$$p_{1\dots k} = \lambda_1 \dots \lambda_k \frac{t^k}{k!} \quad (6.21)$$

or substituting the constant dose, d , for $c(t)$ in the definition of λ ,⁶

$$p_{1\dots k} = \alpha_0 (1 + \alpha'_k d) \dots (1 + \alpha'_1 d) \frac{t^k}{k!} \quad (6.22)$$

⁶The α_i ' in Eq. (6.22) contain a constant of proportionality between the administered dose and target cell concentration and are, therefore, slightly different from the α_i in Eq. (6.20).

Crump Formulation. Guess and Crump (1976) have suggested an alternative formulation that contains the multistage result of Eq. (6.22) as a special case. The formulation is advantageous in that it can be applied in the high-dose range and robust programs are available from Crump for obtaining the parameters from experimental data. As presently formulated, it applies to the case of constant (steady-state) dosage.

This model is noteworthy because the EPA has used this methodology in assessing many of the nonthreshold health effects discussed in the Consent Decree Water Criteria Documents (EPA, 1980c, App. C). However, this use does not mean that the model is necessarily more accurate in describing dose-response data than other models described in this report. For example, the EPA itself has used the Weibull model (see below) in preference to the Crump model to set the water quality criteria for arsenic.

This formalism assumes that the prevalence of carcinogenic response obtained after k hits is

$$p_{1\dots k} = 1 - \exp \left[- \sum_{i=0}^k q_i d^i \right] \quad q_i \geq 0 \quad (6.23a)$$

where the q_i are parameters to be determined. The power dependence of the dose variable is suggested by the power series in dose that would result if one multiplied out the coefficients in Eq. (6.22). In the limit of low dose, the exponential may be expanded to give

$$p_{1\dots k} = \sum_{i=0}^k q_i d^i \quad (6.23b)$$

For a low constant-dose experiment, the coefficients of the power series in Eq. (6.22) may be identified with the q_i .⁷

⁷The identification is conditional, however. If the α_i are first determined from data by Eq. (6.22) subject to all $\alpha \geq 0$, then the q_i are

It should be noted that the Crump formalism implicitly contains the $\lambda_i = a_i + b_i c$ additivity assumption. Looking at Eq. (6.22), it is clear that a power series containing all powers of d arises only because of the algebraic form of λ_i . Hence the criticism of the additivity assumption is also directed against this model.

A problem with the Crump model is the absence of any term showing how the response probability depends on exposure time. This is important when one wishes to estimate the response in a group of animals or humans that have not been exposed to an agent for a full lifetime as are the laboratory test animals used to calibrate the model. An ad hoc procedure that attempts to account for exposure time introduces a power of time multiplier in the formulation of Eq. (6.23a) giving

$$p_{1\dots k} = 1 - \exp \left(-t^k \sum_{i=1}^k w_i d^i \right) \quad w_i \geq 0 \quad (6.24)$$

where w_i = set of unknown parameters replacing the q_i .

The justification for this approach is that the low-dose limit of this expression is identical to the time-dependence of the multistage result of Eq. (6.22). This ad hoc procedure has also been adopted by the Environmental Protection Agency in setting water quality criteria (EPA, 1980b) except that while k has still been left to describe the summation range, the exponent of time has been empirically fixed at three in an attempt to achieve better fits to complete age-response data. However, to be consistent with the multistage model as originally described, the exponent should equal the number of hits, k .

uniquely determined and $q_i \geq 0$ as required in Eq. (6.23). On the other hand, if the q_i are first determined from fitting data to Eq. (6.23), subject to all $q_i \geq 0$, then there is a possibility that not all the q_i will be positive or zero.

Probit Model

This is the oldest statistical model applied to carcinogenesis. It is not based on the concept of a "biological mechanism" such as a series of hits but rather on the concept of tolerance. The idea is that each subject in a population has a tolerance, T , to the action of a toxic substance, defined in terms of a dose or target cell concentration variable, above which the subject responds and below which it does not. The tolerances are distributed over the population and are described by a probability density. In the case of the probit model, this probability density is taken to be log normal.

The probability of response in a population exposed to a carcinogenic agent, P_r , at dose d is therefore given by the integral

$$P_r = \int_{-\infty}^{\alpha + \beta \log d} f(x) dx = \int_0^d g(T) dT \quad (6.25a)$$

where $f(x)$ = standard normal distribution function,
 $g(T)$ = probability density of tolerances.

As written, this model applies only to a nonvarying dose and fixed exposure period (generally lifetime). Use of the model to predict response under conditions where exposures are less than lifetime can be approximated by substituting the empirical relation dt^n in the log term of Eq. (6.25a), where d is the dosage concentration over the exposure period and t is the exposure time in lifetime units. If dose-age-response data are available, then n may be determined from the data. However, in the case of lifetime exposure experiments, n cannot be determined from the data and its value must be chosen arbitrarily. Two selections are $n = 1$, corresponding to using an equivalent lifetime average dose in Eq. (6.25a), and $n = 4$, corresponding to the mean exponential value determined by Druckrey (1967) over several carcinogens and tumor types. With n fixed, the model is a two parameter model: α and β .

A special version of this model is the Mantel-Bryan model (Mantel and Bryan, 1961) in which $\beta = 1$ identically. The reason for this selection is that the lower the value of β , the higher the response P_r at a given low dose. Mantel and Bryan found that for all substances then studied, β was at least one. Hence they forced β to equal one in the attempt to be conservative and assure that the risk to a chemical was overestimated. However, the current movement is away from this model, leaving questions about the conservative nature of risk estimation to confidence limit investigations.

Weibull Model

This model may also be viewed as a tolerance model. It is a function of both the dose and length of time of exposure to that dose. It differs most significantly from the hit or multistage models by allowing more flexibility in describing the dependence of prevalence (or incidence) upon exposure time. As a consequence, this model may fit age-response-dose data when the hit or multistage models fail.

The Weibull model for prevalence (following Doll, 1971) is

$$P_r = 1 - \exp(-\alpha d^k t^n) \quad (6.25b)$$

where d = dose,

t = exposure time (lifetimes).

Note that if $k = 1$, for a fixed value of time the Weibull model degenerates to the one-hit model. Generally, this is a three parameter model that requires the existence of dose-response data as a function of age (i.e., time of exposure). However, it may also be fit to lifetime dose-response data. In this case, $t = 1$, n is irrelevant, and only two parameters in Eq. (6.25b), d and k , need be determined by curve fitting.

Inclusion of Background

Even in animals or humans who have not been exposed to a known carcinogenic agent, tumors still appear at a low prevalence rate. This is because the tissues of the animals are either predisposed to tumor formation or they are exposed to other sources of carcinogens. This background prevalence rate must be taken into account when performing a complete dose-response analysis.

It is important to draw a distinction between two types of background. One type--the nonspecific background--includes spontaneous, thermally driven cancer transformations as well as the transformations induced by carcinogens other than the one under consideration. The other type--the specific background--includes the contribution to cancer prevalence from other sources of the carcinogen under consideration. The nonspecific background is generally the only type associated with laboratory animal experiments because great care is taken to eliminate secondary sources of the agent under test. However, when predicting human response to environmental agents, the specific background must be considered. For example, in the case of water-borne arsenic toxicity from a power plant, the total human exposure will be due to both the arsenic emitted from the plant as well as the low level of arsenic normally present in groundwater. The distinction of background types is necessary because the contribution of each type to the overall cancer prevalence is generally calculated in a different manner, depending upon the dose-response model.

In the case of the multistage model, we have already shown the procedure for including background in Eq. (6.17). The nonspecific background (at the i^{th} level) is accounted for by the a_i term and the specific background is added to it as part of the total concentration $c(t)$. This approach has already been carried through in our derivation of the multistage models, and Eqs. (6.20) to (6.24), therefore, express the total tumor prevalence, e.g., the prevalence observed in animal experiments.

When considering prospective environmental studies that aim at quantifying the health risk to a population due to toxic substance exposure, the metric of primary concern is the *added risk* over total

background. Hence in the prospective case, the multistage added risk, P_{ar} , is given by

$$P_{ar} = p_{1...k}(d_{total}) - p_{1...k}(d_b) \quad (6.26)$$

where d_{total} = sum of administered dose and specific background dose, d_b .

When dealing with the inclusion of nonspecific background in the one-hit, multihit, probit, or Weibull models, Abbott's method (Abbott, 1925) is a generally employed method. In this approach, unlike the multistage model, the tumor response as a result of added substance is assumed to be functionally independent of the response resulting from nonspecific background. The method equates the total prevalence for any dose, P_r^* , to the sum of a constant nonspecific background term, p_{bn} , and a toxic substance term

$$P_r^* = p_{bn} + (1 + p_{bn})P_r \quad (6.27a)$$

where P_r = any of the nonmultistage prevalences previously formulated.

The specific background is included as part of the dose (or concentration) term in those models. The added risk, P_{ar} , in prospective environmental studies is simply

$$P_{ar} = P_r(d_{total}) - P_r(d_b) \quad (6.27b)$$

where d_{total} and d_b are defined as in Eq. (6.26). The Abbott expression, Eq. (6.27a), is used when calibrating the one-hit, multihit, probit, or Weibull models from observed total cancer prevalence in

animals. Note that the p_{bn} is, in effect, an additional parameter. Hence the one-hit, multihit, probit, Mantel-Bryan, and Weibull models, with background included, become two, three, three, two, and four parameter models, respectively.

Fitting to Experimental Data

Before response models can be used to estimate the risk of exposure at arbitrarily low dose, the parameters of the model must be evaluated from known response data at high dose. In this section, we briefly outline the methods for obtaining the parameter estimates as well as the confidence bands about them and the dose-response relations they determine. This subject has been outlined previously by Rai and Van Ryzin (1979) and we follow their discussion closely.

Form of Data. n_i animals in the i^{th} test group are assumed to be exposed to dose d_i (generally time-independent) for a fixed period of time, $i = 1, \dots, m$, where m is the number of nonzero dose levels. In addition, there is a control group containing n_0 animals. At the end of exposure period, the number of animals exhibiting tumors in each group, x_i , $i = 0, \dots, m$, is recorded.

Best Fit. The method of maximum likelihood is used to estimate the parameters in the models. The probability or likelihood, L , of observing a particular set of x_i is given by the expression

$$L = \prod_{i=0}^m \binom{n_i}{x_i} \left[P_r^*(d_i) \right]^{x_i} \left[1 - P_r^*(d_i) \right]^{n_i - x_i} \quad (6.28a)$$

where P_r^* is given by Eq. (6.27a); in the case of the multistage model, P_r^* should be identified with $p_{1\dots k}$ (as in Eqs. (6.20) to (6.24)). Through its dependence on P_r^* , L depends on the parameters of the model. For example, in the one-hit model, $L = L(\theta, p_{bn})$ (cf. Eqs. (6.5) and (6.27)). The best values for the parameters are then established by numerically maximizing $\log L$ with respect to the parametric variables. The methods for doing this are now relatively standard and we do not review them in detail. Instead, we refer the reader to the computer

programs of Rai and Van Ryzin (1980) for the multihit and one-hit models (the MULTI80 program), the SAS User's Guide (1979) for the probit model, and the PAR or P3R routines in the BMDP Biomedical Computer Programs package (BMDP, 1981) for the Weibull model. (If the background is known to be zero, then the Weibull prevalence may be linearized by taking double logarithms and parameters may be estimated by standard least squares techniques.) Obtaining parameters for the multistage model is more complicated than doing so for the others because the number of hits (k) is not known initially. However, Guess and Crump (1976) derived a search procedure for locating the likelihood maximum efficiently and have developed a program (GLOBAL79) based on their findings (Crump and Watson, 1979).

Confidence Limits. The programs above also provide estimates of the variances about the model parameters. They generally proceed from computation of the large sample, asymptotic information matrix I,

$$I = - \begin{bmatrix} E(\partial^2 \ell / \partial \beta_1^2) & E(\partial^2 \ell / \partial \beta_2 \partial \beta_1) & \dots \\ E(\partial^2 \ell / \partial \beta_1 \partial \beta_2) & E(\partial^2 \ell / \partial \beta_2^2) & \dots \\ E(\partial^2 \ell / \partial \beta_1 \partial \beta_3) & E(\partial^2 \ell / \partial \beta_2 \partial \beta_3) & \dots \end{bmatrix}$$

where $\ell = \log L$,

β_i = set of parameters for a particular model.

It is statistically well-known (Cramer, 1945) that an estimate for the variance of each parameter is given by the diagonal elements of the inverse of I [$I^{-1} = (a_{ij})$], i.e.,

$$\text{var } \hat{\beta}_1 = a_{11}, \quad \text{var } \hat{\beta}_2 = a_{22}, \quad \dots$$

where $\hat{\beta}_i$ = the value of the parameter β_i at the likelihood maximum.

The upper confidence band, $h(d; \hat{\beta}_i)$, about the response function P_r can be obtained from estimating the variance of P_r by

$$\begin{aligned} \text{var } [P_r(d; \hat{\beta}_i)] &= \sum_i (\text{var } \hat{\beta}_i) (\partial P_r / \partial \beta_i)^2 \\ &+ 2 \sum_{i>j} \text{cov} (\hat{\beta}_i, \hat{\beta}_j) (\partial P_r / \partial \beta_i) (\partial P_r / \partial \beta_j), \end{aligned} \quad (6.28b)$$

where the partials are evaluated at the appropriate β_i and $\text{cov} (\beta_i, \beta_j) = a_{ij}$; and then substituting it into

$$h(d; \hat{\beta}_i) = P_r(d; \hat{\beta}_i) + z_\gamma \left\{ \text{var } [P_r(d; \hat{\beta}_i)] \right\}^{1/2} \quad (6.29)$$

where $z_\gamma = 100(1 - \gamma)^{\text{th}}$ percentile point of the standard normal distribution.

(Note also that while $h(d; \hat{\beta}_i)$ is the upper confidence band on prevalence (or risk) when dose is viewed as the independent variable, it is the lower confidence band on dose when prevalence (or risk) is viewed as the independent variable.)

INTERMODEL COMPARISON OF DOSE-RESPONSE MODELS

Any or several of the dose-response models that have been described may be capable of fitting data in the experimental range, although the one-hit model is very often inadequate. When extrapolations are made over several orders of magnitude of dose, the predicted low-dose responses differ widely. This is demonstrated in Figs. 6.2 and 6.3, which show data from Guess and Crump (1977) and Rai and Van Ryzin (1979). Figure 6.2 shows the extrapolations obtained with the Mantel-Bryan, one-hit, and multihit models when applied to the dose-response data (Maltoni, 1975) for vinyl chloride.⁸ The mean (maximum

⁸Vinyl chloride is known to have a nonlinear detoxification pathway

Fig. 6.2 — Low-dose extrapolations of various models for vinyl chloride

Fig. 6.3 — Low-dose extrapolations of various models for benzopyrene

likelihood) curve for the one-hit model is indicated along with the lower 97.5 percent confidence curves (confidence about dose for fixed risk) for the one-hit, Mantel-Bryan, and multihit models (Guess and Crump, 1977; Rai and Van Ryzin, 1979). Similar curves have also been obtained for benzopyrene⁹ (data of Lee and O'Neill, 1971) in which the multihit, Mantel-Bryan, and Crump multistage models may be compared (Fig. 6.3). The mean line for the multistage model is indicated along with its lower 97.5 percent confidence curve and those of the multihit and Mantel-Bryan models.

The confidence bands are plotted in preference to the mean lines because they are useful for assessing the "virtual safe dose" (VSD). The VSD is the lowest dose level within a confidence band that could account for a risk, preselected by common agreement, above which no individual should be exposed. This risk (e.g., prevalence of 10^{-6} or 10^{-8}) is usually selected to keep the number of cases of disease caused by a toxic substance below a hundred cases nationwide.

Examination of the curves in Figs. 6.2 and 6.3 show that the models predict very different VSDs. If we take the highest allowable risk as 10^{-6} , then Fig. 6.2 shows that the multihit and Mantel-Bryan procedures predict VSDs that differ by two orders of magnitude, with the one-hit VSD lying in between. In this case the multihit result at 5.6×10^{-4} ppm is the lowest allowable dose, whereas the Mantel-Bryan result at 8×10^{-2} ppm is the highest. In Fig. 6.3, the multihit and Mantel-Bryan procedures predict VSDs that differ again by about two orders of magnitude, whereas the multistage result differs by nearly four orders of magnitude from the multihit case. (The one-hit confidence curve in Fig. 6.3 would be very similar to the multistage curve. Guess and Crump (1977) have shown that these are always quite close because there is always a possibility that the linear term in Eq. (6.23) may have a non-zero coefficient and dominate the response function at low dose.)

and a complete analysis should, therefore, include pharmacokinetics (Hefner et al., 1975; Watanabe et al., 1976).

⁹Likewise benzopyrene is nonlinear in tissue distribution (Rees et al., 1971; Gehring et al., 1976).

The conclusion that the estimate of a virtual safe dose is extremely model-dependent is inescapable. The uncertainty in estimating a VSD is determined by the mathematical form of the model chosen as much as it is determined by the spread of confidence bands that always occurs when extrapolating far from a region of data.

Furthermore, no one model can generally be chosen over the others because it has a firm biological basis. In no model are the biochemical kinetics of the molecular biology of carcinogenesis (or any other toxic effect) specified. In addition, the individual models may be criticized as follows. The probit and Weibull models are strictly statistical and have no representation whatsoever of biological events. The multihit model assumes that the transition rate λ is the same for all hits, yet there is no reason a priori for assuming that the biochemical or biophysical environment is the same for each hit (even, for example, if the same type of nucleotide were involved). The reason for making λ hit-level independent is primarily to keep the number of parameters low, and because the resulting model can fit a variety of curve shapes in the high-dose range. The multistage model removes this restriction but at the cost of more parameters (hence fewer degrees of freedom) and the questionable additivity assumption statistically correlating background and added toxic substance effects. The multihit model has been suggested by Cornfield and Mantel (1977) to be superior to the other models because, for $k > 1$, the slope of the risk curve is zero ($\partial P_r / \partial d = 0$) at zero dose and this is consistent with the low-dose behavior of several detoxification schemes. However, this is only a boundary condition and constrains the dose-response curve to a form in the intermediate dose range that will not generally be compatible with detailed pharmacokinetics. Thus, perhaps the best that can be achieved with current knowledge is to determine the range of the VSD predicted by various models.

PHARMACOKINETICS

Pharmacokinetics encompasses the set of kinetic procedures describing the rate of uptake of any biologically active compound, the distribution of this agent to various tissues, its metabolism, and excretion. Formally, it consists of a set of time-dependent material balances whose solution allows the determination of the concentration of that compound in a tissue or fluid of interest. We are particularly interested in those applications that allow the concentration of the toxic agent to be estimated for the target tissues, e.g., those where tumors are known to arise under the action of a carcinogen.

Historically, toxicologic response has been estimated from statistical dose-response equations and formal pharmacokinetics have not been used. However, in the last decade there has been a growing awareness (e.g., Wodicka et al., 1978) that certain quantitatively important aspects of toxic agent response can only be described by pharmacokinetic formalisms. Perhaps the most important example is non-linear activation/deactivation kinetics. It is common for animal data to be collected in a dose range where these kinetics depend on dose in a highly nonlinear manner. Hence, even while the utilities have not traditionally dealt with the biochemistry and physiology underlying pharmacokinetics, we have placed some emphasis on this area in the anticipation that accurate treatment of dose-response effects may force consideration of the phenomena involved.

In the following sections, we will describe the principles underlying pharmacokinetics showing the form of the equations involved, their solution, parameterization, and their limitations.¹⁰ We will indicate how nonlinearities may arise in these systems from metabolic and active transport considerations and will show that under certain constraints on exposure, only nonlinear pharmacokinetic descriptions need be carried in detail through the dose-response modeling of animal data.

¹⁰Note that the set of pharmacokinetic models discussed below and the experimental justification for them are far from complete. We have chosen the most robust models to describe but more details on these and other models may be found in the books of Wagner (1975), Notari (1971), and Teorell et al. (1974), and in the papers by Bischoff and Dedrick (1970), Bischoff et al. (1970, 1971), Dedrick et al. (1973a, 1973b), Morrison et al. (1975), and Harrison and Gibaldi (1977).

Compartment Models

These are the simplest pharmacokinetic models that may be considered. The term "compartment" generally refers to an organ space but it is common to lump several organs, having the same ability to take up, bind, and metabolize a chemical substance into a single space (compartment). If no binding of substance to tissue proteins, membranes, etc., occurs, this compartment will have a volume equal to the sum of the volumes of the lumped organs.

Figure 6.4 shows the scheme underlying the example model we will discuss. Basically this is a model of an animal or human body that accounts for toxic substance exposure via respiration or ingestion and that describes the concentration of this agent in the blood and tissues. Figure 6.4 shows a two-compartment, open (i.e., allowing excretion) model with elimination and toxic substance input occurring in the same compartment. The blood compartment is generally identifiable with the real peripheral blood volume, so any chemical concentrations specified for this compartment would be the ones observed in a laboratory analysis of blood. The tissue compartment is lumped and will comprise such organs as the muscle, liver, and spleen. k_{12} and k_{21} are transport parameters, V_T and V_B are the tissue and blood volumes of

Fig. 6.4 — The open two-compartment model with elimination and input from the blood compartment

distribution, k_e is the first-order urinary clearance rate (generally quite similar to creatinine clearance if the kidney filtration does not involve active transport), and $k_o(c_o - Rc_B)$ describes the chemical uptake rate (c_B is the concentration of chemical agent in the blood).

The form of the input rate has been chosen to model several routes of administration. One route is inhalation where c_o is the concentration of toxic substance in the respired air (identifiable with the term "dose"), Rc_B is the concentration that the toxic substance would have in air if in thermodynamic equilibrium with the blood at concentration c_B , and $k_o(c_o - Rc_B)$ is a Fickian mass transfer across the alveolar (lung) membranes. Another route is ingestion. In this case the input term represents transfer across the gastrointestinal linings and c_o is the constant concentration of substance in the gastrointestinal tract.¹¹

The material balances that describe this two-compartment model are

$$\begin{aligned}\dot{c}_T &= k_{21}c_T/VT - k_{12}c_B/VT \equiv \alpha_{11}c_T + \alpha_{12}c_B \\ \dot{c}_B &= \underbrace{-k_{12}c_B/V_B + k_{21}c_T/V_B}_{\text{tissue/blood transport}} + \underbrace{k_o(c_o - Rc_B)/V_B}_{\text{input}} - \underbrace{k_e c_B/V_B}_{\text{excretion}} \quad (6.30) \\ &\equiv \alpha_{21}c_T + \alpha_{22}c_B + k_o c_o/V_B\end{aligned}$$

This is a *linear pharmacokinetic model* because all terms on the right-hand side are proportional to a concentration or are constant. These equations may be solved by standard methods (see App. A) to yield

¹¹The model is only roughly descriptive of actual events in this case since the chemical concentration along the gut lumen is variable, and the substance is not usually given by continuous feeding but rather by periodic gavage (stomach tube). However, c_o is interpretable as the approximate time- and space-averaged gastrointestinal concentration.

$$c_T(t) = c_o \left\{ \beta_T(\lambda_- - \lambda_+) + \left[\alpha_{12}\beta_B + (\alpha_{11} - \lambda_-)\beta_T \right] e^{\lambda_+ t} + \left[(\lambda_+ - \alpha_{11})\beta_T - \alpha_{12}\beta_B \right] e^{\lambda_- t} \right\} \quad (6.31a)$$

$$c_B(t) = c_o \left\{ \beta_B(\lambda_- - \lambda_+) + \left[(\lambda_+ - \alpha_{11})\beta_B + \alpha_{12}^{-1}(\alpha_{11} - \lambda_-)(\lambda_+ - \alpha_{11})\beta_T \right] e^{\lambda_+ t} + \left[(\alpha_{11} - \lambda_-)\beta_B + \alpha_{12}^{-1}(\lambda_- - \alpha_{11})(\lambda_+ - \alpha_{11})\beta_T \right] e^{\lambda_- t} \right\} \quad (6.31b)$$

where λ_- , λ_+ , β_T , and β_B are functions of α_{11} , α_{12} , α_{21} , and α_{22} only and are given by App. A equations (A.3) and (A.8). Appendix A also summarizes the procedures for obtaining values of the parameters in this equation. Note that for this model, c_T and c_B are directly proportional to the inhalation dose, c_o , an observation that will be important when we consider the coupling between pharmacokinetics and dose response.

Nonlinear Pharmacokinetics

As will be seen in the next section, the most significant impact of pharmacokinetics on dose response involves those cases where transport between compartments, nonreactive binding of toxic agent to various cellular components, or metabolism are highly nonlinear functions of concentration. Accordingly, the rest of this section describes several examples of these nonlinearities to demonstrate the incompleteness of a dose-response model that does not account for these phenomena via rescaling the dose-variable from the administered dose to the concentration of toxic agent actually found in the target tissue.

A strong pharmacokinetic nonlinearity may occur, for example, when a substance present at high concentration in the liver is detoxified (i.e., generally converted to a more polar compound, more easily excreted by the kidney) because the amount of substance may exceed the

capacity of liver enzymes to carry out this transformation at their normal rate. The tissue material balance for a two-compartment model would then differ from Eq. (6.30) by a nonlinear reaction term, i.e., the new equation¹² would be

$$\dot{c}_T = \alpha_{12}c_B + \alpha_{11}c_T - \frac{kc_T/V_T}{K + c_T} \quad (6.32)$$

The parameters (k/V_T) and K , like α_{12} and α_{11} , are unknown and must be determined from fits to serial blood and excretion data on the reactant product species.¹³ If transport were nonlinear, the α_{ij} terms would be similarly replaced by other nonlinear functions of concentration.

There is at least one instance of nonlinear pharmacokinetics, potentially of great importance to dose-response extrapolation, which is considerably more complicated than those examples discussed so far. In this situation, a metabolite of the toxic substance, not the substance itself, becomes the toxic agent.

A potential problem with simple compartment models is their failure to describe tissue concentrations that are immediately identifiable with particular organs. This problem is most severe when modeling the nonlinear pharmacokinetics of a toxic substance that is metabolized or transported differently in different organs because widespread lumping of organ spaces into a "tissue compartment" is no longer possible. Furthermore the compartment model parameters, such as the transport constants k_{12} and k_{21} or the distribution volumes, are not identified a

¹²The form of the reaction term shown here is the Michaelis-Menten type, a very common expression from enzyme kinetics, based upon the assumptions of a dynamic equilibrium between substrate (reactant species) and detoxifying enzyme, and first-order kinetic decay of the substrate-enzyme complex. The equilibrium constant is K and the decay rate constant is k . k reflects the mass of enzyme present.

¹³Several computer programs are available for the nonlinear fitting of such data. One specifically adapted for pharmacokinetic use is the NONLIN program, available from C. Metzler at the Upjohn Company, that both integrates the differential equations and performs the statistics. The program provides estimates of confidence limits about the parameters. Comments on the peculiarities of NONLIN as well as other available computer programs appear in Wijnand and Timmer (1979).

priori from physiologically known values of blood flows or organ volumes. As a consequence, a second class of pharmacokinetic model, the physiologic model, has been developed over the last decade (Bischoff et al., 1971; Dedrick et al., 1973a; Morrison et al., 1975; Harrison and Gibaldi, 1977; Ehrlich et al., 1980). These models have the advantage of allowing an a priori estimation of some parameters, providing for some aspects of scaling from one animal species to another, and providing an unambiguous framework for the introduction of tissue-specific metabolism data. They have the disadvantage of requiring experimental toxic substance concentrations and transport constants for a large number of tissues to maintain the models' organ-specific character.

For many toxic substances, however, it is not necessary to retain this level of detail, it being sufficient to have a description of the blood and target tissue spaces with all other spaces included in a lumped region. Thus, compartment models (often with greater than two compartments) will generally be the most economic pharmacokinetic procedures to use in estimating risk. For those other instances that do require organ-specific modeling, we have included a detailed description of the physiologic models in App. B.

PHARMACOKINETICS AND DOSE RESPONSE

In this section we briefly discuss the implications that linear and nonlinear pharmacokinetics have on dose-response estimation. We also present a simple set of response equations, based upon linear pharmacokinetics, that allow preliminary estimates of risk to a human population to be made from animal response data.

The Respective Roles of Linear and Nonlinear Pharmacokinetics in Dose Response

As might be expected, the most significant modifications of dose-response relations stem from the inclusion of nonlinear pharmacokinetics where target tissue concentrations cannot be replaced by simple scalar functions of the administered dose. Of particular importance are the substances that undergo saturable (nonlinear) detoxification in the body. Two of the known examples of such substances are vinyl chloride

(Hefner et al., 1975; Watanabe et al., 1976) and bromobenzene (Reid et al., 1973; Zambaglione et al., 1973). Each of these compounds produces a highly reactive metabolite that is scavenged away by glutathione (a detoxifying agent concentrated in the liver) at low doses. At higher doses, however, where most dose-response experiments are conducted, the supply of glutathione is rapidly depleted and more severe toxicity is noted in the test animals.

This effect can be observed in the relative shape of dose-response curves (a) and (b) in Fig. 6.5. Curve (a) is the type of response curve obtained using a one-hit model and assuming linear (non-glutathione-dependent) pharmacokinetics. Curve (b) takes detoxification kinetics into account. It shows lower risk at low doses than curve (a) because glutathione has not yet been depleted, detoxification occurs, and the target tissue concentration is lower. It is important to observe that at a fixed risk (say, the maximum allowable risk to an individual, P_0), the highest dose consistent with not exceeding this risk can be vastly different between the linear and nonlinear cases (compare d_a and d_b). This may amount to several orders of magnitude.

Fig. 6.5 — Hypothetical dose-response curves for an agent undergoing nonlinear detoxification

Cornfield (1977) has attempted to build such pharmacokinetic considerations into a steady-state dose-response model that has been termed the statistico-pharmacokinetic model. However, we have not considered this model because the pharmacokinetic mathematical formalism that has been built into it is not general enough to include the wide range of pharmacokinetic situations encountered in practice.

Estimating Human Response from Animal Response Data--Simple Equations Based on Steady-State Linear Pharmacokinetics

In this section we present a simple set of dose-response equations, useful for making preliminary estimates of risk to a human population from animal response data. To keep these relations uncomplicated, several simplifying assumptions will be made. Later inclusion of detailed pharmacokinetics can refine this approach.

The assumptions that we make are:

1. Linear pharmacokinetics applies and administered dose is proportional to the target tissue concentration.
2. Three modes of exposure are possible: ingestion, inhalation, and dermal.
3. The dose concentration is constant over the exposure period and steady-state concentrations are obtained in the animal or human subjects.

Dose-Scaling. The dose-response models we presented earlier all involve the target tissue concentration and one or more statistical parameters. Under assumption 1, the target tissue concentration is proportional to dose. However, the constant of proportionality depends on the animal species exposed to the toxic agent because of species-specific absorption, metabolism, and excretion rates. This must be taken into account if the parameter values derived from animal dose-response data are to be used to estimate human response.

This may be accomplished approximately by dose-scaling, a method in which the (steady-state) target tissue concentration is replaced by a term proportional to the concentration of toxic substance in the

exposure medium (air, water, or feed according to assumption 2) and to species-specific average intake rate constants.

The particular forms used can be rationalized by deriving them from a simple one-compartment pharmacokinetic model:

$$V \frac{dc}{dt} = I - k_c c \quad (6.33)$$

where V = volume of distribution of the toxic agent (l),
 I = input rate (mg/day),
 k_c = kidney clearance (l/day),
 c = concentration of toxic agent in the body (mg/l).¹⁴

Assuming steady-state behavior (assumption 3),

$$c = \frac{I}{k_c} \quad (6.34)$$

The kidney clearance is proportional to a power of body weight, even across species, so that it is expressible as $k_c = kW^{.69}$ (Dedrick, 1974; exponent for creatinine clearance). The toxic mass input rate, I , depends upon the route of entry. For ingestion,

$$I = Fc_f r = (hW^{.67})c_f r \quad (6.35a)$$

where c_f = concentration of toxic substance in food (ppm or mg/kg),
 r = fraction of food absorbed,
 F = food intake per day (kg/day).

¹⁴Equation (6.33) is equivalent to the blood compartment of Eq. (6.30) with $k_0 c_0 = I$, $V = V_B$, $k_{21} = k_{12} = 0$, and $k_c = k_e/V_B$. To keep I simple, back flow of toxic agent is ignored and $R = 0$.

The second equality results from the assumption that the mass of food intake is proportional to the calorie need per day which is, in turn, proportional to the oxygen uptake per day, a quantity well-known to be described by $hW^{.67}$ (FASEB, 1971). The constant h is species-specific because the calories/kg of food and activity levels are different between man and laboratory animals (EPA, 1980b).¹⁵ The constant r is species- and toxic-agent-specific. For inhalation,

$$I = R c_a r = (fW^{.67}) c_a r \quad (6.35b)$$

where c_a = concentration of toxic substance in air (mg/m³),
 R = species-specific inhalation rate (m³/day), which is
proportional to $W^{.67}$ as shown by the second equality,¹⁶
 r = fraction of toxic agent absorbed (related to the Henry's
constant of the gas).

Like h , f is species-specific and r is primarily toxic-agent-specific. For dermal exposure,

$$I = K c_w = (gW^{.67}) c_w \quad (6.35c)$$

where K = mass transfer coefficient across total skin area (the second equality shows that this is proportional to the 2/3 power of body weight) (kg/day),
 c_w = concentration of toxic substance in water (ppm or mg/kg).

Here g may be both species- and toxic-agent-specific.

¹⁵EPA begins with $F = hW^a$ but later changes to $F = k'W$ when correcting for species differences in food calorie content. We have derived values of h from their F data but have left the exponent a unchanged to avoid inconsistency.

¹⁶EPA again changed their scaling from $R = fW^a$ to $R = iW$ when accounting for species differences in inhalation rates. We have used the former relation throughout, since within all species, the breathing rates scale by W^a , $a \neq 1$ (FASEB, 1971).

If Eqs. (6.35) are substituted in Eq. (6.34) along with our expression $k_c = kW^{.67}$, the target tissue concentrations (mg/l) take on standard dose forms, i.e.,

$$c = \begin{cases} d_{1s} = c_f h_s r_s / k_s & (c_f = \text{ppm by weight}) \quad (\text{ingestion}) \\ d_{2s} = c_a f_s r_s / k_s & (c_a = \text{mg/m})^3 \quad (\text{inhalation}) \\ d_{3s} = c_w g_s / k_s & (c_w = \text{ppm by weight}) \quad (\text{dermal}) \end{cases} \quad (6.36)$$

where d_{js} = standard dose for route j and species s .

Note that the similarity of all weight exponents has led to the disappearance of weight dependence in the final expressions. The feed and inhalation rate constants are given in Table 6.1. If pharmacokinetic data allowing the determination of r_s (dimensionless), g_s ($\text{kg}^{1/3}/\text{day}$), or k_s ($1/\text{day}/\text{kg}^{2/3}$) do not exist, a default approach may be considered in which it is assumed that no differences in absorption or excretion exist between species.¹⁷ Under this assumption, r_s , g_s , or k_s may be set equal to an arbitrary default value (unity in the Rand program), because any error in this value will become part of the model parameter when fitting animal data and will cancel out identically when later estimating the human response.

Simple Forms of Dose-Response Equations. The dose-response equations shown in Eqs. (6.3), (6.12), (6.23b), (6.25), and (6.25a) can now be written in standard dose forms, consistent with the three assumptions above. The equations derived below have been incorporated as part of the Rand computer program.

¹⁷For k_s to be independent of species, one must assume that, across species, all metabolites are removed by the same clearance mechanism (generally non-active glomerular filtration) and tissue distributions and transport are similar.

Table 6.1
FEED AND INHALATION RATE COEFFICIENTS

Species		h_s	f_s
		(kg ^{1/3} /day)	(m ³ /kg ^{2/3} /day)
Man	(s = 1)	0.1154	1.18
Rat	(s = 2)	0.0352	0.449
Mouse	(s = 3)	0.0404	0.403

Two observations must first be made. First, from empirical observation, the appropriate time variable to use in all our dose-response expressions is not chronological time but fractional lifetime. Second, when estimating human risk from animal response data, the dose-response model must be formulated so that the model parameters obtained from the animal data are directly transferable to the human case and are not confounded by implicit dependence on species-dependent dose-scaling factors or exposure times. Accordingly, the dose-response equations are recast below in a form that clearly identifies the dose-scaling and exposure time functionalities.

One-Hit Model. From Eqs. (6.2) and (6.3) and the standard dose definitions in Eq. (6.36), the one-hit model is

$$P_r = 1 - e^{-\alpha t_s^d j s} \quad \begin{array}{l} \text{(animal response } s \neq 1) \\ \text{(human response } s \neq 1) \end{array} \quad (6.37)$$

where t_s = fraction of lifetime over which exposure occurs.

When $s \neq 1$ and this is used in Eq. (6.28), a set of animal data (including controls) can be fit by a program such as MULTI80 (Rai and Van Ryzin, 1980) to obtain the product αt_s^d . α can be derived from this, combined with the appropriate human exposure time, t_1 , and total dose,

d_{j1} , and substituted into Eq. (6.37) to estimate the total human risk from the toxic agent under consideration. If the total dose, d_{j1} , includes a prospective background dose of the toxic agent originating from a nonutility source, d_b , the added human risk above background should be estimated from Eq. (6.27a).

Multihit Model. From Eqs. (6.2), (6.9), and (6.12) and the standard dose expressions, Eq. (6.36), the multihit model is

$$p_r = \int_0^{\alpha t_s d_{js}} \frac{x^{k-1} e^{-x}}{\Gamma(k)} dx \quad \begin{array}{l} \text{(animal response } s \neq 1) \\ \text{(human response } s \neq 1) \end{array} \quad (6.38)$$

Equation (6.38) is used with Eq. (6.28) and MULTI80 to estimate the product, αt_s , and k . The human response is then estimated in the same way as with the one-hit model.

Crump Model. From Eqs. (6.24) and (6.36),

$$p_{1...k} = 1 - \exp \left[- \sum_{i=1}^k (w_i t_s^k) d_{js}^i \right] \quad \begin{array}{l} \text{(animal response } s \neq 1) \\ \text{(human response } s \neq 1) \end{array} \quad (6.39)$$

where t_s = fraction of lifetime over which exposure occurs.¹⁸

When $s \neq 1$, this expression can be used directly to fit a set of animal response data (including controls). This differs from the hit models because background has already been included in Eq. (6.39). Provided each of the animal and human response cases involves a single t_s (i.e., not age-response data), the fit can be accomplished with GLOBAL79 (Crump and Watson, 1979) yielding best fit values for the set of $w_i t_s^k$ products and k . Knowing t_s for the animal experiments, w_i can be derived,

¹⁸Alternatively, the exponent on time can be set equal to three in accordance with EPA procedures (EPA, 1980a). However this version of the Crump model should be considered a new model and not a close approximate of the actual multistage model.

combined with the human exposure time of interest, t_1 , and total dose, and substituted back into Eq. (6.39) to yield the total human response estimate due to the toxic agent of interest. If the total dose, d_i , includes a prospective background dose of the toxic agent from a nonutility source, d_b , the human added risk above background should be calculated from Eq. (6.26).

Probit Model. This model, as presented in Eq. (6.25), differs from the hit and multistage models because it does not have an explicit dependence on time. As we have already noted, one approach to deal with this uses equivalent lifetime doses¹⁹ whenever exposures are less than full lifetime, i.e.,

$$d_e = dt$$

where d_e = equivalent dose,
 d = actual (standard form) dose,
 t = actual exposure time.

Using this approach, the probit becomes

$$P_r = \frac{1}{\sqrt{2\pi}} \int_{-\infty}^{\alpha + \beta \log(d_{js} t_s)} e^{-x^2/2} dx \quad \begin{array}{l} \text{(animal response } s \neq 1) \\ \text{(human response } s = 1) \end{array} \quad (6.40)$$

α and β can be estimated by substituting Eq. (6.40) into Eq. (6.28) and, using SAS (1979), for example, fitting the latter to animal response data (including controls). The human added risk above background is then estimated from Eq. (6.27a) using Eq. (6.40) along with the human total dose, d_{j1} , nonutility background dose, d_b , and exposure time, t_1 .

¹⁹An improved alternative formulation suggested by Druckrey (1967) increases the dependency of the response probability on exposure time by replacing the upper limit of Eq. (6.40) by $\alpha + \beta \log d_{js} t_s^n$. The geometric mean value of n over several tumor types and carcinogens is 4. If this alternative is followed, n may be treated either as another free parameter or, if data are limited, set equal to the mean value of 4.

Weibull Model. From Eq. (6.25a) and the standard dose definitions in Eq. (6.36), the Weibull model is

$$P_r = 1 - \exp(-\alpha d_{js}^k t_s^n) \quad (6.41)$$

where t_s = fractional lifetime over which exposure occurs
(dimensionless).

α , k , and n can be estimated from animal data by substituting Eq. (6.41) in Eq. (6.28) and maximizing the likelihood function by an appropriate version of the PAR or P3R routines in the BMDP computer package (BMDP, 1981). The human added risk above background can then be estimated from Eq. (6.27a) by substituting Eq. (6.41) into it with the appropriate human exposure time, t_1 , total dose, d_{j1} , and nonutility background dose, d_b .

SELECTION OF DOSE-RESPONSE MODELS

None of the dose-response models available for estimating human response is to be preferred over others as having a more sound molecular biologic-biochemical basis. Selection of the best model for a given toxic substance on this basis, or the development of a new improved model, awaits further characterization of the transformation and subsequent expression events involved in carcinogenesis. Hints exist that some kind of staging model may be involved in the development of human cancer such as T24 human bladder cancer. It has been shown for this cancer that very small changes may occur in normal human DNA (c-bas) sequences (on the order of chemical changes induced by carcinogens) that lead to tumor formation (Santos et al., 1982). Results such as these are suggestive that the qualitative nature of carcinogenesis may soon be unfolded for a few specific tumors, but quantitative results for most tumors still seem distant.

This biological situation forces one to analyze data with dose-response models selected for other, more pragmatic reasons. One

important consideration is that models may be chosen only if there are sufficient data points available (dose-response pairs) to allow determination of the parameters.

If just one dose-response pair (plus control) is available, only the one-hit or Mantel-Bryan model can be parameterized. If two dose-response pairs (plus control) are available, the multihit, probit (nonexposure time-dependent), Weibull (nonexposure time-dependent), and multistage ($k \leq 2$) models can and should be used in preference to the one-hit and Mantel-Bryan models. If age-response data (i.e., dose, exposure time, response data) are available for three or more data sets with at least two doses and two exposure periods represented (plus control), then all the models may be considered. While response predictions (VSDs) may then be obtained from all parameterized models, generally those derived from the models that have the best goodness-of-fit measures are taken as "best." Of course it should be stressed that even an excellent fit in the observed data range does not mean that the associated model is structurally correct and capable of yielding accurate low-dose extrapolations.

When dose-response models are not calibrated from animal data but rather from epidemiological data, many data reduction problems involving the matching of exposed and control populations are encountered (e.g., are they matched in age, sex, race, and other disease factors?). Except for one, we do not discuss these problems, mentioning them only so that utility environmental health specialists are aware of such difficulties and check for their treatment in epidemiological data references. The one point that we do mention is the following. Epidemiological age-response studies often present data in the form of dose-time-incidence (rather than prevalence) data. Because incidence is defined as the rate of cancer induction in a population per unit time, the prevalence equations (P_r) that we have presented in this report must be differentiated with respect to time before attempting to fit these incidence data. (Note, however, that to use these differentiated equations without further modification requires that all members of the epidemiological population be exposed to the toxic agent from birth. Otherwise age-time effects must also be taken into account.)

SOURCES OF UNCERTAINTY IN RISK ESTIMATION

Throughout this chapter, we have discussed various ways by which uncertainty is introduced into estimates of risk. In this section, we collect these sources of uncertainty together and add others which have not yet been presented.

Selection of Dose-Response Model

Selection of the model is one of the major factors contributing to risk uncertainty. As we have shown above (Fig. 6.3), the differences between virtual safe doses (at the 10^{-6} risk level) may vary by four orders of magnitude depending on the models compared. Fundamental questions regarding the biochemical definition of a hit, the role and kinetics of DNA repair, the validity of assuming the additivity of background and toxic agent effects, and the accuracy of the methods for including time dependence in the statistical models are some of the underlying difficulties. This uncertainty will not be significantly reduced until more knowledge is available regarding metabolic and molecular biologic kinetics or new, far more sensitive carcinogenic assays are developed.

Error Resulting from Extrapolation

Even after a dose-response model is selected, the extrapolation away from the high-dose range to the low-dose domain leads to further uncertainty. The confidence bands about any mean estimate generally flare greatly as the region of experimental data is left. This effect is exemplified in the benzopyrene data of Fig. 6.3 where the Crump mean (maximum likelihood) and lower confidence lines are plotted. At the 10^{-6} risk level, the difference in the dose variables is two and half orders of magnitude.

Pharmacokinetics

The simple dose-response equations in the previous section are subject to considerable error because of the assumption of linear pharmacokinetics. The error introduced by this assumption depends on the toxic substance involved and can only be reduced by collecting

detailed pharmacokinetic data. The simplest such approach would involve the determination of toxic agent time histories in blood following various dosages. All the curves should be dose-scalable if the pharmacokinetics are linear. If the substance has nonlinear pharmacokinetics, such as vinyl chloride, then the continued use of linear pharmacokinetics and a dose-response model (such as the probit) can lead to uncertainties in the VSD on the order of five orders of magnitude at a risk level of 10^{-5} (Gehring et al., 1978). A more accurate treatment would involve the formulation of nonlinear pharmacokinetic models including realistic representations of the biochemistry involved.

Uncertainty in Environmental Dose

The amount of toxic agent to which a human is exposed in the environment is subject to considerable uncertainty depending on how well the environmental microbiology, chemistry, and transport are known. Since this dosage is the "administered dose" input of the pharmacokinetic model, any errors in this dose will be reflected in a target tissue dose of increased uncertainty.

Interspecies Scaling

The use of animal data to estimate human response introduces uncertainty. The uncertainty stems both from the quantitatively different tissue distribution and detoxification pathways between species (pharmacokinetic uncertainties) as well as a differential sensitivity to the genetic effects of a toxic substance (molecular biologic uncertainty implicit in the dose-response parameters). The pharmacokinetic uncertainty can be reduced by selecting test animal species that are known to exhibit metabolism similar to humans. The genetic effect uncertainty is difficult to reduce other than by matching tumor types between animal and human.

Sensitive Subgroups

Tumorigenic response may differ between animals or humans of different sex, age, dietary habits, genetic makeup, etc. Laboratory

animals of different strains, for example, often exhibit considerably different responses to the action of a carcinogenic agent. If these differences are to be taken into account, separate dose-response data must be available for each sensitive subgroup. Human epidemiologic data frequently give response breakdowns by sex, age, smoking habits, and other key risk factors but such extensive data are available for only a limited set of toxic substances.

Estimating the Size of the Exposed Population

When estimating the absolute prevalence of human cancers resulting from an environmental toxic agent, the size of the population at risk must be estimated. In general, this population is migratory, so both the number of individuals exposed and the length of time of their exposure is highly variable. For example, if a small community located near a power plant undergoes rapid growth during the lifetime of a plant, the added cancer risk will depend strongly on when this growth occurs. While this effect may be approximately taken into account by using a time-averaged expected population level, a more accurate approach would use a combined population and dose-response model. However, the uncertainty of the long-term demographic projection of population changes would still remain in the final risk estimate.

7. THE ECONOMIC ANALYSIS

INTRODUCTION

The regulation of toxic pollutant discharges from coal-fired power plants will impose several types of costs on society. These costs include both direct costs of complying with the regulations and secondary costs resulting from the direct costs. In a complete analysis of the costs of regulating toxic pollutants, both types of costs should be considered.

Although some of the direct costs are paid by the government, most are imposed on the utility industry. Utility costs include the capital and operating expenditures associated with installing and operating necessary control equipment. They also include any changes in utility profits caused by the control program. The government must pay the costs of administering the program, including monitoring and enforcement expenses.

Secondary costs arise from the changes in direct costs. Increased direct costs may lead to higher electricity rates. These higher rates will not only affect the electricity costs and spending decisions of the public but will also increase the costs of all businesses that use electricity. Some businesses may have to increase prices, which in turn may affect both sales and employment in the region surrounding the power plant. Although there may be compensating effects from the increased purchases of control equipment by the utility, these may not occur in the plant area.

In our cost assessment, we consider only the direct control costs paid by the utility. Including secondary or indirect costs would require an extensive economic analysis beyond the scope of this project. In addition, under the provisions of the Clean Water Act, the government is not required to consider such general costs in determining the appropriate level of control. The direct costs include the capital expenditures for equipment as well as the operating and maintenance costs of using that equipment. We have derived the methodology and database for our cost analysis from (1) the EPRI Technical Assessment

Guide (EPRI, 1979); (2) the Coal Ash Disposal Manual (Bahor et al., 1979, 1981); (3) EPA (1980a); (4) Colley et al. (1978); and (5) Knight et al. (1980).

Each of these documents provides us with different types of information. EPRI (1979) is a general reference that describes the EPRI costing methodology. Bahor et al. (1979, 1981) and Knight et al. (1980) identify technical and cost data applicable to coal ash and FGD sludge disposal. Finally, EPA (1980a) and Colley et al. (1978) specify cost information for a range of specific power plant effluent handling systems and waste treatment technologies.

EPRI (1979) details a series of assumptions, data, and methodology intended for EPRI and EPRI contractor use as a basis for assessing the value of alternative research and development programs and projects. This material comes from many sources, not all of which agree. Because of this, the EPRI data have been normalized to achieve consistency.

In providing the data and methodology, EPRI specifies several basic assumptions and a set of conditions. First, all data are provided on a national basis, and, where appropriate, on a regional basis. Second, the data provided are not intended to be exhaustive, but rather they are intended to offer a basis from which a broader set of case studies could be developed. Third, EPRI presents the then current regulatory and economic conditions of the utility industry and the public utility commissions. Clearly these conditions are not necessarily consistent across utilities, and utility commissions are likely to change over time. Thus the EPRI guide is based on current, average conditions.

The methodology used to compare costs for alternative systems is called the "revenue requirement method." This method, highly appropriate for utilities, estimates the amount of revenue that must be collected from customers to compensate a utility for all expenditures associated with the implementation of alternative decisions involving money. The revenue requirement of an alternative is the discrete charge associated with that alternative and is to be used for determining the economic consequence of that alternative.

To compare the revenue requirements of alternative investments, one must calculate the levelized equivalent of these requirements. The levelized value of a stream of revenue requirements is constant over the

entire period under consideration, such that the sum of the present worths of the annual levelized values just equals the sum of the present worths of the actual annual revenue requirements. This levelized annual value of a stream of annual revenue requirements is calculated by first multiplying each year's revenue requirements by the appropriate present worth factor and summing the resulting present worth values. The single present worth value is then converted to an equal payment annuity by multiplying the result by the capital recovery factor. The capital recovery factor is equal to the return plus the sinking fund depreciation.

The remaining documents are more specialized. Bahor et al. (1979) present a method for making an economic comparison of alternative coal ash disposal systems developed for new power stations. EPA (1980a) specifies the cost, energy, and land requirements of the various treatment and control technologies that the EPA has recommended for steam-electric plants. Colley et al. (1978) presents estimates of the capital and operating expenses for wastewater treatment and disposal technologies that were found to be most applicable to the control of priority pollutants in the utility industry. Finally, Knight et al. (1980) provide information that can be used to make first-order estimates of the costs of FGD sludge disposal systems.

Although these documents are in many instances quite supportive, many of their calculations are based on rather diverse assumptions. Rather than detail each of these diversities at this time, we can state that we considered the assumptions used in the EPRI studies to be the standard. We therefore adjusted, normalized, and extrapolated the data in the remaining studies accordingly, as required.

The primary database and relevant assumptions drawn from these documents are discussed in the cost calculations for the case study. These calculations are presented in Chap. 8.

COST ALLOCATION

Control procedures implemented to reduce discharges of one priority pollutant may also reduce discharges of other pollutants. Consequently, it may not be appropriate to allocate the entire control cost to the primary toxic material. In effect we are assigning either an upper

bound to the cost or a lower bound to the benefits of the control measures. To avoid this we should ideally either include the additional benefits of controlling the other pollutants or allocate only some appropriate fraction of the control cost to the original priority pollutant.

Economists have discussed for years the problem of allocating joint costs. In spite of the extended debate, no one has ever produced the "correct" method to do this. In essence, all proposed approaches for such cost allocation are arbitrary. With respect to pollution control costs, these approaches include:

- Uniformly distributing costs over all chemicals controlled.
- Uniformly distributing costs over all chemicals controlled and regulated.
- Allocating costs based on relative amounts of control and relative risks associated with emissions of each chemical.

Other alternatives have been proposed, but none of them has more validity than the others.

In practice, it should not be necessary to allocate control costs. When a utility investigates the alternatives that it can use to meet control regulations for a toxic pollutant, it should compare the total net costs of all alternatives that meet the regulation. There is no need to allocate the control costs between pollutants if only one pollutant must be controlled. The situation does not change for two or more regulated pollutants. Schemes that control both suitably should be compared on the basis of their total costs, without dividing these costs between substances.

We can compare the total costs of alternative schemes, so long as the benefits are similar. In the case of utility decisions when faced with control regulations, the benefits can be defined to be either meeting or not meeting the regulations. All options that meet the regulations have the same benefits--so long as costs are defined to include monetary benefits associated with each option. For government officials and those making regulatory decisions, the benefits must

include risk reduction associated with all controlled chemicals. Although this may involve additional risk assessment, it avoids having decisions being made on the basis of some completely arbitrary cost allocation scheme.

8. CASE STUDY

INTRODUCTION

To demonstrate how the overall methodological framework would be used, we have applied it to a simplified, illustrative case study. In this case study we investigate and compare the costs and risks associated with controlling arsenic emissions from a hypothetical coal-fired power plant located on the Hanford Nuclear Reservation near Richland, Washington. The plant is sited adjacent to the Columbia River, which is used as a source of fresh makeup water and as the receiving waters for plant discharges. Although we calculate all arsenic emissions from this plant, we will limit our assessment of control costs and risks to those associated with regulating the direct discharges from the power plant. More specifically, as described in Chap. 2, we compare the costs and risks associated with alternative control strategies for the ash handling and disposal system of the plant.

We have not incorporated emissions to the atmosphere and soil or groundwater for several reasons. The emphasis in our project has been to develop a methodology that could be applied to surface water pollution. Emissions to the atmosphere and groundwater have been, or are being, considered in parallel work. Consequently, although our methodology can incorporate discharges to all media, as well as the subsequent intermedia transfers, we have not done so in this initial case study. We can demonstrate the general application of the methodology concentrating on direct discharges. Future work will present a more integrated and complete case-study analysis.

In the sections that follow, we will describe the power plant, site, and relevant risk and cost analysis. On the basis of preliminary calculations, we determined that incorporating only direct surface water discharges was not unreasonable for the plant and site considered. Ignoring the long travel times of groundwater and the attenuating effects of adsorption on the soil matrix, we found that ash pond leaching would have to be unusually large to be significant compared to

direct discharges. Similarly, surface water contributions from atmospheric discharges would also be small. Although total arsenic emissions to the atmosphere are important, because of meteorological and hydrologic conditions, only a small fraction of these emissions will reach the Columbia River. Therefore, we should be able to ignore these emissions also, without creating significant errors that would diminish the value of the case study.

DESCRIPTION OF CASE STUDY

Plant Site

We assume the power plant is located near the north central border of the Hanford Nuclear Reservation northwest of Richland, Washington. As shown in Fig. 8.1, the plant site is adjacent to the Columbia River. In this area the Columbia is fast and deep, with flows varying between 36,000 and 450,000 cubic feet per second (cfs). The region is characterized by semi-arid climate and relatively flat terrain. Although the surrounding land is not heavily populated, some of it is used for the Hanford Reservation, irrigated and dry land agriculture, wildlife preservation, hunting, and recreation. Principal cities within 50 miles of the plant include Yakima, Richland, Kennewick, and Pasco.

We obtained detailed geological, meteorological, hydrological, and geographic data for this location from Safety Analysis and Environmental Impact Reports (WPPSS, 1972, 1973a, 1973b) for nuclear power plants proposed for the reservation.

Pollutant

For several reasons, we have chosen to use inorganic arsenic emissions from the hypothetical power plant as the subject of our study. First, as discussed earlier, 13 inorganic trace elements are found more frequently than organic priority pollutants in power plant discharges and constitute a greater potential health hazard. Second, information about the use of organic pollutants in power plant processes and their discharge in liquid waste streams is not sufficient for control cost and risk analysis. Although data about the trace elements and understanding of their behavior in power plants are limited, they are generally far

SOURCE: Onishi et al. (1982).

Fig. 8.1 – Power plant location for our case study

better than information about the organic priority pollutants. Third, of the 13 toxic trace elements commonly found in plant discharges, only arsenic, lead, and mercury have both well-documented chronic health effects and sufficient quantitative data to develop dose-response relationships. Fourth, of these three trace elements, only arsenic is known to be carcinogenic through both inhalation and ingestion and appears in significant concentrations in numerous power plant emissions.

Virtually all arsenic emitted by power plants enters as a component of the coal supply. During combustion, most of this arsenic will be volatilized and will emerge from the furnace as arsenic trioxide in the flue gas. As the flue gas cools, the arsenic condenses to form

particulates. The particulate arsenic not removed by the plant's flue gas cleaning equipment is emitted to the atmosphere in particulate form. Arsenic in the collected fly ash, bottom ash, and FGD sludge may be discharged to surface waters or may leach into groundwater as a result of handling, transport, treatment, storage, and disposal operations. Arsenic in the plant makeup water may also be discharged as a result of cleaning or water treatment, or in blowdown streams from boilers and cooling systems. Finally, arsenic in the plant coal supply may reach surface water or groundwater as a result of runoff and leaching from coal storage piles.

Although arsenic may appear in several forms in plant waste streams, we have chosen to combine these forms into a single general species. This considerably simplifies the analysis, because very little is known about the forms and behavior of arsenic species in power plants and the environment. Where the behavior of different species might be important, we will try to discuss how the variations would affect the study calculations.

Power Plant

In our study, we have chosen to investigate a baseload coal-fired power plant having a nominal generating capacity of 500 MWe. The characteristics of this hypothetical plant are given in Table 8.1. A schematic diagram of plant water systems is shown in Fig. 8.2. Fresh water for all uses is withdrawn from the Columbia River, passed through plant systems, and returned as overflow from either the combined ash basin or runoff collection pond. We will discuss these systems and other assumptions in more detail as necessary later in the analysis.

PLANT EMISSIONS

Coal Composition

The composition and properties of coal have been extensively described in the literature (EPRI and EPA, 1979; Bahor et al., 1979; Braunstein et al., 1977; Dvorak et al., 1978; Kuhn et al., 1980; Smith, 1980). For the purposes of our analysis, five characteristics of coal are important. These are the (1) heating value, (2) ash content, (3)

Table 8.1

DESCRIPTION OF NOMINAL CASE-STUDY POWER PLANT

Characteristic	Assumption
Generating capacity	500 MWe
Capacity factor	0.60
Thermal efficiency	0.37
Boiler type	Tangentially fired pulverized-coal burners; dry bottom furnace
Cooling system	Mechanical draft cooling towers
Air pollution control system	Cold-side electrostatic precipitator with lime wet scrubber for SO ₂ removal

Fig. 8.2 – Schematic diagram of power plant water systems

moisture content, (4) sulfur content, and (5) arsenic concentration. From the above references we have summarized in Table 8.2 normal ranges and typical values for these parameters.

It is evident that arsenic concentrations in coal can vary over wide ranges. This variation occurs not only between coal type and location, but also within seams at a given location. The overall average concentration, however, is close to the mean crustal abundance of arsenic, 5.0 to 6.0 ppm (EPRI and EPA, 1979; Knight et al., 1980; Braunstein et al., 1977; Heit, 1977).

For the purposes of our case study, we have assumed a maximum arsenic concentration of 106.0 ppm and a nominal concentration of 6.0 ppm. We have also chosen a typical Illinois subbituminous coal with 15 percent ash, 10,000 Btu/lb heating value, and 4 percent sulfur. Although this high sulfur, high ash coal would not normally be used by a power plant in Washington, it provides a wide range of emissions for the calculations.

Table 8.2
NORMAL VALUES FOR COAL CHARACTERISTICS

Property	Minimum	Maximum	Typical
Heating value (Btu/lb)	7,100	14,300	10,000
Ash content (%)	1.9	16.0	10.0
Moisture content (%)	1.1	36.9	10.0
Sulfur content (%)	0.1	5.0	2.0
Arsenic content (ppm)	0.5	106.0	5.0

Coal Consumption and Arsenic Production

The daily coal requirement of a coal-fired power plant is given by

$$M_c = 37140.08W/(e_{pl}h) \quad (8.1)$$

where M_c = daily coal consumption (t/day)

W = rated plant capacity (MWe),
 γ = plant capacity factor,
 e_{pl} = plant efficiency,
 h = coal heating value (Btu/lb).

Arsenic release will depend on coal consumption rates and coal arsenic concentration. The potential daily release rate of arsenic (or other toxic pollutant present in the coal) can be calculated from the daily coal consumption rate using

$$P_c = 2205.0M_c p_c \quad (8.2)$$

where P_c = pollutant content of coal consumed (lb/day),
 p_c = pollutant concentration in coal (lb/lb).

The emitted arsenic may be released to the environment in several ways, the most important of which are:

- Atmospheric release in flue gas and fly ash or as dust from coal piles and ash disposal sites.
- Waterborne release via direct discharge from the plant or as runoff from coal piles and ash disposal areas.
- Discharge to soil and groundwater by leaching from coal piles, ash disposal areas, and FGD sludge disposal areas.

The specific distribution of arsenic in plant discharges will depend on the equipment, processes, water management, and pollution control technologies used in the plant. This has been discussed in more detail in Chap. 3 and App. D.

Atmospheric Emissions

For a dry bottom, tangentially fired, pulverized-coal furnace, total ash production will be partitioned as 80 percent fly ash (FA) and 20 percent bottom ash (BA) (EPA, 1980a). We can calculate the partitioning of our pollutant between fly ash and bottom ash using the

methods of Dvorak (1977) based on the work of Klein et al. (1975a). This method relies on partitioning factors developed from measurements of trace element concentrations at different points within a power plant. To use this approach, we first determine with Eqs. (8.1) and (8.2) how much of the pollutant enters the plant through its daily coal consumption. With this information, the amount of pollutant leaving the furnace in the collected bottom ash can be found using the partitioning factors from

$$P_{ba} = P_c F_{ba} f_{ba} \quad (8.3)$$

where P_{ba} = pollutant content of bottom ash (lb/day),
 F_{ba} = bottom ash fraction of total ash (dimensionless),
 f_{ba} = partitioning factor for bottom ash (dimensionless).

The partitioning factor represents the ratio of arsenic concentration in the bottom ash to concentration in the coal. For arsenic, this fraction has the value 0.39.

The amount of pollutant in the fly ash can be found by subtracting the bottom ash fraction from the total input of pollutant, as shown below.

$$P_{fa} = P_c - P_{ba} \quad (8.4)$$

where P_{fa} = pollutant content of fly ash (lb/day).

Arsenic or other trace element pollutants in the fly ash will be captured by both the ESP and the FGD scrubbing system. If we assume that the plant meets EPA's new source performance standards (NSPS) for particulate emissions under the Clean Air Act, we can find a minimum total collection efficiency for the particulate control system. For power plants the standard is defined to be 0.3 lb/million Btu of energy input. Energy input is the total heat content of the coal consumed by the power plant. Thus, we can calculate the permitted particulate emissions from our plant as follows.

$$E_{pt} = (6.615 \times 10^{-4}) M_c h \quad (8.5)$$

where E_{pt} = particulate emission rate (lb/day).

Total fly ash and bottom ash generation by the plant can be calculated from

$$G_{fa} = 2205.0 M_c A_c F_{fa} \quad (8.6)$$

$$G_{ba} = 2205.0 M_c A_c F_{ba} \quad (8.7)$$

where G_{fa} = fly ash generation rate (lb/day),
 G_{ba} = bottom ash generation rate (lb/day),
 A_c = ash content of coal (dimensionless),
 F_{fa} = fly ash fraction of total ash (dimensionless).

With this information, we can determine the required particulate control efficiency from

$$e_{ptr} = 1.0 - E_{pt}/G_{fa} \quad (8.8)$$

where e_{ptr} = required particulate control efficiency (dimensionless).

Given a minimum or assumed control efficiency, fly ash collection and emission rates can easily be determined using

$$D_{fa} = G_{fa} e_{pt} \quad (8.9)$$

$$E_{fa} = G_{fa} - D_{fa} \quad (8.10)$$

where D_{fa} = fly ash collection rate (lb/day),
 E_{fa} = fly ash emission rate (lb/day),
 e_{pt} = particulate control efficiency (dimensionless).

To simplify the analysis we will assume that all particulate and arsenic collection is accomplished by the plant ESP. This does not affect the determination of atmospheric emission rates but makes the direct discharge calculations less difficult and easier to understand. It also provides more of a worst case analysis, because all collected arsenic will then be transported to the combined ash basin. The overflow from this basin passes directly into the Columbia River. Arsenic removed by the FGD system would be retained in the sludge, which is sent to a separate disposal site isolated from the surface water network. Any arsenic present in the blowdown from the FGD system will also end up in the combined ash basin, as shown in Fig. 8.2.

We can now calculate the amounts of pollutant emitted from the plant and retained in the collected fly ash. Emitted pollutant can be found from

$$P_{efa} = P_{fa}(1.0 - e_{pt})f_{fa} \quad (8.11)$$

where P_{efa} = pollutant emitted in stack gas (lb/day),

f_{fa} = partitioning factor for emitted fly ash (dimensionless).

Retained (collected) pollutant is determined by subtracting emitted pollutant from total pollutant as shown below.

$$P_{rfa} = P_c - P_{ba} - P_{efa} \quad (8.12)$$

where P_{rfa} = pollutant retained in collected fly ash (lb/day).

This last result represents the total amount of pollutant retained in the fly ash or FGD sludge, depending on the design and relative removal efficiencies of the air pollution control system.

Using Eqs. (8.1) through (8.12), we have calculated the amounts of arsenic emitted and collected in bottom ash and fly ash for the case-study power plant. These results and assumed values are shown in Table 8.3.

Table 8.3

ATMOSPHERIC EMISSION CALCULATIONS AND ASSUMPTIONS

Variable	Calculated or Assumed Value
W	500 MWe
γ	0.60
e_{pl}	0.37
h	10,000 Btu/lb
M_c	3,011.1 t/day
P_c	106×10^{-6} lb/lb
P_c	703.8 lb/day
F_{ba}	0.20
f_{ba}	0.39
P_{ba}	54.90 lb/day
P_{fa}	648.9 lb/day
E_{pt}	1.992×10^{-4} lb/day
A_c	0.15
F_{fa}	0.80
G_{fa}	7.967×10^5 lb/day
G_{ba}	1.991×10^5 lb/day
e_{pt}	0.990
D_{fa}	7.887×10^5 lb/day
E_{fa}	7.967×10^3 lb/day
f_{fa}	4.0
P_{efa}	25.95 lb/day
P_{rfa}	622.8 lb/day

Surface Water Emissions

As we discussed in Chap. 3, power plants potentially may discharge to surface waters a large number of different waste streams. Under normal circumstances, however, many of these waste streams will not contain significant amounts of arsenic not present in plant intake water. These waste streams include

- Boiler blowdown.
- Wastes from clarification, ion exchange, reverse osmosis, evaporation, lime-soda softening, and other makeup water treatment processes.
- Floor and yard drains and spills.
- Laboratory and sampling streams.
- Miscellaneous sanitary waste streams.
- Metal cleaning wastes including boiler tube cleaning, boiler fireside washing, and air preheater washing.

Total discharges from these streams should be no more than about one million gallons per day (mgd) (CH2M, 1980; EPA, 1980a; Rice and Strauss, 1977). We have included these streams in our plant water system diagram (Fig. 8.2) for clarity. As shown in the figure, we assume that boiler blowdown is used for fly ash sluicing and that the other waste streams are sent to the combined ash basin. Because these streams generally do not make significant contributions to total arsenic emissions, we will not consider them further in the analysis. Instead, we will concentrate on potentially more important sources of arsenic emissions.

Flue Gas Desulfurization Scrubber. We selected a lime wet scrubber as the FGD system for our hypothetical coal-fired power plant. Under normal circumstances, this system would remove some of the arsenic passing through the particulate control equipment. As discussed earlier, however, we have assumed that all arsenic collection is accomplished by the ESP. Consequently, we will not attempt to calculate arsenic emissions from the scrubber sludge disposal system. Arsenic that may normally be present in the recirculating scrubber liquid will

be discharged in the blowdown stream that passes into the combined ash ponds in our simplified scheme. In a more comprehensive analysis, where groundwater emissions must be considered, it would be necessary to partition the arsenic between collected ash and scrubber sludge, and to consider leaching from the sludge disposal system.

Coal-Pile Runoff. Measurements of trace element concentrations in coal-pile runoff are limited to a few reports (USDOE, 1979; EPA, 1980a; Cox et al., 1979). In general, priority pollutant concentrations in coal-pile runoff depend on the pH of the infiltration water. The pH, in turn, depends on the rainfall and coal characteristics. Normally, the pH of runoff should not be extreme, and trace element concentrations will be small (less than 1 percent of the concentrations in the raw coal). Accordingly, measurements of arsenic in runoff have found concentrations between 0.005 and 0.6 ppm, with mean values ranging between 0.02 and 0.17 ppm (EPA, 1980a). Arsenic concentrations in the raw coal were not reported with these measurements, however, so we can not conclude anything about the relative values.

The amount of runoff from a storm can be calculated from the following relationship developed for the EPA (1974):

$$R = 0.855F + 0.0082 \quad (8.13)$$

where R = runoff (acre-in./acre),

F = precipitation (in.).

Using Eq. (8.13) and assuming that the arsenic concentration in runoff is 2 percent of that in the raw coal, we can estimate the contribution from coal-pile runoff from

$$P_{cp} = 12.418RAp_c \quad (8.14)$$

where P_{cp} = priority pollutant content of runoff water (lb/day),

A = area of coal storage pile (acres),

p_c = priority pollutant concentration in coal (lb/lb).

In this calculation we have converted annual precipitation to a mean daily precipitation rate and converted acre-in. to pounds of water. Normal annual precipitation in the power plant region averages 6.4 in. Because this value is atypically small for power plant sites, we have also calculated runoff rates for an annual precipitation rate of 35 in. These results are presented in Table 8.4. As shown in Fig. 8.2, the power plant collects coal-pile and area runoff together in a pond and discharges the overflow into the Columbia River.

Table 8.4
ARSENIC DISCHARGES IN COAL-PILE RUNOFF

Variable	Calculated or Assumed Value
A	5.5 acres
p_c	106×10^{-6} lb/lb
F--normal	6.4 in./yr
--enhanced	35.0 in./yr
R--normal	5.48 acre-in./acre
--enhanced	29.9 acre-in./acre
P_{cp} --normal	0.03967 lb/day
--enhanced	0.2167 lb/day

Cooling Water. The case-study power plant uses mechanical draft cooling towers. The only significant discharge from this type of system is cooling tower blowdown. To calculate the blowdown rate, we first determine the heat rejection requirements of the power plant, based on its thermal efficiency. From CH2M (1980) we know that cooling water carries off 45 percent of the total heat input to the plant. Therefore,

$$H = 107.48 \dot{W} / e_{pl} \quad (8.15)$$

where H = heat transferred to cooling water (kg-cal/s).

If we assume a water temperature increase of 20°F (11.1°C), the once-through cooling water requirements for our plant can be found from

$$F_c = 2054.4H \quad (8.16)$$

where F_c = once-through cooling water flow (gpd).

In our recirculating system, the cooling water passes through the condenser several times. After each pass, the water is sent through a mechanical draft cooling tower where heat is removed through evaporative cooling. The evaporative losses cause concentration of dissolved nonvolatile pollutants and suspended material in the remaining water. The cycles of concentration measure how much these concentrations increase from the intake water. In addition to evaporation, water is lost through drift, the entrainment of water droplets in the tower air stream. From EPA (1974) we can calculate the blowdown rate for the cooling system given the drift and evaporation rates, and cycles of concentration, using

$$c = (b + v + d)/(b + d) \quad (8.17)$$

which becomes, on rearranging,

$$b = [v - d(c - 1)]/(c - 1) \quad (8.18)$$

$$F_b = bF_c \quad (8.19)$$

where b = blowdown rate (percent),
 v = evaporation rate (percent),
 d = drift rate (percent),
 c = cycles of concentration (dimensionless),
 F_b = blowdown flow rate (gpd).

We assume that (1) the plant operates at five cycles of concentration, (2) the drift rate is 0.005 percent, and (3) the evaporation rate is 1.0 percent per 10°F temperature change (CH2M, 1980; EPA, 1974). Because we are working with a 20°F temperature increase, the overall evaporation rate is 2.0 percent. The cooling water makeup rate is simply the sum of evaporation, drift, and blowdown, and is given by

$$m = b + d + v \quad (8.20)$$

where m = makeup water rate (percent).

Using Eqs. (8.17) to (8.20), we can calculate the cooling water requirements and blowdown rates. These are shown in Table 8.5.

Arsenic is not added to the cooling water in any of the chemicals commonly used for control of biological growth, scale, or corrosion. Consequently, the only arsenic present in the cooling water discharge will be that present in the makeup water, although the concentration may

Table 8.5

COOLING SYSTEM CHARACTERISTICS

Variable	Calculated or Assumed Value
H	87,146 kg-cal/s
F_c	1.790×10^8 gpd
c	5
d	0.005 %
v	2.0 %
b	0.495 %
m	2.5 %
F_b	8.862×10^5 gpd

be greater because the cooling water is recycled through the system (EPA, 1980a). For the purposes of our case study, we have assumed no additional contribution of arsenic to plant discharges from cooling water blowdown and have used the blowdown as bottom ash sluice water.

Ash Transport Water. In the base case, the power plant uses wet handling for both bottom ash and fly ash. Bottom ash and slag are collected from the furnace hoppers, ground, and mixed with water for sluicing to the ash pond. Fly ash collected by the ESP system is pneumatically conveyed from the hoppers to a water ejector, where it is mixed with water to form a slurry. This is pumped to the ash pond. Collected economizer and air preheater ash are also collected in water-filled tanks and pumped to the ash pond.

From CH2M (1980) we can calculate the requirements for fly ash and bottom ash transport water, using the following assumptions:

- Sluiced fly ash has a 5 percent solids content.
- Sluiced bottom ash has a 1 percent solids content.
- Bottom ash and fly ash are treated in a combined pond system.
- Pond overflow is 98 percent of sluice water.

On this basis, we can calculate ash transport discharges from our previous results using the following relationships.

$$F_{ba} = 11.86G_{ba} \quad (8.21)$$

$$F_{fa} = 2.277D_{fa} \quad (8.22)$$

$$F_o = .98(F_{ba} + F_{fa}) \quad (8.23)$$

where F_{ba} = bottom ash sluice water (gpd),

F_{fa} = fly ash sluice water (gpd),

F_o = sluice water discharge rate (gpd).

The concentration of arsenic in the ash transport water can be found from these sluice water flow rates and the arsenic content of the collected fly ash and bottom ash. In mathematical terms we have

$$p_s = 1.198 \times 10^5 (P_{rfa} + P_{ba}) / (F_{ba} + F_{fa}) \quad (8.24)$$

where p_s = pollutant concentration in combined ash transport water (ppm).

The concentration of arsenic in the combined ash pond sluice water will not be the concentration in the pond overflow. Suspended materials will settle out in the pond and dissolved metals may be precipitated. Unfortunately, the chemistry is particularly complex, not understood, and dependent on the characteristics of the individual wastes in the pond. Consequently, we must make simplifying assumptions to estimate the arsenic concentration in our overflow discharge.

From EPA (1980a) we obtained Tennessee Valley Authority data describing plant operation conditions and arsenic concentrations in intake water and ash pond discharges. Assuming a mean coal arsenic content of 6.0 ppm, we calculated arsenic concentrations in the ash transport water by the methods described above. Comparing these results to the measured concentrations in the pond overflows from each plant, we concluded that the ratio of pond overflow concentration to transport water concentration averages between 5.0 and 6.0 percent. For our analysis, we will use a value of 0.059. The pollutant concentration in overflow water is then given by

$$p_o = a p_s \quad (8.25)$$

where p_o = pollutant concentration in ash pond overflow (ppm),

a = ratio of concentrations in overflow and transport water
(dimensionless).

With this concentration and the overflow discharge rate we can calculate the arsenic content of the overflow from

$$P_o = 8.345 \times 10^{-6} P_o F_o \quad (8.26)$$

where P_o = pollutant content of overflow water (lb/day).

Using Eqs. (8.21) through (8.26) with our previous results, we calculate pollutant discharge rates for ash pond overflows and coal-pile runoff. These results, and the appropriate assumptions, are presented in Table 8.6. The value for arsenic concentration in the ash pond overflow was chosen to be a worst case situation. Normally, we would expect the concentration to be a factor of 20 smaller--about 0.06 ppm--corresponding with the nominal arsenic concentration in coal. This overflow concentration agrees well with the values available in the literature. Two reports (Bahor et al., 1981; EPA, 1980a) found ash pond overflow concentrations as shown in Table 8.7.

As shown in Tables 8.4 and 8.6, emissions from the coal pile are more than two orders of magnitude smaller than the worst case ash pond

Table 8.6
ARSENIC DISCHARGES IN ASH POND OVERFLOW

Variable	Calculated or Assumed Value
P_c	106×10^{-6} lb/lb
F_{ba}	2.361×10^6 gpd
F_{fa}	1.773×10^6 gpd
F_o	4.051×10^6 gpd
P_s	19.64 ppm
a	.059
P_o	1.155 ppm
P_o	39.05 lb/day

Table 8.7

ARSENIC CONCENTRATIONS IN COMBINED ASH POND OVERFLOWS
(ppm)

Minimum	Mean	Maximum
0.005	0.038	0.038
0.0035	0.067	0.416
<0.005	0.013	0.05
<0.005	0.022	0.035
<0.005	0.034	0.100
<0.005	0.028	0.130
<0.005	0.008	0.040
<0.005	0.030	0.070
<0.005	0.123	0.360
<0.005	0.036	0.163
<0.005	0.041	0.130
<0.005	0.033	0.100
<0.005	0.032	0.070

NOTE: Values on the first line are from Bahor et al. (1981); all others are from EPA (1980a).

discharges. For that reason, we have not included in the case study the costs and effects of controlling coal-pile discharges. We have assumed that coal-pile runoff could easily be routed to the combined ash ponds for treatment and disposal.

Groundwater Emissions

Priority pollutant emissions to soil and groundwater will come primarily through leaching from ash and sludge ponds and landfills. The amount and chemical characteristics of such leachate will depend on a number of influences, including (1) coal and ash composition, (2) plant processes, (3) treatment and disposal techniques, (4) soil characteristics, and (5) pond or landfill lining. For the purposes of our case study, we have assumed that disposal ponds or landfills would be lined to minimize the amount of leaching into local groundwater. As discussed earlier, our preliminary analysis of leaching from disposal ponds indicated that its contribution to surface water contamination

would be negligible, compared to the contribution of direct discharges from the plant. Consequently, we have ignored these emissions in the current case-study analysis.

DESCRIPTION OF ANALYSIS

Control and Treatment Alternatives

As described above, we have limited base case plant arsenic emissions to those contained in discharges from the combined ash disposal ponds and coal-pile runoff. Because the ash handling system emissions far exceed those from the coal pile, we consider only modifications to the ash transport system in our analysis. More specifically, we will compare the costs and chronic health risks associated with two alternative ash handling systems: (1) dry ash handling with landfill disposal and (2) wet ash handling with total recirculation of transport water. We assume that either of these alternatives would be able to eliminate arsenic discharges into surface water from the ash handling system, leaving only the coal-pile discharges.

Cost Estimates

In this section we estimate the levelized annual revenue requirements for the alternative ash transport and disposal systems considered in our analysis. Our discussion is divided into four primary subsections. In the first, we outline the basic assumptions used to estimate costs. In the second subsection, we discuss variations to these assumptions. We estimate the alternative system costs in the third subsection. Finally, in the fourth subsection, we estimate the range of costs associated with the variations in the basic set of assumptions.

In these calculations, our objective is to demonstrate general cost trends. We do not intend that these cost numbers represent precise or absolute dollar estimates. Further, we do not intend to capture the entire range of realistic case studies in the variations to the basic costs. Simplified cost estimates necessarily limit the parameters that can be varied.

Our cost calculations are based primarily on information contained in Bahor et al. (1981). Unfortunately, we cannot present consistent costs for all alternatives in our analysis. Bahor et al. (1981) base their cost calculations on unstated design assumptions and do not include data for a once-through wet transport system consistent with our base case. As a consequence, we must limit our cost information to the two alternative ash transport systems mentioned above. Although this limitation prohibits us from comparing the absolute costs and risks associated with arsenic control, we can still compare the costs and benefits of the two alternative control schemes and their variations.

Base Case Assumptions. In Table 8.8 we present the basic assumptions used to estimate the transport and disposal costs. Where appropriate, these assumptions are taken directly from Bahor et al. (1981). In other instances, we have modified the data to reflect the specifics of our case.

Variations to Basic Assumptions. We assess the sensitivity of our cost estimate to changes in selected parameters from our basic parameters. Those parameters that we select to vary are listed in Table 8.9. In all cases, the basic assumptions are indicated by an asterisk (*) in the table.

Cost Calculations for Alternative Cases. In this section we estimate the levelized annual revenue requirement for the two basic alternative systems: (1) wet ash handling with total recirculation of transport water (Table 8.10), and (2) dry ash handling with truck transport to landfill (Table 8.11). We use the method described in EPRI (1979) and Bahor et al. (1981), the assumptions summarized in Table 8.8, and data from EPA (1980a).

For the basic system using wet ash transported in a slurry a distance of one mile to a narrow valley, we estimate a \$16.80 per ton levelized annual revenue requirement (Table 8.10). For the alternative system using dry ash transported by truck one mile to a landfill with a 75-foot embankment, we estimate a \$16.17 per ton levelized annual revenue requirement (Table 8.11).

Table 8.8

BASIC ASSUMPTIONS

Plant and Disposal System
Newly constructed baseload power plant and disposal system
Disposal site lifetime of 30 years with a mid-1980 startup
Levelized capacity factor of 60 percent
Peak in-plant and out-of-plant conveyor capability to handle 100% capacity factor
Power unit heat input requirement: 9230 Btu/kWh
Coal ash is 182,000 t/yr for a 500 MWe plant
Coal ash is 80% fly and 20% bottom ash
Fly ash collection efficiency is 99%
Disposal cost estimates include in-plant handling, out-of-plant transportation, and disposal site costs
Economics
1980 capital costs and mid-1980 operating and maintenance costs
Thirty year fixed charge rate ranges from 16.0% to 26.6% annually and varies as discussed in EPRI (1979)
Thirty year levelizing factor for operation and maintenance costs ranges from 1.820 to 3.444 and varies as discussed in EPRI (1979)
Land cost is \$1500 per acre
Wet Ash Handling
Ash sluicing to pond with hydraulic in-plant ash handling system
Pumps and transport lines have 100% capacity
Disposal pond has 70% capacity factor over 30 year life
Spare pumps and pipeline for water delivery and recycle
Coal ash slurry pumped at 10% ash by weight
Pond location varies from 1 mile to 25 miles
Fly ash slurry settles to dry density of 60 lb/ft (969 kg/m ³)
Disposal site is lined
Four types of disposal site: wide and narrow valleys with dam, and ponds with 25-ft and 50-ft embankments on flat land
Dry Ash Handling
Pneumatic in-plant fly ash handling system
Bottom ash collected hydraulically and dewatered
Dry ash disposal with truck or conveyor belt transport
Hauling, placing, and compaction done by utility
Disposal site location varies from 1 mile to 25 miles
Four types of disposal site: wide and narrow valleys with toes, and sites with 75-ft or 150-ft embankments on flat land
Dry coal ash density or 85 lb/ft (1360 kg/m ³)
Belt can transport up to 200,000 t/yr of ash
Yearly operation and maintenance cost is 17% of total capital cost
Conveyor belt has 100% capacity factor and landfill has 70% capacity factor over 30 year plant life

Table 8.9

VARIATIONS IN BASIC ASSUMPTIONS

Parameter Being Varied	Cases
Distance from plant to disposal site	* 1 (mi) 5 10 25
Ash transport and disposal system	Wet (slurry transport) 25-ft embankment (flat) 50-ft embankment (flat) * Narrow valley with dam Wide valley with dam Dry (truck transport) Uncompacted ash 75-ft embankment (flat) 150-ft embankment (flat) Narrow valley with dam Wide valley with dam Compacted * 75-ft embankment (flat) 150-ft embankment (flat) Narrow valley with dam Wide valley with dam Dry (belt transport) Uncompacted 75-ft embankment (flat) 150-ft embankment (flat) Narrow valley with dam Wide valley with dam Compacted 75-ft embankment (flat) 150-ft embankment (flat) Narrow valley with dam Wide valley with dam
Ash transport water recirculation	* Complete recirculation Partial recirculation

NOTE: * refers to basic assumptions used in Table 8.10 (wet slurry) and in Table 8.11 (dry truck transport).

Table 8.10

LEVELIZED ANNUAL REVENUE REQUIREMENT: WET ASH/POND SYSTEM

Cost Item	Estimated Levelized Cost (Thousands of Dollars)
Total capital cost	
In-plant handling system	929
Dam appurtenances	41
Embankments	3,912
Site preparations	4,612
Drainage ditch	233
Equipment for disposal area	79
Transport system	240
	Subtotal 10,416
Total replacement cost	
Transport system	677
Equipment for disposal area	120
	Subtotal 797
Yearly operation and maintenance cost	
In-plant handling system	158
Dam appurtenances	4
Drainage ditch	23
Equipment for disposal area	38
Transport system	290
	Subtotal 513
Total levelized cost per year	3,017
Levelized cost per dry ton	\$16.80

NOTE: Case assumptions include (1) narrow valley with dam, (2) 182,000 t/yr ash, (3) 10 percent cost of capital, (4) 1 mile transport distance.

Table 8.11

LEVELIZED ANNUAL REVENUE REQUIREMENT: DRY/LANDFILL SYSTEM

Cost Item	Estimated Levelized Cost (Thousands of Dollars)
Total capital cost	
In-plant handling system	2,190
Sediment ponds	11
Site preparations	7,405
Drainage ditch	158
Equipment for disposal area	93
Transport system	64
Drainage pipe	32
On-site haul road	16
	Subtotal 9,969
Total replacement cost	
Transport system	75
Equipment for disposal area	189
	Subtotal 264
Yearly operation and maintenance cost	
In-plant handling system	372
Drainage ditch	12
Equipment for disposal area	55
Transport system	65
On-site haul road	3
	Subtotal 507
Total levelized cost per year	2,951
Levelized cost per dry ton	\$16.17

NOTE: Case assumptions include (1) landfill with 75-foot embankment, (2) 182,000 t/yr ash, (3) 10 percent cost of capital, (4) 1 mile transport distance.

Cost Estimates for Variations on Alternatives. In Table 8.12 we estimate the levelized cost per dry ton for a variety of alternative designs, including dry transport alternatives (pipeline, truck, and belt) and ash water recirculation alternatives (partial and full). Figure 8.3, taken from Bahor et al. (1981), shows how the costs presented in Table 8.12 would vary with transport distance. The costs in the table assume a distance of 1 mile.

We generated the numbers in Table 8.12 as follows. The basic alternative system costs (identified by *) were estimated in Tables 8.10 and 8.11 following the approach detailed in Bahor et al. (1981). The remaining entries in the "Full" column were extrapolated from data presented in Table 9-10 of Bahor et al. (1981), knowing that the data in Table 9-10 represent total ash quantities of 200,000; 400,000; 600,000; 870,000; and 1,700,000 metric tons and that the costs in Table 8.12 assume a single ash quantity of 182,000 metric tons.

The entries in the "Partial" column were determined using Tables VIII-18 to VIII-29 of EPA (1980a). We estimated the ratio for once-through over partial recirculation costs from these tables assuming a new 500 MWe power plant facility. This ratio was determined for the costs of capital equipment, operation and maintenance, land, and energy. Using these resulting ratios and the methodology applied to developing the levelized costs in Tables 8.9 and 8.10, we calculated each of the appropriate costs shown in the "Partial" column.

In examining the entries in Table 8.12, we find that our basic assumptions for wet and dry transport (indicated by * in Tables 8.8 and 8.11) are close to the least-expensive options. In general, conveyor belt transport is the most-expensive option. As expected, partial recirculation is less expensive than full recirculation, generally by about 10 percent. We do not consider the incremental risk presented by partial recirculation, however, because no data are available to determine pollutant emissions from these systems.

For dry truck transport the choice of a flat terrain with embankment or a valley with dam does not significantly affect the levelized annual cost. Further, neither the size of the embankment nor

Table 8.12

LEVELIZED ANNUAL COSTS OF ASH DISPOSAL ALTERNATIVES
(1980 \$/t)

Disposal Method	Ash Water Transport Recirculation	
	Full	Partial
Pipeline transport		
Wet ash		
25-ft embankment (flat){a}	28.84	25.52
50-ft embankment (flat)	35.48	31.37
Narrow valley with dam	* 16.80	14.87
Wide valley with dam	17.41	15.41
Truck transport		
Dry uncompacted ash		
75-ft embankment (flat)	17.90	15.84
150-ft embankment (flat)	17.00	15.05
Narrow valley with dam	17.27	15.28
Wide valley with dam	19.84	17.56
Dry compacted ash		
75-ft embankment (flat)	* 16.17	14.29
150-ft embankment (flat)	15.94	14.11
Narrow valley with dam	15.59	13.80
Wide valley with dam	17.29	15.30
Belt transport		
Dry uncompacted ash		
75-ft embankment (flat)	26.30	23.28
150-ft embankment (flat)	25.41	22.21
Narrow valley with dam	25.84	22.87
Wide valley with dam	28.25	25.00
Dry compacted ash		
75-ft embankment (flat)	24.60	21.77
150-ft embankment (flat)	24.78	21.93
Narrow valley with dam	23.90	20.35
Wide valley with dam	25.70	22.74

NOTE: * refers to basic alternatives used in Tables 8.10 and 8.11.
{a} Flat refers to flat terrain with a surrounding embankment.

SOURCE: Bahor et al. (1981).

Fig. 8.3 — Levelized annual revenue: correction factors for transport distance

the width of the valley has significant effects on the levelized annual cost.

Risk Assessment

The calculated control costs and emissions associated with the base case and alternative systems are summarized in Table 8.13. For each

Table 8.13
CASE-STUDY ALTERNATIVES

Case	Control Cost{a} (\$/yr)	Emissions (kg/s)
Base (wet ash handling without recirculation)	{b}	2.06×10^{-4}
Dry ash handling with landfill disposal	2,951,000	1.14×10^{-6}
Wet ash handling with total recirculation	3,017,000	1.14×10^{-6}

{a} Levelized capital, operating, and maintenance costs.

{b} Cost not determined.

case, we assumed that the annual precipitation rate was 35 inches, to maximize the coal-pile runoff. Note that the emissions associated with the two alternative systems include only this runoff, because both systems eliminate the ash transport water discharges. We calculated the net risk for each of these cases, to compare the costs and benefits of controlling arsenic emissions from the ash handling system. In the analysis, we limited our study to considering only exposure by direct ingestion of drinking water from surface sources. These limitations were necessary because (1) project scope and objectives emphasized surface water contamination, and (2) we did not have the resources necessary to make the required environmental transport calculations for the other media. Without these calculations, we could not evaluate how plant discharges would affect:

- Concentrations in the atmosphere, soil, and groundwater.
- Atmospheric deposition rates.
- Pollutant uptake by plants and animals.

Dermal contact pathways were not included because investigators have not found them to be significant uptake routes for arsenic in water.

Surface Water Transport

This analysis simulated the migration and fate of arsenic after its discharge from the power plant into the Columbia River.¹ The plant was located at River Kilometer 608, downstream of the Priest Rapids Dam. The total length of the study area was 169 km, divided into 16 computational segments between Priest Rapids (River Kilometer 638.8) and McNary (River Kilometer 469.8) Dams. The plant site and study area are shown in Fig. 8.1. The simulation of arsenic transport and fate was performed using the surface water contaminant transport model, SERATRA, which has been described in Chap. 4 and in Onishi et al. (1982).

Because our risk analysis investigates chronic health effects, we have been concerned with steady-state conditions. This permits us to use mean annual discharge rates, pollutant concentrations, and exposure rates. Although the transport model SERATRA simulates transient river conditions, it can be used to determine equilibrium states when all boundary conditions are constant. The time-dependent results were obtained for a period of 25 days, simulated at one hour intervals. These results provide not only equilibrium conditions, but also insight into the physical mechanisms creating the equilibrium conditions in each segment of the river.

The most significant attenuation mechanism in rivers is dilution; therefore, using the smallest realistic river flow rate will yield a conservative estimate of the arsenic concentrations. Discharges on this reach of the Columbia River are controlled by the operation of the Priest Rapids Dam, which is legally bound to maintain the discharge above 36,000 cfs. Although the normal flow of the Columbia greatly exceeds this value, we chose to use it for the base case calculations.

In this study, SERATRA simulated the transport of sand, silt, clay, dissolved arsenic, and arsenic adsorbed by sand, silt, and clay. The model was driven by boundary conditions at Priest Rapids Dam (segment 1), the power plant site (segment 3), and a point 117 km downstream of Priest Rapids Dam where the Snake River enters the Columbia (segment 11). Background levels of sediment and arsenic enter the study area via

¹This discussion is summarized from Onishi et al. (1982).

the Columbia and Snake Rivers, while the arsenic loading occurs at the power plant site. Initial assumptions for the simulation of sand, silt, and clay are shown in Table 8.14. Table 8.15 shows assumptions relating to river flows and arsenic levels. Sediment concentrations for both rivers were taken at the low end of the data range, to enhance the levels of dissolved arsenic in the water.

Arsenic distribution coefficients (K_d) govern contaminant exchange between dissolved and particulate phases and must be specified for each sediment fraction. We obtained a bulk sediment K_d of $13 \text{ m}^3/\text{kg}$ from a

Table 8.14

INITIAL SIMULATION ASSUMPTIONS FOR SAND, SILT, AND CLAY

Material	Particle			Upstream Conc.		Snake River Conc.	
	Size (mm)	Density (kg/m^3)	K_d (m^3/kg)	Particle (mg/l)	Arsenic (kg/kg)	Particle (mg/l)	Arsenic (kg/kg)
Sand	0.100	2650	2.5	2.0	3.54×10^{-6}	0.20	3.78×10^{-6}
Silt	0.030	2650	12.5	6.0	1.77×10^{-5}	2.85	1.89×10^{-5}
Clay	0.002	2650	25.0	2.0	3.54×10^{-5}	0.95	3.78×10^{-5}

Table 8.15

INITIAL SIMULATION ASSUMPTIONS FOR RIVER FLOWS AND ARSENIC LEVELS

River	Flow (m^3/s)	Dissolved Arsenic (kg/m^3)	Arsenic Discharge	
			Coal Pile (kg/s)	Ash Pond (kg/s)
Columbia	1020	1.42×10^{-6}	6.12×10^{-7}	2.05×10^{-4}
SNAKE	269	1.51×10^{-6}	0.0	0.0

Lake Michigan study (Woolson, 1977) and apportioned it between the three sediment fractions. Background arsenic levels were calculated assuming a bulk concentration of 1.6×10^{-6} kg/m³. The arsenic distribution transfer rate (K_j) controls the rate of adsorption and desorption in the model. The value 1.0 means that phase equilibrium will be reached after one day.

Table 8.16 presents background levels and the results of the base case calculations of arsenic concentrations in the Columbia River. For the base case, the table shows how much arsenic is dissolved in the water and how much is adsorbed to the three sediment fractions. For the background case, only the dissolved and total concentrations are given. A complete discussion of the assumptions, calculation techniques, results, and their implications can be found in Onishi et al. (1982).

Table 8.16
BACKGROUND AND COMPUTED BASE CASE ARSENIC CONCENTRATIONS
(10^{-6} mg/l)

River Segment	Base Case				Background	
	Sand	Silt	Clay	Dissolved	Dissolved	Total
1	7.1	110	71	1420	1420	1600
2	7.1	110	71	1420	1420	1600
3	7.2	110	72	1620	1420	1600
4	7.3	110	73	1610	1420	1600
5	7.5	110	74	1610	1420	1600
6	7.4	110	75	1610	1420	1600
7	3.6	110	76	1610	1420	1600
8	1.8	120	78	1600	1420	1600
9	0.91	120	79	1600	1420	1600
10	0.46	120	80	1600	1420	1600
11	0.26	100	70	1580	1420	1600
12	0.13	100	70	1580	1420	1600
13	0.06	100	70	1580	1420	1600
14	0.03	100	70	1570	1420	1600
15	0.02	100	70	1550	1420	1600
16	0.01	100	69	1530	1420	1600

Exposure Calculations

General Information. As discussed earlier, we confined the exposure calculations to ingestion of surface water from the Columbia River. This restricts the exposed population to that living downstream of the power plant in communities that consume Columbia River water. To increase this population somewhat, we also included communities that consumed groundwater from wells resupplied by surface water injection. Withdrawals from the Columbia River for both domestic and municipal uses have been documented in WPPSS (1972, 1973a, and 1973b). The exposed populations and their locations are shown in Table 8.17. Population figures were obtained from the preliminary 1980 Census information.

The exposed population was divided into three subregions, corresponding to the river segment from which they derived their water. Each subregion population was further divided into four groups--infants, children, teenagers, and adults--as defined in Chap. 5. In the absence of specific Census information describing age distribution in 1980, we used the 1970 statewide age distribution for Washington to partition each subregion population. The final population figures are shown in Table 8.18.

Table 8.17
POPULATION LOCATION FOR CASE STUDY

City	Population	River Distance{a}	River Segment
Richland	36,516	42	8
Kennewick	34,397	52	10
Pasco	26,296	52	10
Burbank	3,146	55	11

{a} Distance measured in km downstream from plant site.

Table 8.18

POPULATION GROUPS FOR CASE STUDY

Subregion	River Segment	Population Group			
		Infant	Child	Teenager	Adult
1	8	621	6,646	4,418	24,830
2	10	1,031	11,046	7,343	41,271
3	11	54	572	381	2,139

In the current calculations, we have assumed that plant operation levels and emissions and regional population would be constant over a plant life of 35 years. If emissions were not constant, we could calculate the environmental concentrations associated with varying emission levels and average these over the life of the plant. Similarly, we could use population projections to estimate an average population over the plant lifetime. Although this would change the results somewhat, with the current procedure the change would be proportional to the population increase. Because these calculations are only illustrative, not definitive, there would be little gained from including these additional complications. In an actual analysis, however, these variations should be considered.

We have also assumed that human lifespan averages 72.0 years, and that the background arsenic concentration in food consumed in the region is 1.0×10^{-8} mg/kg. Other information required for the analysis is shown in Table 8.19.

Drinking Water Concentrations. Arsenic concentrations in the Columbia River will be reduced by drinking water treatment processes used in the various communities. Investigation into the water treatment processes used at Pasco, Richland, and Kennewick near the hypothetical plant site indicates that these communities use conventional coagulation and filtration processes for their Columbia River water supplies. Their treatment includes the following steps:

Table 8.19

MISCELLANEOUS ASSUMPTIONS FOR OUR CASE STUDY

Assumption	Population Group			
	Infant	Child	Teenager	Adult
Weight (kg)	7.2	21.5	50.6	71.5
Food consumption (kg/day)	0.55	1.19	1.50	1.28
Water consumption (l/day)	0.55	0.71	0.71	1.01

- Prechlorination (frequently).
- Coagulation with alum.
- Sedimentation.
- Dual media filtration (anthracite/sand).
- Postchlorination.
- Reservoir storage.

The effectiveness of all processes has not been thoroughly studied, but Sorg and Logsdon (1978) have summarized the current understanding. They have found that removal efficiency for the above standard coagulation, sedimentation, filtration, and chlorination processes depends on four variables: (1) the arsenic oxidation state (III or V), (2) initial arsenic concentrations, (3) the type and dose of coagulant used (iron or alum), and (4) the pH of the treated water (high pH reduces removal). Although the affected cities do not all use the same standard process, we have assumed so to simplify the analysis.

Using information from Sorg and Logsdon (1978), therefore, we can estimate how effectively this process will remove arsenic from the Columbia River water. For simplicity, we assume that all forms of arsenic in the river (dissolved and adsorbed) reach the treatment plant and are similarly affected by treatment. Moreover, we can also assume that most of the arsenic will be in the arsenate (V) form. The arsenite (III) form oxidizes to arsenate in well-mixed and oxygenated surface waters. From measurements, we know that the Columbia has an average pH

of 8.5 and is almost always saturated with dissolved oxygen. Also, if drinking water is chlorinated before coagulation, this would further convert arsenite to arsenate. Assuming that the pH of treated drinking water is between 7.0 and 7.5 (desirable for efficient coagulation with alum), from Fig. 2 of Sorg and Logsdon (1978) we can estimate that the arsenic removal efficiency should be about 75 percent for the standard treatment process. Note, however, that relatively small changes in some of the assumptions could reduce this removal efficiency significantly. For this reason, we must use sensitivity analysis to determine how these variations would affect our results.

Health Effects

Arsenic can be found everywhere in the environment, in fresh and salt water, soils, rocks, and biological organisms. As a consequence of this natural abundance, trace levels of arsenic can be found in many types of foods (Mahaffey et al., 1975; Jelinek and Corneliussen, 1977; Burrus and Sargent, 1976; EPA, 1980b), particularly fish and shellfish. Although arsenic levels in water vary greatly, only in rare instances do concentrations in groundwater exceed 0.050 ppm, the drinking water quality standard in the United States (EPA, 1980b; Waldron, 1980). Average background concentrations in fresh waters have been estimated as (1) 0.002 mg/l (world mean) (Klein et al., 1975a) and (2) 0.0064 mg/l (U.S. mean) (Heit, 1977). The Food and Drug Administration has studied arsenic intake in humans over a period of years. From these studies, Jelinek and Corneliussen (1977) have estimated that between 1971 and 1974 average arsenic intake was between 10 and 20 $\mu\text{g/day}$.

Arsenic taken in by humans is excreted primarily in urine, but it also tends to bind to keratin in skin, hair, and nails. Human metabolism of arsenic is not well understood, but it has become clear that arsenic metabolism and pharmacokinetics depend heavily on the chemical compound or form of arsenic in the body (EPA, 1980b). Animal experiments on its carcinogenicity have generally given negative results. As a consequence, researchers have been unable to develop an adequate animal model to study the carcinogenic properties of arsenic (Waldron, 1980). For an extensive discussion of the toxicology,

pharmacokinetics, and health effects of arsenic, the reader should refer to EPA (1980b).

Unfortunately, measurements of power plant arsenic emissions to surface waters do not distinguish between dissolved and particulate forms, much less between chemical species. Similarly, the behavior of the different chemical species in the environment is not well understood. As a consequence, although researchers may have evidence that some forms of arsenic are more or less toxic than others, we do not have the data necessary to distinguish between these forms and follow them throughout the environmental transport and exposure analysis. Were these data available, the proper procedure would be to specify and follow each potentially chronically toxic species through the methodology.

All evidence for carcinogenic effects has been epidemiological. Several studies have thoroughly reviewed the case for relating arsenic exposure to lung and skin cancer (IARC, 1973; NIOSH, 1975; for example). Frequently cited studies include Lee and Fraumeni (1969), Ott et al. (1974), and Pinto et al. (1977), which present evidence indicating that atmospheric arsenic (primarily arsenic trioxide) can increase the incidence of lung cancer in exposed workers. These studies are discussed and used by the EPA Carcinogen Assessment Group in making its Final Risk Assessment on Arsenic (CAG, 1980). The EPA (1980b) has also described numerous studies investigating the relationship between the ingestion of arsenic-contaminated water and an increased incidence of skin cancer and vascular problems in the exposed population. One study (Morton et al., 1976) examined the incidence of skin cancer in a population exposed to water-borne arsenic in Lane County, Oregon. Basal and squamous cell carcinoma incidence rates were determined and compared to arsenic levels in the water, but neither type of carcinoma correlated with these levels. The actual arsenic concentrations in the water sources were lower than expected at the outset of this study, so the lack of correlation does not necessarily contradict positive correlation at higher concentrations. Other studies by Tseng et al. (1968) and Tseng (1977), based on exposure of a Taiwanese population to arsenic

concentrations roughly tenfold those in southern Lane County, showed a positive correlation between skin cancer and arsenic in the water supply. Only these studies provided sufficient information from which to generate dose-response relationships.

The Tseng studies suffer from the problems common to most epidemiological investigations. First, being retrospective, they did not accurately estimate actual doses of arsenic ingested by the population. Second, Tseng et al. (1968) did not determine the chemical form of the arsenic ingested, or whether other pollutants were also present in the contaminated water supplies. However, a later follow-up study by Irgolic (1979) suggests that the arsenic was in both the arsenite and arsenate forms, with 80 to 90 percent in the arsenate form. At some sites, a portion of the arsenic may have been present as a methylated arsine. Also, another follow-up study by Lu et al. (1977) has found nonarsenical fluorescent compounds in some of the water samples. Some of these compounds may be carcinogenic. Finally, the studies did not investigate the nutritional experience and presence of other health effects in the population--conditions that might have made them significantly more susceptible to the observed diseases. In spite of these weaknesses, we use the data in these studies to develop approximate dose-response relationships for skin cancer from arsenic ingestion.

In the case study, we have used five alternative dose-response relationships coupled with the Tseng et al. (1968) and Tseng (1977) data. The five models considered are the multihit, one-hit, Crump version of the multistage, probit, and Weibull models. The Tseng articles gave skin cancer response to chronic ingestion of arsenic-containing water for three age ranges and three dose ranges. The prevalence rates and dose midpoints are shown in Table 8.20. These doses were scaled to standard dose form using Eq. (6.36) of Chap. 6, or

$$d = c_f h_1 r_1 / k_1$$

where c_f = concentration of pollutant (ppm),
 h_1 = feed rate constant ($\text{kg}^{1/3}/\text{day}$),

Table 8.20

SKIN CANCER PREVALENCE RATES FOR ARSENIC INGESTION

Dose (c _f ppm)	Age (yr)		
	30	50	70
0.15	0.0015	0.0065	0.0481
0.45	0.0043	0.0477	0.1634
1.20	0.0224	0.0983	0.2553

r_1 = fraction of ingested food absorbed (defaulted to 1)
(dimensionless),

k_1 = coefficient of body-weight-scaled clearance
(defaulted to 1) ($1/\text{day}/\text{kg}^{2/3}$).

The models have been parameterized using the complete age-response data set. In addition, parameter values were obtained using only the three data points that correspond to lifetime dosing. This latter approach, while clearly not providing the best estimate of expected response because only a portion of the available data has been used, is included just for information, because most toxic substance animal-response data are restricted to lifetime exposures.

The dose-response parameters for the one-hit and multihit models were determined from Eqs. (6.37), (6.38), and (6.27) of Chapter 6 using the maximum likelihood approach of MULTI80 (Rai and Van Ryzin, 1980). The probit parameters were determined from Eqs. (6.40) and (6.27) of Chap. 6 SAS/Probit (SAS, 1979). The Crump parameters were estimated, where possible, from Eq. (6.39) of Chap. 6 using GLOBAL79 (Crump and Watson, 1979) or, for fixed k , BMDP (BMDP, 1981). The Weibull model parameters were estimated from Eqs. (6.41) and (6.27) of Chap. 6. Because spontaneous background is negligible for arsenic, the Weibull parameters were determined by using multiple linear regression after taking double logarithms of $1 - P_r$ in Eq. (6.41).

The exposure time, t_1 , that appears in these equations was expressed in dimensionless units of fractional lifetime (human lifetime taken as 70 years).

For fits to the full age-response data, we employed all the prevalences in Table 8.20 and an estimated zero spontaneous rate of arsenical type skin cancer. The parameter values obtained are: (1) $\alpha = 1.17$ (0.10) for the one-hit model; (2) $\alpha = 4.53$ (0.81) and $k = 1.59$ (0.11) for the multihit model; (3) $\alpha = 0.569$ (0.159) and $\beta = 1.607$ (0.126) for the probit model (of first power in exposure time); and (4) $\ln \alpha = 1.528$ (1.297), $k = 1.19$ (0.14), $n = 3.881$ (0.33) for the Weibull model. We could not obtain parameter estimates for the Crump model because the GLOBAL79 program does not handle nonlifetime exposure data without modification. The standard deviations of the parameters obtained are shown in parentheses. Because spontaneous background (P_b) is zero, added risk (for the epidemiological conditions of Tseng) is given directly by Eqs. (6.37), (6.38), (6.40), and (6.41) of Chap. 6.

Figure 8.4 shows mean added risk versus arsenic concentration in water, with the lifetime (70 year) epidemiologic data points for four models. The inset figure shows the fit of the Weibull and multihit models to the full set of age-response data. The Weibull model provides the best fit, because of its greater flexibility in fitting exposure time using the parameter n . The multihit model provides a good fit to the mid-range exposure time data (50 year data) but underestimates the effects of shorter exposure times and overestimates the effects for longer times. These effects are also apparent in χ^2 statistics² computed to measure the goodness of fit of the model prevalences to the Tseng prevalence data. The χ^2 values are 189 for the probit, 208 for the multihit, and 223 for the one-hit, whereas the Weibull value is about 20. The much lower Weibull value shows that this model is the best of those examined.

For comparison, Fig. 8.5 shows the added risk versus concentration relations when the models were fit only to lifetime exposure data (the

² χ^2 = sum over i dose-time pairs of $n_i(P_{\text{obs},i} - P_{\text{exp},i})^2 / P_{\text{exp},i} / (1 - P_{\text{exp},i})$, where n_i equals the total number of subjects in dose-time cohort i , $P_{\text{obs},i}$ equals the observed prevalence for this cohort, and $P_{\text{exp},i}$ equals the prevalence expected from a model for this cohort.

last column of Table 8.19). The Crump model was also fit to these data, but the program produced only a degenerate one-hit model, because the condition $q_i \geq 0$ (cf. Eq. (23) of Chap. 6) was not met for greater numbers of hits (i.e., $k > 1$). Note that the model results shown in Fig. 8.5 are substantially different from those shown in Fig. 8.4. Although the models fit the three data points in Fig. 8.5 relatively well, not taking all age-response information into account leads to large changes in the quantitative behavior of the models. Even the Weibull model, which best fit the full data set above, is parameterized in Fig. 8.5 to predict an arsenic concentration associated with an added risk of 10^{-6} that is nearly 50 times lower than that in Fig. 8.4.

Our case study used the models parameterized from the entire set of age-response data. However, the results for the more limited lifetime exposure fit should be remembered as an example of one of the severe restrictions in interpreting the quality of fit to limited dose-response data.

RESULTS OF PRIMARY CASES

Using the nominal values of all parameters and inputs, with the four parameterizable dose-response models, we calculated the net risk for each of the plant alternatives.³ In this situation, net risk means the total lifetime cases of skin cancer in the entire regional population caused by exposure to environmental arsenic levels under the specified power plant alternative, minus the number of cases associated with background arsenic levels in the environment. The background arsenic levels include consumption of arsenic in food products, as well as ingestion of domestic water supplies. The results are presented in Table 8.21.

³The reader should remember that risk analysis is not a precise science that arrives at exact risk estimates. Rather, risk analysis is an exercise that (1) roughly approximates the relative risks of alternative designs, (2) investigates the relative sensitivities of the various input parameters, and (3) identifies weaknesses in assumptions. As such, these risk estimates should not be interpreted as the absolute risks associated with each alternative.

Fig. 8.4 — Mean added risk versus dose for alternative dose-response models
(complete exposure data)

Fig. 8.5 — Mean added risk versus dose for alternative dose-response models
(lifetime exposure data)

Table 8.21

NET RISK FOR PRIMARY CASES
(Lifetime cases)

Case Alternative	Dose-Response Model			
	One-Hit	Multihit	Probit	Weibull
Base Case				
--plant lifetime	0.123	0.0177	0.00164	0.0202
--human lifetime	0.252	0.0364	0.00337	0.3310
Dry handling	--	--	--	--
Recirculation	--	--	--	--

We used two different values for the exposure time in the dose-response models in an attempt to bracket the actual expected number of skin cancer cases. First, we set the exposure time equal to the expected operating time of the power plant (nominally 35 years), and assumed that no cases caused by power plant arsenic would be found in the population after this time. This approach clearly underestimates the expected response. Exposed individuals will still carry a propensity toward disease beyond the end of plant operation and will still develop cancer, although at a lower rate than if they had continued to be exposed to arsenic. Therefore, in the second case, we set the exposure time equal to a full human lifetime (72 years), providing an overestimate of the actual expected number of skin cancer cases. This bracketing was necessary because no epidemiological data are available describing the occurrence of arsenical skin cancer during the period following a cutoff to arsenic exposure. The results for the alternative cases for both exposure periods are shown in Table 8.21.

As one would expect from the structure of these dose-response models, the one-hit, multihit, and probit risks for human lifetime exposure are scaled directly from the plant lifetime exposure risks. The Weibull model, however, shows a much larger variation between the two exposure periods, because this model has a nonlinear time dependence as discussed earlier.

The table shows no net risk for either dry handling or complete ash handling water recirculation. As described earlier, we assumed for both of these cases that the plant discharges no ash transport water. Under these circumstances, the only arsenic emissions to the Columbia River are those from the coal-pile runoff, which are negligibly small compared to the background arsenic concentrations in the river.

Comparing these results with the cost information in Table 8.8, we see that the dry ash handling system is slightly preferred but only for a new power plant without an existing ash handling system. Although we did not determine the incremental costs of adding recirculation to an existing wet handling system, information given by EPA (1981) indicates that this option should be less expensive than converting to dry handling.

9. SENSITIVITY ANALYSIS AND CONCLUSIONS

SENSITIVITY ANALYSIS

Uncertainty analyses comprehend an extensive range of theoretical and numerical approaches. The most rigid of these approaches involves an analytic tool which, for the most part, assigns a distribution around each parameter within a calculation. These distributions represent the uncertainty in the parameters and must be convolved to arrive at a net distribution expressed in analytic terms.

This rigid approach becomes increasingly unmanageable as the number of discrete calculations increases. Less rigid approaches use some aspect of this analytic uncertainty distribution, such as the mean and the variance, and approximate the net uncertainty numerically. The least rigid approach involves three point estimates of each parameter (a best estimate, a low, and a high value) rather than a distribution. The sensitivity analysis approach consists of examining the net effect of changing the value of one input parameter at a time (between the low, best, and high estimates). For the more sensitive parameters (i.e., those that have more significant influence on the net risk), point changes in two or three parameters simultaneously can be assessed in terms of net effect. In this way, sensitivity analysis can be considered as a somewhat informal class of uncertainty analysis.

Objectives

The specific objectives of our sensitivity analysis are to:

- Estimate, with reasonable certainty, the range in anticipated risk.
- Determine how much each input parameter influences the estimated net risk, and to order the parameters in terms of this contribution.
- Assess the synergistic impact of varying two parameters simultaneously.

- Understand the effect of model selection on the value of risk estimated.

Procedure

To carry out this analysis, we first ran our base case with *nominal* values for all parameters using each of the four dose-response models and two exposure periods discussed earlier. In this nominal case, the resulting risks are highly sensitive to the choice of dose-response model, varying by a factor of nearly 100 between the one-hit and the probit models.

We then considered the remaining models in the analysis and determined which parameters met two conditions: (1) they might significantly affect the results, and (2) their values are not well established and may have large variation or uncertainty. For each of these parameters, we established both optimistic and pessimistic values, in addition to their nominal values that were used in the basic analysis. "Optimistic" values should lead to reduced estimates of risk, and "pessimistic" values should cause higher levels of risk. These nominal, optimistic, and pessimistic values for all selected parameters are shown in Table 9.1. The specific parameters are identified and defined in App. G.

Because we used a worst case definition of pollutant emissions by the power plant, we tested the sensitivity of our results to variations in parameter values beginning with the surface water transport analysis. Although SERATRA has many data requirements, in our simplified analysis only two parameters are physically related to contaminant behavior: (1) the distribution coefficient, K_d ; and (2) the distribution transfer rate, K_j .¹ The distribution coefficient is the equilibrium ratio of the particulate contaminant to the dissolved contaminant, or

$$K_d = \frac{\text{sediment contaminant (kg/kg)}}{\text{dissolved contaminant (kg/m}^3\text{)}}$$

¹This discussion of SERATRA is taken from Vol. 1 of Onishi et al. (1982).

Table 9.1
VALUES FOR PARAMETERS IN SENSITIVITY ANALYSIS

Parameter{a}	Optimistic	Nominal	Pessimistic
K_d (kg/m ³)	--	{b}	0.1 nominal
BMF (mg/kg)	10^{-4}	10^{-2}	10^{-1}
PL (yr)	25	35	45
TF	0.05	0.25	1.00
HS	0.0577	0.1154	0.2308
UW (l/d)	0.5 nominal	{c}	2.0 nominal
TU (kg/d)	2.0 nominal	{c}	0.5 nominal
AO-- α	0.97	1.17	1.37
AM-- α	2.91	4.53	6.15
k	1.81	1.59	1.37
AP-- α	0.2510	0.5688	0.8866
β	1.859	1.607	1.355
AW-- α	0.496	4.616	8.736
k	0.911	1.191	1.471
n	3.221	3.881	4.541

{a} K_d --contaminant distribution constant; BMF--background pollutant concentration in food from outside plant region; PL--plant operating lifetime; TF--fraction of pollutant retained after drinking water treatment; HS--ingestion exposure dose scale factor; UW--consumption rate for drinking water; TU--total food consumption rate; AO--parameter for one-hit dose-response model; AM--parameters for multihit dose-response model; AP--parameters for probit dose-response model; and AW--parameters for the Weibull dose-response model.

{b} Values shown in Table 8.13.

{c} Values determined from tables in Chap. 5.

In our study, the K_d represents the final outcome of all physical, chemical, and biological processes that affect arsenic in water. The distribution transfer rate, K_j , determines the speed of attaining the K_d . Thus, although ultimate equilibrium concentrations are independent

of the K_j , the character of the longitudinal distribution of arsenic before attaining the K_d equilibrium state depends on K_j . Of the two variables, K_d is more fundamental, so we performed a simulation to test model sensitivity to a change in its value. In this simulation, all values of K_d were reduced by a factor of 10 from the nominal situation. The results of this simulation are shown in Table 9.2 and are described in more detail in Vol. 1 of Onishi et al. (1982).

Using the dissolved arsenic concentrations shown in Tables 8.15 and 9.2 with the variations in Table 9.1, we next calculated net risk (assuming base case emissions) for a number of sensitivity cases. These cases included a *best* case (all parameters with optimistic (O) values) and a *worst* case (all parameters with pessimistic (P) values). The results of these two cases and the nominal case are given for the two exposure periods in Tables 9.3 and 9.4 and are shown in Fig. 9.1. The purpose of these two calculations is to demonstrate the extremes of the range in risk expected to be encountered using the various models with a reasonably high degree of confidence, perhaps 99 percent or better.

Table 9.2
ARSENIC CONCENTRATIONS FOR K_d SENSITIVITY ANALYSIS
(10^{-6} mg/l)

Segment	Fraction				Background	
	Sand	Silt	Clay	Dissolved	Dissolved	Total
1	6.5	98	65	1440	1580	1600
2	4.9	73	49	1480	1580	1600
3	3.8	57	38	1700	1580	1600
4	3.4	51	34	1720	1580	1600
5	2.8	42	28	1730	1580	1600
6	2.3	35	23	1750	1580	1600
7	0.92	26	17	1760	1580	1600
8	0.36	19	13	1770	1580	1600
9	0.14	15	10	1780	1580	1600
10	0.06	13	9.2	1780	1580	1600
11	0.05	14	9.2	1740	1580	1600
12	0.02	12	8.0	1740	1580	1600
13	0.01	11	7.8	1740	1580	1600
14	0.00	11	7.7	1730	1580	1600
15	0.00	11	7.7	1710	1580	1600
16	0.00	11	7.7	1680	1580	1600

Table 9.3

NET RISK RESULTS FOR SENSITIVITY ANALYSIS ON
PLANT LIFETIME EXPOSURE

Dose-Response Model					
Variable	Value	One-Hit	Multihit	Probit	Weibull
All	Nominal	0.123	0.0177	0.00164	0.0200
	O	0.00266	0.00000112	0.00000	0.000023
	P	2.829	4.170	8.654	8.672
K _d	P	0.133	0.0193	0.00179	0.0219
BMF	O	0.123	0.00246	0.000000137	0.0107
	P	0.121	0.0659	0.0609	0.0310
PL	O	0.0875	0.0126	0.00117	0.00546
	P	0.158	0.0227	0.00210	0.0534
TF	O	0.0245	0.00349	0.000313	0.00402
	P	0.490	0.0738	0.00763	0.0818
HS	O	0.0613	0.00588	0.000168	0.00883
	P	0.245	0.0531	0.0128	0.0460
UW	O	0.0776	0.0127	0.00163	0.0133
	P	0.173	0.0207	0.00114	0.0267
TU	O	0.0776	0.0127	0.00163	0.0133
	P	0.173	0.0207	0.00114	0.0267
D-R	O	0.102	0.00214	0.00000734	0.00121
	P	0.143	0.0958	0.112	0.0639
All	Nominal	0.123	0.0177	0.00164	0.0200
K _d - TF	P	0.533	0.0808	0.000779	0.0891
BMF - TU	O	0.0777	0.00137	0.0000000423	0.00645
	P	0.172	0.0768	0.0588	0.0411
UW - HS	O	0.0388	0.00423	0.000179	0.00580
	P	0.345	0.0621	0.00990	0.0607
D-R - PL	O	0.0726	0.00152	0.00000524	0.000409
	P	0.184	0.123	0.144	0.200
TF - UW	O	0.0155	0.00253	0.000319	0.00264
	P	0.691	0.0896	0.00622	0.109
HS - D-R	O	0.0508	0.000609	0.000000309	0.000643
	P	0.287	0.246	0.411	0.177
BMF - PL	O	0.0876	0.00175	0.0000000960	0.00288
	P	0.156	0.0847	0.0783	0.0819

Table 9.4

NET RISK RESULTS FOR SENSITIVITY ANALYSIS ON
HUMAN LIFETIME EXPOSURE

Dose-Response Model					
Variable	Value	One-Hit	Multihit	Probit	Weibull
All	Nominal	0.252	0.0364	0.00337	0.329
	O	0.00765	0.00000323	0.00000	0.000694
	P	4.526	6.675	13.851	67.344
K _d	P	0.274	0.0397	0.00369	0.360
BMF	O	0.252	0.00512	0.000000305	0.176
	P	0.250	0.136	0.126	0.503
PL	O	0.252	0.0364	0.00337	0.331
	P	0.252	0.0364	0.00337	0.331
TF	O	0.0504	0.00719	0.000645	0.0659
	P	1.009	0.152	0.0158	1.343
HS	O	0.126	0.0121	0.000346	0.145
	P	0.504	0.109	0.0264	0.754
UW	O	0.159	0.0262	0.00336	0.218
	P	0.355	0.0426	0.00236	0.439
TU	O	0.160	0.262	0.00336	0.218
	P	0.355	0.0425	0.00235	0.439
D-R	O	0.209	0.00440	0.0000151	0.0123
	P	0.295	0.197	0.230	1.684
All	Nominal	0.252	0.0364	0.00337	0.329
K _d - TF	P	1.097	0.167	0.00161	1.464
BMF - TU	O	0.160	0.00285	0.0000000925	0.107
	P	0.354	0.158	0.121	0.667
UW - HS	O	0.0798	0.00872	0.000369	0.0953
	P	0.711	0.128	0.0204	0.997
D-R - PL	O	0.209	0.00440	0.0000151	0.0123
	P	0.295	0.197	0.230	1.684
TF - UW	O	0.0320	0.00521	0.000658	0.0434
	P	1.422	0.185	0.0129	1.795
HS - D-R	O	0.105	0.00125	0.000000639	0.00657
	P	0.590	0.507	0.845	4.638
BMF - PL	O	0.252	0.00512	0.000000305	0.175
	P	0.250	0.136	0.126	0.502

Fig. 9.1 – Net risk under nominal, optimistic, and pessimistic assumptions

Over this wide confidence band, the resulting risks vary by as much as a factor of 10^6 for the multihit model and by as little as 1000 for the one-hit model. Using the probit model, the optimistic case estimates essentially zero net risk. The results of the *best* and *worst* cases demonstrate such a wide range that the numbers in themselves may not provide very much useful information.

To investigate the effect of any single parameter, we ran a series of calculations where each parameter was varied individually between optimistic and pessimistic values. By comparing each of these optimistic/pessimistic doublets with the nominal case, the best case, and the worst case, we could readily identify the most sensitive parameters. The results of these calculations are presented in Tables 9.3 and 9.4 and Figs. 9.2A through 9.2D. For the one-hit model (Fig. 9.2A), the most sensitive parameters appear to be TF and HS, and the least sensitive parameters K_d and BMF. For the other dose-response models, the most sensitive parameters are (1) multihit--BMF, TF, HS, and D-R; (2) probit--BMF and D-R; and (3) Weibull--D-R and TF. The least sensitive parameters are (1) multihit-- K_d , UW, TU, and PL; (2) probit-- K_d , UW, TU, and PL; and (3) Weibull-- K_d , UW, and TU. These results for the multihit, probit, and Weibull models are illustrated in Figs. 9.2B, 9.2C, and 9.2D. Across all four models, generally the most sensitive parameter is TF and the least sensitive, K_d .

The sensitivity of the net risk estimates clearly depends on the model selected for dose-response calculations. Results from the probit model are much more sensitive to parameter variations than results from the other models. Moreover, this model also gives slightly reduced risks for the pessimistic values of two parameters, UW and TU. The model appears to be extremely sensitive to the background arsenic consumption level (as demonstrated by variations in BMF). This may be an undesirable characteristic in most analyses, as we would prefer that net risks be relatively independent of the absolute background risks.

In summary, comparing Figs. 9.2A, 9.2B, 9.2C, and 9.2D with each other and with Fig. 9.1, we observe that the choice of dose-response model has more effect on the risk estimates and the sensitivity of these risk estimates than the choice of any of the parameters.

Fig. 9.2A – Sensitivity of risk to single parameter variation
(one-hit model)

Fig. 9.2B – Sensitivity of risk to single parameter variation (multihit model).

Fig. 9.2C — Sensitivity of risk to single parameter variation (probit model)

Fig. 9.2D – Sensitivity of risk to single parameter variation
(Weibull model)

Finally, to aid in determining the linearity of several of these parameters, we ran several cases where two parameters were varied simultaneously--both either optimistically or pessimistically. The results of these runs are shown in Tables 9.3 and 9.4 and Figs. 9.2A through 9.2D. None of these results are surprising. They suggest that the effects of simultaneous variations in parameter values may be, at most, mildly synergistic. Comparing results for corresponding models in Figs. 9.2A to 9.2D with Figs. 9.3A to 9.3D, we observe most often that the effects of two parameters are roughly the sum of the individual effects--that important individual parameters, such as TF, remain important when combined with less significant variables.

Limitations and Utility of Sensitivity Analysis

As stated earlier, we performed our sensitivity analysis primarily to determine reasonable upper and lower bounds on our calculation of net risk and to learn how much each input parameter affected these results. This procedure, however, has certain limitations that should be discussed. First, in selecting low and high values around the best estimate of each parameter, we do not necessarily consider the likelihood that these values will ever be achieved. That is to say, no uncertainty distribution is stated or implied. Second, for calculations involving, for example, n discrete steps, there will be nearly $(n - 1)!$ possible combinations of parameter values to be calculated. In our case, there are perhaps $n = 12$ important parameters, so that a complete sensitivity analysis is not practicable. Finally, a sensitivity analysis, unlike the more sophisticated analytic approaches, cannot easily assign magnitudes to synergistic effects between parameters.

Although it has some limitations, a thorough sensitivity analysis can be extremely useful. It can identify those input parameters that are sufficiently important to warrant more careful consideration in the overall analysis. This consideration may involve

Fig. 9.3A — Sensitivity of risk to double parameter variation
(one-hit model)

Fig. 9.3B — Sensitivity of risk to double parameter variation
(multihit model)

Fig. 9.3C – Sensitivity of risk to double parameter variation (probit model)

Fig. 9.3D – Sensitivity of risk to double parameter variation
(Weibull model)

- Performing a more detailed sensitivity analysis.
- Using an alternative model to change the sensitivity.
- Investigating the value of the parameter more thoroughly.

The choice of an appropriate action will depend on the state of current knowledge about the parameter. Although the results of an analysis may be quite sensitive to the parameter value, if that value is well known (i.e., has a narrow uncertainty range), additional investigation may be unnecessary. If, on the other hand, the parameter value is highly uncertain, because of limited knowledge, more research may be indicated.

Once we have identified those parameters that contribute most to the overall uncertainty, we must determine how much to spend in reducing this uncertainty. Although such a value impact study is beyond the scope of this report, we can suggest one simplistic approach to the problem. In this approach, one compares the cost of reducing the uncertainty in each parameter to the amount that total uncertainty is reduced. Those measures that have the highest ratio of uncertainty reduction to cost should be implemented first, other factors being equal.

CONCLUSIONS

Risk analysis for emissions of toxic materials has only recently become an important area. As a consequence, although the basic approach can be defined, applying this procedure to specific cases can be extremely difficult. Potential problems exist at every stage of risk assessment, and these problems will probably not be solved either easily or quickly. In addition to complicating the analysis, they contribute to the overall uncertainty in risk estimates that makes regulation of toxic emissions so complex and controversial.

During the course of our study, we have been able to identify many of these potential problem areas. Some are general, in that they relate to the selection and application of models and procedures for risk calculations. Other problems are more specific, in that they may be true for some analyses and not for others. These problems arise from

the characteristics of the particular pollutant or plant environment under consideration. In this section we discuss first some of the more general problems associated with risk assessment methodology, then we consider specific conclusions that can be drawn from our case study.

Environmental Transport

In this type of risk analysis, the multimedia environmental modeling is potentially the most difficult and time consuming task. By selecting a composite modeling approach, we have tried to develop a methodology that produces realistic results yet remains sufficiently flexible to be of use in a variety of circumstances. Appropriate models can be selected on the basis of problem requirements, data availability, user resources, and other factors. Unfortunately, the best models (in terms of all criteria) are not generally the most compatible models. Intermedia transfers and coupling of model results may be difficult, because these models are frequently designed by people with different purposes and requirements. In an integrated multimedia model, this problem has often been solved by simplifying the individual models, making them consequently less realistic and probably less accurate. In the composite approach, one must accept that certain intermediate conversions will be necessary, and try to minimize the difficulty of these conversions. As experience with this approach increases, these problems will undoubtedly become less important.

Properties of Toxic Materials

Risk assessment of chronic health effects has traditionally been associated with release of radionuclides by nuclear facilities. Although certainly not uniform in behavior or effect, radionuclides are in many ways more alike and better understood than the various toxic pollutants. Few of the priority pollutants have been studied in any detail. The best understood toxic materials are the trace elements. Yet no two of these pollutants behave similarly, and more information is needed about the (1) chemical and biological transformations and primary chemical species in air, soil, and water, (2) physical properties of these species, and (3) behavior in the various plant and animal organisms that provide secondary and tertiary exposure pathways. Many

of the organic materials have been studied even less than the trace elements and virtually nothing is known about what happens to them in the environment.

As a consequence, the exposure calculations must be highly uncertain. Even for the direct exposure pathways such as consumption of drinking water, we need to know far more about how the toxic organics and trace metals are affected by drinking water treatment and distribution processes. This type of information must be developed to reduce the uncertainty in future risk calculations, even given a set of realistic and usable computer models.

Health Effects

A primary weakness and source of uncertainty in the risk assessment methodology lies in the determination of health effects. The uncertainty in this area arises both from incomplete information about the behavior of toxic pollutants in the human body and their consequent health effects, and from a lack of understanding about which dose-response and extrapolation models are most realistic for particular pollutants. As we found in the case study, even though arsenic is a relatively well-known pollutant, we do not have the type and quality of data needed for accurate dose-response calculations. For the large majority of toxic trace elements and organic pollutants, we have even less information. Moreover, as we saw earlier, the choice of dose-response model can significantly influence the results of our risk calculations. Unfortunately, we have no real basis for selecting one rather than another, except for mathematical convenience.

Because of these problems, the calculation of health effects will remain extremely uncertain until we have the necessary research into the pharmacokinetics and health effects of the various pollutants. This work must include properly designed experiments to develop quantitative information about the chronic health effects, and particularly the carcinogenicity, of the materials. The selection of dose-response models will also remain relatively arbitrary, subject to the criteria discussed in Chap. 6, until the scientific community begins to understand the basic mechanisms of cancer.

In spite of these limitations, we should be able to improve the procedure for dealing with variable exposure rates and populations in the analysis. By necessity, we have assumed constant population distributions and exposure rates. In the near future, we expect to be able to develop and incorporate procedures to vary regional populations and exposure levels. Although the mathematical formulation of some dose-response relationships may not permit this, at least some should be amenable to modification.

Control Costs

The costs of controlling surface water emissions of toxic materials from power plants cannot be uniquely defined for two primary reasons. First, these costs depend not only on the type and specific source of pollution, but also on plant (1) processes, (2) waste streams, (3) water use and management systems, and (4) other pollution control requirements. Second, although a number of alternative control schemes exist for various toxic pollutants, the electric utility industry has had little or no experience with them. Utility discharges typically have large volumes and low pollutant concentrations. Most of the proposed control schemes have not been applied to this type of waste stream in a large-scale operation. Consequently, the costs and effectiveness of these processes have not been well defined.

Case-Study Results

Our case study was designed to illustrate how part of the overall risk-cost assessment methodology could be applied to a real problem. It was a hypothetical example, not related to any existing power plant, based on a combination of pessimistic assumptions and default values obtained from the general literature. Moreover, although we used a minimal Columbia River flow, this flow still exceeds the mean discharge of all but about 15 U.S. rivers. Similarly, the Columbia has a relatively high background arsenic concentration of 1.6 $\mu\text{g/l}$, more than seventy times greater than the EPA water quality criterion corresponding to a lifetime risk of 10^{-5} (EPA, 1980c). Under these circumstances, one should not consider our case-study results to be necessarily definitive

or typical of all coal-fired power plants. We can, however, still use these results and the sensitivity analysis to draw some limited conclusions about the risk analysis of arsenic emissions.

The arsenic concentrations of Table 8.16 and net risk values given in Table 8.21 indicate in our case that the incremental arsenic exposure and risk associated with our plant discharges are small. Arsenic concentrations in the Columbia River increased by 12.5 percent above background levels. Accordingly, we found that the arsenic emissions could lead to between 0.00164 and 0.329 additional cases of skin cancer in the regional population (Table 8.20), corresponding roughly to incremental skin cancer risks of between 0.016 and 3.29 in a million. Compared to the normal background levels of skin cancer in the United States, even the highest rate would be too small to detect with any confidence. In essence, we are calculating final risk estimates that cannot be verified. Thus, verification of the overall procedure must come from proper calibration and testing of the models at each stage of the analysis.

The sensitivity analysis indicated that net risk results can be extremely sensitive to the values of certain parameters and choice of dose-response model. The probit model is unduly sensitive to background arsenic exposure rate and would probably not be suitable for a real risk analysis. For the other dose-response models, the results vary significantly with the value of the coefficient specifying pollutant removal during drinking water treatment (TF). For arsenic, this coefficient cannot easily be specified. As described in Sorg and Logsdon (1978), arsenic removal will vary with a number of factors, including

- Chemical form of the arsenic present in the water.
- Initial arsenic concentration.
- Initial pH of the treated water.
- Treatment chemicals and processes used.

An actual risk analysis should use, whenever possible, measured values of pollutant removal for the water and treatment processes involved. If these are not available, the analyst should be careful to cover the potential range of removal values in the sensitivity analysis.

Other potentially important parameters may be more difficult to assess. The background pollutant concentration in outside food supplies (BMF) can affect the results of the multihit and probit models in particular. Unfortunately, for most pollutants this information may be totally unknown. Even for the trace elements, data are limited and site-specific information may not be available. Other constants, including the ingestion exposure dose scale factor, or feed rate constant (HS), and the dose-response model parameters, may also have to be obtained from the literature. When literature data are incomplete, uncertain, or inconsistent, sensitivity analysis must be used to determine or bound the effects of uncertainty in these parameters.

Case-Study Improvement

For various reasons, we limited the case study to direct exposure through consumption of surface water. Although this demonstrates how one might approach a risk analysis, it does not clearly illustrate the interconnections between environmental media and the potential importance of other exposure pathways. We have discussed these pathways in general terms in this report. For our specific case study, other significant pathways that could be considered in a more complete risk analysis include (1) inhalation of atmospheric arsenic (which may lead to increased incidence of lung cancer), (2) consumption of aquatic organisms, and (3) consumption of contaminated animal and vegetable products. Incorporating these pathways would require characterizing stack emissions from the power plant and calculating regional concentrations and deposition rates with an atmospheric transport model. An overland transport model could then be used to determine how much of the deposited pollutant remains in the surface soil and how much passes into the surface water network through runoff. Specific exposure calculations for the additional ingestion pathways would require collecting extensive information about local production of aquatic, animal, and vegetable products.

In addition to the above extensions, other improvements to the analysis could be made. Not only for the case study, but in general, integration of variable exposure rates and regional population

characteristics should provide more accurate estimates of the health effects than the time-averaged static values that we have assumed initially. The value of this integration, however, will depend on whether one can obtain good quantitative information about health effects of the pollutant. In the absence of such information, little benefit may be derived from the increased effort. As with the other areas of the calculations, the results depend on the quality of the data, given reasonable computer models. In a real analysis, one should use as much site-specific information as possible, and rely only as a last resort on default or literature values.

We have attempted to outline a unified approach to the risk assessment of toxic pollutants. This is at best a difficult process. Most of the priority pollutants listed by the EPA have not been studied extensively. What we do know about them indicates that they have few similarities in their behavior in either the environment or in man. Similarly, the characteristics of power plants and their sites vary significantly. Under these conditions, a risk assessment methodology must be formulated in extremely general terms, if it is to be sufficiently flexible to deal with a majority of the problems of interest. Unfortunately, this generality may make it more difficult to apply the methodology to a specific case.

The risk assessment process always demands a thorough understanding of the problem. One cannot expect to do an accurate analysis without knowing the basic characteristics of the power plant, regional environment, and toxic pollutant. Without this knowledge, the analyst will be unable to make the most appropriate assumptions, select the best tools and approaches, or evaluate the quality of the results. Not only would this cast doubt on the validity of the results, but it might also cause the work to be more difficult, extensive, and costly than otherwise necessary under the circumstances.

Appendix A

SOLUTION AND PARAMETERIZATION OF THE TWO-COMPARTMENT LINEAR PHARMACOKINETIC MODEL

This appendix summarizes the method of solution of Eq. (6.30),

$$\dot{c}_T = \alpha_{11}c_T + \alpha_{12}c_B \quad (A.1)$$

$$\dot{c}_B = \alpha_{21}c_T + \alpha_{22}c_B + k_o c_o / V_B$$

where

$$\alpha_{11} = -k_{21}/V_T \quad \alpha_{12} = k_{12}/V_T \quad (A.2)$$

$$\alpha_{21} = k_{21}/V_B \quad \alpha_{22} = -(k_{12} + k_o R + k_e)/V_B$$

Equations (A.1) are linear, nonhomogeneous, ordinary differential equations that can be solved analytically. The homogeneous case ($k_o c_o = 0$) can be solved by matrix methods (Kaplan, 1958). The eigenvalues of the coefficient matrix are real and distinct and are given by

$$2\lambda_{\pm} = (\alpha_{11} + \alpha_{22}) \pm \sqrt{(\alpha_{11} + \alpha_{22})^2 - 4(\alpha_{11}\alpha_{22} - \alpha_{21}\alpha_{12})} \quad (A.3)$$

It can be shown that $\lambda_{\pm} < 0$. The general solution for the homogeneous case is

$$c_{T,h} = C_1 e^{\lambda_+ t} + C_2 e^{\lambda_- t} \quad (A.4a)$$

$$c_{B,h} = C_1 e^{\lambda_+ t} v_{12} + C_2 e^{\lambda_- t} v_{22} \quad (A.4b)$$

where

$$v_{12} = (\lambda_1 - \alpha_{11})/\alpha_{12} \quad v_{22} = (\lambda_2 - \alpha_{11})/\alpha_{12} \quad (A.5)$$

and these have been determined from

$$(\underline{A} - \lambda \underline{I}) \underline{v} = 0, \quad \underline{A} = \begin{bmatrix} \alpha_{11} & \alpha_{12} \\ \alpha_{21} & \alpha_{22} \end{bmatrix}$$

to within an arbitrary constant.

CASES OF ADMINISTRATION BY INHALATION, CONTINUOUS INGESTION, OR INFUSION

In these cases, $k_o c_o$ in Eq. (A.1) is not zero and a particular solution must be found for the nonhomogeneous Eq. (A.1). Proceeding with the method of variation of parameters (Kaplan, 1958) (in which the C_1 , C_2 constants of Eqs. (A.4) are replaced by time-dependent functions and substituted in Eq. (A.1) to obtain expressions for these functions), one obtains the particular solution to Eq. (A.1):

$$c_T^* = \frac{k_o c_o \alpha_{12} / V_B}{\alpha_{22} \alpha_{11} - \alpha_{21} \alpha_{12}} > 0 \quad (\text{A.6a})$$

$$c_B^* = \frac{k_o c_o \alpha_{11} / V_B}{\alpha_{22} \alpha_{11} - \alpha_{21} \alpha_{12}} > 0 \quad (\text{A.6b})$$

The general solution is the sum of the particular (Eqs. (A.6)) and homogeneous solutions (Eqs. (A.4)), i.e.,

$$c_T(t) = c_T^* + c_{T,h}(t)$$

$$c_B(t) = c_B^* + c_{B,h}(t)$$

The boundary conditions here are

$$c_T(0) = 0, \quad c_B(0) = 0$$

which allow the C_1 , C_2 constants in Eqs. (A.4) to be evaluated. The final inhalation or continuous-intake solution is thus

$$c_T(t) = c_T^* + \left[\frac{\alpha_{12} c_B^* + (\alpha_{11} - \lambda_-) c_T^*}{\lambda_- - \lambda_+} \right] e^{\lambda_+ t} + \left[\frac{-\alpha_{12} c_B^* + (\lambda_+ - \alpha_{11}) c_T^*}{\lambda_- - \lambda_+} \right] e^{\lambda_- t} \quad (\text{A.7a})$$

$$c_B(t) = c_B^* + \left[\frac{(\lambda_+ - \alpha_{11}) c_B^* + \alpha_{12}^{-1} (\alpha_{11} - \lambda_-) (\lambda_+ - \alpha_{11}) c_T^*}{\lambda_- - \lambda_+} \right] e^{\lambda_+ t} \quad (\text{A.7b})$$

$$+ \left[\frac{(\alpha_{11} - \lambda_-) c_B^* + \alpha_{12}^{-1} (\lambda_- - \alpha_{11}) (\lambda_+ - \alpha_{11}) c_T^*}{\lambda_- - \lambda_+} \right] e^{\lambda_- t}$$

where c_B , c_T are given by Eqs. (A.6), $\lambda_{\pm}$ by Eqs. (A.3), and α_{ij} by Eqs. (A.2).

Equation (6.31) in the main text is equivalent to Eqs. (A.7) if the equation's dependence on inhalation concentration (i.e., dose) is separated out by redefining Eqs. (A.6) as

$$\begin{aligned} C_T^* &= c_o \beta_T \\ C_B^* &= c_o \beta_B \end{aligned} \tag{A.8}$$

and substituting these expressions in Eqs. (A.7).

PARAMETERIZATION OF THE MODEL

Equations A.7 describe the time-dependent concentration of drug in the blood and tissue (the presumed target of our toxic substance). However, the parameters in these compartmental equations are not known a priori and must be obtained from laboratory data (often human data in the case of drugs, but generally rat or mouse data in the case of environmental toxic substances).¹ The data most commonly available are those that comprise the experimental $c_B(t)$ curve and a limited amount of biopsy tissue concentration data. In the case of a substance that truly exists in only two distinct compartments, the blood curve shows a biphasic time dependence after an initial uptake section (see Fig. A.1). In the case of inhalation, the minimum set of parameters is composed of the α_{ij} set plus k_o/V_B (rate of respiratory uptake).

All the parameters may be determined from a nonlinear least squares or maximum likelihood fit to the blood and tissue data. If the transport between the blood and tissue compartment is equal in both directions ($k_{12} = k_{21}$), then only three of the α_{ij} need be determined

¹Although the subsequent description explicitly describes the parameterization of linear pharmacokinetic models, the approach also pertains to nonlinear generalizations of these models.

and a fit to the blood data alone is sufficient. In this case, the two slopes of the biphasic region (see Fig. A.1) and the intercept of the more outlying portion can be thought of as determining the α_{ij} unknown parameters. The k_o/V_B parameter in the inhalation curve is determined from the early rise and peak of the blood curve. (A convenient computer program for the nonlinear fitting of such data is found in the BMDP package (BMDP, 1981).)

Fig. A.1 – Concentration profile of an agent in blood if the agent follows the two-compartment formulation

OTHER CONSIDERATIONS

While the two-compartment linear pharmacokinetic model we have presented will apply to several substances, it must be modified if the agent is bound to tissue, is highly soluble in lipids, or is metabolized. Linear binding to tissues only leads to a rescaling of Eq. (6.30). High lipid solubility will require the addition of a third compartment representing adipose tissue. Linear metabolism would require that the equation representing the site of metabolism be modified to include a reaction sink term, and if the metabolite is the active species (itself not subject to nonlinear transport or metabolism), the addition of a second set of differential mass balances

to follow its history. However, these modifications will not affect the result, obtained in Eq. (A.1), that all compartment concentrations are proportional to dose.

Appendix B

PHYSIOLOGICAL PHARMACOKINETIC MODELS

These models were developed to allow a partial a priori prediction of drug or toxic agent distribution in the body. Because of their greater detail, they also provide better estimates of target cell toxic agent concentrations than the values inferred from a simple compartmental analysis. However, from the standpoint of potential users, these models pose difficulties because a large amount of experimental data is required, much of which will have to be obtained from new experiments.

In effect, these are also compartment models but the strict requirements are added that all compartments must correspond in volume to organs (or tissues), that all organs involved in agent distribution must be included, and that all transport parameters be defined in terms of interorgan blood flow, membrane transport, etc. Organ regions may be lumped but only if their binding and transport properties are very similar. Over the last decade, several applications of these models have been made to pharmaceuticals, including methotrexate (MTX) (Bischoff et al., 1971; Dedrick et al., 1973b), arabinosyl cytosine (Dedrick et al., 1973a; Morrison et al., 1975), digoxin (Harrison and Gibaldi, 1977), and lithium (Ehrlich et al., 1980).

As an example, we show how the physiologic model appears for the antitumor agent, methotrexate. The organ volumes (compartments) are shown in Fig. B.1 along with the circulatory connections between them. Note that the agent is cleared by the liver, finding its way into the bile and thence into the tubular cavity of the gut (the gut lumen) where a portion of it is reabsorbed (this is termed biliary recirculation). The model consists of writing a material balance for each organ.¹ For

¹Metabolism does not occur in these balances, which were modified from the early work of Bischoff et al. (1970, 1971) and Dedrick et al.

Fig. B.1 — Pharmacokinetic compartments of MTX distribution

the kidney, the balance is

$$V_K \dot{c}_K = Q_K \left(c_P - \frac{c_K}{R_K} \right) - k_K \frac{c_K}{R_K} \quad (B.1)$$

where V_K = organ volume,

c_K = average organ concentration,

(1973a). By 1978, assays were developed that recognized metabolism by the intestinal bacteria as well as conversion of MTX to polyglutamate forms. We have not updated this particular set of equations because we wish to keep them simple and focus on issues of transport. A metabolism example for another agent is discussed later.

c_P = plasma concentration,

Q_K = flow rate of blood to the kidney,

R_K = equilibrium tissue/plasma concentration ratio to account for the fraction of total organ concentration linearly bound to the tissue, and

k_K = urinary clearance corresponding to glomerular filtration.

Note that c_K/R_K is the unbound or free drug concentration in the kidney.

For muscle,

$$V_M \dot{c}_M = Q_M \left(c_P - \frac{c_M}{R_M} \right) \quad (B.2)$$

For the liver (see Fig. B.1),

$$V_L \dot{c}_L = \underbrace{(Q_L - Q_S - Q_G)c_P}_{(1)} + \underbrace{Q_S c_{Se}}_{(2)} + \underbrace{Q_G c_{Ge}}_{(3)} - \underbrace{Q_L \frac{c_L}{R_L}}_{(4)} - \underbrace{k_L \frac{c_L}{R_L}}_{(5)} \quad (B.3)$$

where term 1 is the inflow from the arterial circulation (hepatic artery), terms 2 and 3 are the inflows from the spleen and gastrointestinal system (the portal inflow), term 4 is the outflow from the liver, and term 5 is the first-order biliary clearance. For the plasma compartment (see Fig. B.1),

$$V_P \dot{c}_P = Mg(t) + \left(Q_L \frac{c_L}{R_L} + Q_K \frac{c_K}{R_K} + Q_M \frac{c_M}{R_M} + Q_B c_B \right) - (Q_L + Q_K + Q_M + Q_B) c_P, \quad (B.4)$$

where $Mg(t)$ is a function defining the administration of the dose M via injection or infusion, the first term in parentheses represents the organ venous outputs, and the second term in parentheses represents the

total arterial inflow to the organs. The flow down the biliary duct is modeled to allow for a lag time resulting from slow flow in the duct (rat) or perhaps retention in the gall bladder (human) via

$$\frac{1}{T} \dot{r}_1 = k_L \frac{c_L}{R_L} - r_1; \quad \frac{1}{T} \dot{r}_2 = r_1 - r_2; \quad \frac{1}{T} \dot{r}_3 = r_2 - r_3 \quad (\text{B.5})$$

where T = residence time.

The gut lumen is modeled by breaking up the tubular cavity into four equal sections. The material balances for the sections (each denoted by a number) are

$$\frac{V_{GL}}{4} \dot{c}_1 = r_3 - \frac{1}{4} \frac{k_G c_1}{K_G + c_1} - k_F V_{GL} c_1 \quad (\text{B.6})$$

$$\frac{V_{GL}}{4} \dot{c}_i = - \frac{1}{4} \frac{k_G c_i}{K_G + c_i} + k_F V_{GL} (c_{i-1} - c_i), \quad i = 2, 4$$

where the $k_G c_i / (K_G + c_i)$ terms represent the nonlinear absorption of agent from the contents of the lumen (k_G , K_G are constants), and k_F is the fecal clearance rate.

The remaining organs of the spleen, gastrointestinal tissues, and bone marrow are different from the organs considered so far. Underlying the organ balances above is the assumption that the concentration dynamics are flow-limited; the organ is thus treated as a well mixed reactor with the outflow concentration equal to the concentration throughout the organ. In the case of MTX, this assumption breaks down for the spleen, G.I. tract, and marrow.

These organs must have their extracellular fluid spaces, which are flow-limited, treated separately from their intracellular spaces. The reason is that the membranes that separate these spaces contain enzyme-like pumps responsible for transport. These pumps may be saturated (forced to operate at near maximum capacity) at high drug levels and thus the pharmacokinetics become quite nonlinear. If these effects were

not taken into account, then toxic agent concentrations in these tissues (which can be viewed as target tissues) would be greatly overestimated if one were to assume they were like most other tissues.

To treat these organs, the extracellular space is described as being similar to the kidney or muscle spaces,

$$V_{Xe} \dot{c}_{Xe} = Q_X(c_p - c_{Xe}) - J_X, \quad (B.7)$$

where Xe = extracellular space of organ X (either the marrow or spleen),

J_X = function describing the rate of material flow across the membrane barrier.

J_X is given by

$$J_X = \frac{k_e V_X c_{Xe}}{K_e + c_{Xe}} - \frac{k_i V_X c_{Xi}}{K_i + c_{Xi}} + bV_X(c_{Xe} - c_{Xi}), \quad (B.8)$$

where c_{Xi} = intracellular concentration of agent.

The first two terms represent the facilitated transport balance between the extracellular and intracellular spaces, and the last term allows for linear transport.²

The balance on the intracellular space is more complicated because, in the case of MTX, not only saturable transport is involved but also strong binding of the drug to a particular enzyme. The balance here is

$$V_{Xi} \dot{c}_{Xi} = J_X / \left[1 + \epsilon a_X / (\epsilon + c_{Xi})^2 \right] \quad (B.9)$$

² b is very small for MTX and becomes important only at high concentrations where the first terms are saturating.

where a_X = strong binding enzyme concentration, and
 ϵ = dissociation constant of the drug-enzyme complex.

The balance for the G.I. tissues is slightly different from that in Eq. (B.7) because allowance must be made for absorption of agent from the gut lumen (see Fig. B.1). The G.I. material balance is

$$V_{Ge} \dot{c}_{Ge} = Q_G(c_P - c_{Ge}) - J_G + \sum_{i=1}^4 \frac{k_G c_i}{K_G + c_i} \quad (B.10)$$

for the extracellular space, and is Eq. (B.9) for the intracellular space.

For this particular example, the physiologic model is composed of the 17 organ, lumen, and biliary equations given above. For other chemical substances, the physiologic model will be different. As a further example, the case of arabinosyl cytosine (another antitumor agent) can be considered. The same organs are involved as in Fig. B.1 but no biliary recirculation occurs, eliminating the need for Eqs. (B.5) and (B.6) (Morrison et al., 1975). On the other hand, the active species of this compound is not the form administered but a phosphorylated species; arabinosyl cytosine also undergoes deamination to a nontoxic metabolite. Therefore this metabolism requires that material balances be written for all three major chemical species involved³ for each of the seven organ spaces, leading to a model consisting of 21 equations.

PARAMETERIZATION OF THE MODEL

Equations (B.1) to (B.10) are examples of the nonlinear ordinary differential equations that describe the concentration of a toxic agent in a specified anatomical region. As in the compartment models, the parameters must be obtained from experimental data but some are available from literature references.

³Arabinosyl cytosine, arabinosyl uridine, and arabinosyl cytidine triphosphate.

In particular, the organ volumes (V), blood flow rates (Q), biliary residence times (T), and fecal flow rates (k_F) are available from Table 1 in Bischoff et al. (1971) for the mouse, rat, dog, monkey, and man. This table excludes the spleen and marrow but these values may be found in Table I of Dedrick et al. (1973a) for the rat and Table 2 of Morrison et al. (1975) for the mouse. For animals with weights different from the standard body weights used in these references, scaling relationships are available that allow the prediction of appropriate volume, flow, and clearance parameter values (Adolph, 1959; Smith, 1951; Bischoff et al., 1971, Appendix). The general form of these correlations is parameter = K (body weight)^a and values of the K and a are found in the above references.

The parameters in the model (R_X , k_G , K_G , k_e , K_e , k_i , K_i , b , ϵ , and a_X) must be obtained from experimental data on animals or humans. In general, the plasma/tissue partition constants (R_X) and the transport parameters (k_e , K_e , k_i , K_i , and b) must be obtained from fitting the integrated form of the physiological equations to time-dependent tissue concentration data obtained at various dosage levels. This requires extensive animal data, close collaboration with the experimentalists, and probably some biopsy data to extend the animal results to humans. (For example, in the case of digoxin, the R_X values for rats and humans are quite different (Harrison and Gibaldi, 1977).) Gut reabsorption rates may be estimated from animals or humans having surgically removed biliary function, or perhaps from extensive fitting to plasma concentration curves. Dissociation constants (ϵ) may be estimated from isolated enzyme studies or cellular assays. The strong-binding enzyme content may be determined from biochemical analyses of tissue biopsy specimens. If a compound of interest undergoes metabolism, then all these tests must be available for the metabolites as well.

Integration of the differential equations must generally be done numerically and can be accomplished by the same programs described in the main text, in the section on nonlinear pharmacokinetics. The one major difference between the physiological and compartment models is the number of equations involved. The greater number in the physiologic case often prohibits statistical analysis or makes it quite expensive.

Appendix C

MULTIMEDIA MODEL COUPLING¹

In Chap. 4 we described the environmental models that have been selected for the composite multimedia methodology. This appendix discusses how these models should be combined to simulate contaminant migration and fate in a multiple media analysis.

SEQUENTIAL ENVIRONMENTAL PATHWAYS

As we discussed earlier, coal-fired power plants have four main sources for toxic chemical emissions into the environment. These sources are (1) stacks, (2) coal piles and landfills, (3) ash and sludge ponds, and (4) liquid effluents directly discharged to receiving surface waters. Any other releases may be treated as one or a combination of these four sources. The transport and fate of toxic chemicals released in these four ways will be controlled by transport; degradation and decay; transformation in primary and secondary pathways; and intermedia transfer among air, overland, groundwater and surface waters. These interactions are shown in Fig. C.1.

Stack Effluent Releases

Gaseous and particulate effluents released from a plant stack as the source first enter the environment through the air (Fig. C.2). Hence, an air model (RAPT, ANDEP, or STRAM), as a primary pathway, will be used first to calculate chemical concentrations in the air. The model also calculates wet and dry deposition rates of the chemicals to the land surface and surface water, which are secondary pathways to receive these originally airborne chemicals. Chemicals deposited on the land surface will then migrate to a receiving body of surface water or

¹This appendix has been summarized from Vol. 1 of Onishi et al. (1982).

SOURCE: Onishi et al. (1982).

Fig. C.1 – Interactions between the transporting media

SOURCE: Onishi et al. (1982).

Fig. C.2 — Sequential primary and secondary pathway models for stack effluent

will infiltrate groundwater. With output (wet/dry deposition rates) from the air model, this phenomenon will be simulated by ARM, which estimates runoff, sediment, and chemical (both dissolved and particulate) loadings at a receiving stream edge. ARM also calculates infiltration of water and dissolved chemicals into a groundwater system.

If infiltrated water and dissolved chemicals reach an aquifer, they will migrate through the groundwater system and may seep into surface water systems. These transport and seepage pathways will be simulated by VTT and MMT models, with fluxes of infiltrated water and the chemicals computed by ARM.

Surface water may also receive chemicals originally released from a stack through dry and wet deposition, from the land through runoff and soil erosion, and from the groundwater as seepage. These chemicals will then be transported by surface water or will accumulate in the bed sediment of the surface water body. This instream transport and accumulation will be simulated by one of the surface water models, SERATRA, TODAM, or EXAMS, with an appropriate hydrodynamic model. Sources for the surface water model will be the output (discharges and loadings) from the air, overland, and groundwater models.

Contaminated surface water can infiltrate groundwater systems that may be used as a drinking water or irrigation water source. The groundwater system will then be simulated by the VTT and MMT models using output from the surface water model.

The sequential primary and secondary pathways with their interactions are shown in Fig. C.2. Depending on the specific site and its problems, some of the secondary pathways shown in Fig. C.2 may not be necessary.

Coal-Pile and Landfill Emissions

Sequential primary and secondary pathways for releases from coal piles and landfills are shown in Fig. C.3. The chemicals released through coal-pile or landfill sources have potentially three primary pathways.

The first primary pathway is the groundwater system 1, which receives rainfall infiltration directly from coal piles and landfills. This system includes both unsaturated and saturated groundwater systems. The vertical movement of water from a coal pile to a regional aquifer will be computed by the UNSAT model over a number of time steps. The saturated groundwater model, VTT, will then calculate the water movement in the receiving aquifer with the water flux computed by UNSAT. The water table directly below the coal pile computed by VTT will be supplied to UNSAT to estimate the water flux to the aquifer at subsequent time steps. UNSAT may run a number of time steps before the computed water flux to the aquifer will be supplied to VTT to simulate the movement of saturated water. This loop will be repeated over the

SOURCE: Onishi et al. (1982).

Fig. C.3 — Sequential primary and secondary pathway models for releases from coal piles and landfills

entire simulation duration. Once the water movement in unsaturated and saturated groundwaters is computed by UNSAT and VTT, the chemical transport model, MMT, will simulate the chemical transport in both unsaturated and saturated groundwaters with water velocities and porosities computed from UNSAT and VTT along with additional data on the site-specific retardation and chemical source terms.

Any chemical seepage flux to a surface water will be computed by MMT and be given to surface water models as part of the input data. If water seepage flux to surface water is important, the water flux can also be computed by VTT and can be given to surface water flow models as a part of the input data.

The second primary pathway for releases from the coal pile or landfill is the watershed (overland) that transports runoff, sediment,

and chemicals (dissolved and particulate) over the land surface from the coal-pile and landfill site to the receiving surface water. ARM will simulate this process and provide water, sediment, and chemical contributions to the surface water models.

The third primary pathway is air, where the suspension of chemicals from the coal pile and landfill occurs. As discussed earlier (simulated by the ISC model in this case), the air pathway, in turn, provides the secondary pathways, i.e., to surface water and land surface through dry deposition. These pathway couplings are identical to those discussed for stack emissions. Overland modeling by ARM will provide input to the surface water models and the saturated groundwater system 2 models. Groundwater system 2 is most likely different from groundwater system 1, which receives the toxic chemicals directly from the coal pile and landfill.

These various primary and secondary pathways provide toxic chemical contributions to a receiving surface water body at different locations through seepage from groundwater, direct dry and wet deposition from air, and runoff and soil erosion occurring over land surfaces. This portion of the simulation will be done by SERATRA, TODAM, or EXAMS with appropriate hydrodynamic models. Contaminated surface water at various downstream locations may infiltrate into the saturated groundwater system 3.

Although there are many primary and secondary pathways for releases from coal piles and landfills as discussed, only a few of the pathways shown in Fig. C.3 are likely to be needed for a specific application, partially because the impact of coal piles and landfills may be relatively limited in degree and in space compared to other releases.

Ash and Sludge Pond

The third release source for toxic chemicals from a coal-fired power plant is through an ash and sludge pond (Fig. C.4). In this case, contaminated water in the pond infiltrates the groundwater system. If a regional aquifer reaches the bottom of the pond, the saturated groundwater model, VTT, and the chemical transport model, MMT, are used to simulate this groundwater system. The system represents a primary

SOURCE: Onishi et al. (1982).

Fig. C.4 — Sequential primary and secondary pathway models for releases from ash and sludge ponds

pathway. However, if a regional aquifer does not reach the pond, the vertical water movement in the unsaturated zone downward to a receiving saturated regional aquifer and the subsequent horizontal water movement within the aquifer will be simulated by the combination of UNSAT and VTT as described for the primary groundwater system in the previous section. Chemical migration in both unsaturated and saturated groundwaters is simulated by MMT from the flow conditions simulated by UNSAT and VTT.

Any seepage of groundwater contaminant to surface water will be computed by VTT and MMT and supplied to the surface water modeling component. Chemical concentrations computed by one of the surface water models can be supplied to a more regional saturated groundwater system (secondary pathway). In this case, the chemical concentration in the

surface water must be converted to a chemical flux to the groundwater system.

Liquid Effluent to Surface Water

The fourth release source is direct discharge of liquid effluent into receiving surface water. The primary pathway is, of course, the surface water (Fig. C.5). SERATRA, TODAM, or EXAMS with hydrodynamic conditions simulated by a flow model will simulate the migration and fate of the chemicals released to surface water.

The secondary pathways for this case are the saturated groundwater system and the overland system. The overland system becomes involved when contaminated surface water is used for irrigation. In this case, the water and chemicals supplied to the land surface will be estimated

SOURCE: Onishi et al. (1982).

Fig. C.5 — Sequential primary and secondary pathway models for liquid effluents discharged to surface waters

from the irrigation water pumping rate and chemical concentration of the water. ARM may treat the irrigation effect as if it is a wet deposition from the air model. The computed infiltration of water and dissolved chemicals to the groundwater system will be supplied to regional VTT and MTT models if the analysis of this pathway is deemed necessary.

Groundwaters may receive toxic chemicals from surface water and overland sources in the same or different aquifers depending on the site. Water and chemical contributions from the surface water and the land surface are input to VTT and MMT as fluxes to the groundwater systems.

SPECIFIC COUPLING CONSIDERATIONS

The generic problems associated with coupling one code with another involve the following major areas:

1. Data base.
2. Time discretization differences.
3. Unit conversion.
4. Space discretization differences.

The data base problem requires insuring that data used as input to more than one model are taken from the same source whenever possible. Moreover, if these input data must be used at different time or spatial scales in the different models, the appropriate time or spatial averaging methods must be used to maintain common material or energy balance between the various codes. These averaging processes generally smooth the data, removing variations that often cannot be recovered when returning to the original time or spatial scale.

The various models are chosen so that one model's output acts as a source input or boundary condition to the next model. These models generally have different time constants and spatial resolutions. For example, surface water models of river systems in many cases discretize a modeling area into one-dimensional space and are very dynamic in time

(often requiring time steps in minutes). Codes for regional groundwater systems, however, generally require a modeling area to be at least two spatial dimensions (with monthly time steps). In terms of coupling, the problem involves insuring mass and energy continuity between the various models.

Coupling between the models of the multimedia system will be accomplished by small data manipulation codes that take the output from the "supplying" models and perform the proper time and spatial averaging, interpolation, and appropriate unit conversion to prepare the input stream for the next "receiving" model. This procedure of using the normal output stream from a supplying model does not create any great new difficulty in preparing the receiving model's input. Primary data are rarely found in the proper form in the literature, and small data manipulation codes, as discussed above, are generally required to manipulate these primary data into the proper time and space discretization for model input.

We must also deal with unit conversion and time resolution differences. For unit conversion, one must insure that the proper conversion factors are used to transform the output units from the supplying model to the input units required by the receiving model. Sometimes this will also require combining output streams, because the receiving model may, for example, require concentration and flux as input whereas the supplying model provides only contaminant mass flux and water flux. Time resolution problems involve insuring that proper interpolation or averaging procedures, consistent with the supplying model's output and the receiving model's input requirements, are met in such a way that mass and energy are conserved. For example, the output from some model may represent a time-dependent flux or concentration, whereas another model's input may require the flux or concentration to be constant over time. These interpretational differences between the supplying model and the receiving model, along with time resolution differences, must be taken into account in the small data manipulation codes discussed above.

The major difficulties in code coupling are caused by differences in spatial resolution. Spatial resolution problems arise for a variety of reasons; the more generic ones relate to:

1. Projection (from real latitude-longitude data to some flat system).
2. Coordinate system type (e.g., Cartesian or polar).
3. Coordinate system origin.
4. Coordinate system rotation.
5. Grid (type and scale of spatial resolution) used within the selected coordinate system (e.g., finite difference or finite element).

These major spatial resolution differences can be handled in a generic manner. The approach for these generic problems involves the following steps:

1. Convert data in other projections to latitude and longitude.
2. Convert a projection from latitude and longitude (e.g., Lambert, Standard Mercator, etc.) to a common Cartesian coordinate system with a given origin and rotation using a standard currently available computer program.
3. Convert data with the correct projection but in a grid system with a different origin rotation or regular grid spacing to the common Cartesian system with a standard data base program currently available.
4. Convert data with the correct projection and Cartesian system but on a finite element grid to a finite-difference grid with generally minor modifications using existing programs.

This procedure should be able to address all the generic problems associated with spatial resolution.

Other coupling problems related to spatial resolution are associated with the dimensionality of the codes. For example, the VTT groundwater flow model is a two-dimensional (x-y) finite-difference code, whereas the TODAM surface water code for rivers is one-dimensional with its input represented by line segments through the x-y plane. The coupling of these codes through space must be handled on a problem-specific basis. The user must determine what VTT model nodes or

fraction of nodes map onto what TODAM model river segments. The translator program to convert VTT model output into TODAM model input must take this spatial mapping into account. Similar spatial mappings are also required to link ARM and the selected air model to the selected river model.

COUPLING WITH AIR MODELS

Air models are used for stack, coal pile, and landfill sources (Figs. C.2 and C.3). For these sources, air is the primary pathway. Hence, output from air models will be used by other pathway models, but none of the other pathway models will provide input to the air models. Characteristics of the air models RAPT, ANDEP, STRAM, and ISC were presented in Fig. 4.2.

The models used for simulating stack effluents are RAPT (regional modeling), STRAM, and ANDEP. The output from these models is used as input to the land and surface water models. Outputs from RAPT and STRAM are similar, because they are both based on a sampling grid in the National Meteorological Center (NMC) grid (Onishi et al., Vol. 2, 1982). Output data from ANDEP are generated on a polar grid with an option to output the model results on a Cartesian grid. The inputs required by the overland and surface water models are dry and wet deposition values on a Cartesian grid. Thus, ANDEP's output can be interpolated to the Cartesian grid as input to those secondary pathway models.

To invert the NMC grid results obtained by RAPT and STRAM to latitude and longitude and then to a Cartesian grid, one must

1. Add the lower left-hand corners of the grids to the grid point divided by the number (grid divisions/NMC grid division) for both x and y components. This yields the output grid points in terms of NMC grid points.
2. Convert these grid points to latitude and longitude points using

$$\text{LAT} = 90 - (360/\pi) \tan^{-1} [(X_{\text{NMC}} - 24)^2 + (Y_{\text{NMC}} - 24)^2]^{1/2} / 31.184$$

$$\text{LON} = 80 + (180/\pi) \tan^{-1} [(X_{\text{NMC}} - 24)/(Y_{\text{NMC}} - 24)]$$

3. Use an appropriate map scale factor to convert this latitude and longitude grid to a Cartesian grid compatible as input to the secondary pathway models.

Atmospheric pickup and transport of particles is one of three paths leaving the coal pile and landfill. ISC simulates these sources. One output is dry deposition, which becomes an input to the overland and surface water models. These models require that the output from ISC be on a compatible Cartesian coordinate grid. Because ISC can generate output on a Cartesian grid, the only interface required is a linear interpolation to the compatible grid with a scale factor to ensure that the units are compatible between models.

COUPLING WITH OVERLAND MODELS

The overland model ARM can receive contamination from three sources. First, atmospheric migration may be the source of contaminants for the overland surface site. In this case, the contaminants may have originated from a point relatively far from the area being modeled. Second, the overland surface may receive contaminants by direct application from an area source, such as a coal pile or surface landfill. Third, the overland surface may be contaminated by irrigation water pumped from contaminated surface water.

The overland model ARM can provide contaminant initial and boundary conditions to groundwater and surface water simulation models. ARM can supply the amount of contamination within several different subsurface layers at varying times. ARM also supplies surface water bodies with a time-varying hydrograph of flow, sediment, and contaminants, considering both dissolved and particulate (adsorbed to sediment) phases of the contaminant.

We will discuss below the methods to be used in coupling the overland model with other pathway models. These coupling considerations are summarized in Tables C.1 and C.2.

Table C.1

COUPLING REQUIREMENTS FOR INPUTS TO THE ARM MODEL

Submodel	Spatial Resolution	Time Resolution	Unit Conversion
RAPT	Convert from NMC to Cartesian grid	Linear interpolation or one-to-one correspondence (month to 5 min-hourly)	Convert deposition (kg/m^2) to mass (kg)--English or SI units
STRAM	Convert from NMC to Cartesian grid	Linear interpolation or one-to-one correspondence (month to 5 min-hourly)	Convert deposition (kg/m^2) to mass (kg)--English or SI units
ANDEP	Convert polar to Cartesian grid--linear interpolation	Linear interpolation or one-to-one correspondence (month to 5 min-hourly)	Convert deposition (kg/m^2) to mass (kg)--English or SI units
ISC	Linear interpolation and scale factor	Linear interpolation or one-to-one correspondence (month to 5 min-hourly)	Convert deposition (kg/m^2) to mass (kg)--English or SI units

NOTE: SI--International System of Units

Table C.2

COUPLING REQUIREMENTS FROM THE ARM MODEL TO OTHER PATHWAY MODELS(a)

Submodel	Spatial Resolution	Time Resolution	Unit Conversion
Surface water	Linear interpolation (1 km to 10 km)	Interpolation of linear relationship (5 min to 1 hr)	Movement of water, sediment, and contaminants as flux, concentration, or mass
Ground-water	Convert irregular grid (sq mi) to regular grid (acres to hundreds of acres)	Linear interpolation over time step (5 min to 1 hr)	Movement of water and contaminants as mass per unit time increment

(a) ARM model output for coupling: (1) runoff amount at stream edge; (2) sediment loading at stream edge; (3) dissolved contaminant loading at stream edge; (4) particulate contaminant loading at stream edge; (5) infiltrated water flux to groundwater system; and (6) dissolved contaminant flux to groundwater system.

Interaction Between Overland and Atmospheric Environments

The actual interfacing between the overland and atmospheric models occurs at the land surface. The atmospheric model defines a grid system for identifying contaminant concentrations due to wet or dry deposition. Based on this spatial resolution, actual contamination attributed to a specific watershed catchment would be determined. The contamination would be considered uniformly distributed over the overland catchment using a weighting technique if the catchment existed within several grids as defined by the atmospheric scenario. Atmospheric meteorological information will be provided in time intervals (e.g., one hour) that are compatible with the modeling capabilities of ARM. The contamination of the overland area represents initial conditions for the overland modeling scenario. Updating contaminant contributions from the air at every time increment cannot be done with ARM. Depending on the version of ARM (I or II), one to twelve contaminant depositions can be specified during a simulation period. If a more frequent update of overland conditions is needed, the model can be stopped and conditions can be manually updated.

Interaction Between Overland and Surface Water Environments

The interfacing between overland and surface water models occurs at the boundary of the water body and overland segment. ARM provides the flux of water, sediment, and contaminants, and sediment and contaminant concentrations at the water body boundary. The overland modeling output represents boundary conditions for the surface water models. Temporal and spatial considerations between overland and surface water models are comparable, so little modification to the data is required at the interface.

Interaction Between Overland and Groundwater Environments

The interfacing between overland and groundwater models occurs at several points:

1. Spatial resolution.
2. Temporal resolution.
3. Continuous simulation.
4. Mass balance.
5. Model conceptualizations.
6. Physical points at which the models connect.
7. Actual water-contaminant transport scenario.

Many groundwater models use numerical techniques to compute the migration of water and contaminants. The computations are usually along points called nodes where each node represents the average conditions surrounding it. ARM does not employ this type of technique in computing the migration of water and contaminants to and in the subsurface environment. ARM uses a mass balance approach in which a bookkeeping system is established per time interval for inputs, outputs, and changes within the storage volume. For groundwater models to interface with ARM, a minimum of one node must be associated with the overland area being modeled; several nodes may be associated with the area, depending on the spatial resolution employed in the groundwater model. One or several nodes could then describe the subsurface environment under each overland area being modeled by ARM.

Figure C.6 presents the subsurface conceptualization of water-contaminant movement as described by the ARM model. Four basic zones describe the subsurface environment: surface, upper, lower, and groundwater. The first three deal with unsaturated flow movement, and the last deals with saturated flow movement. Figure C.6 includes a description of each zone. For more information, refer to the Bibliography.

The groundwater codes and ARM would actually interact in the groundwater zone. Because the overland simulation of water and contaminant movement is affected by the movement of water and contaminants below the surface, it would be very difficult to separate the overland and subsurface parts of ARM. An unsaturated groundwater model could be combined with the overland migration modeling sections in ARM but many difficulties would arise:

SOURCE: Onishi et al. (1982).

Fig. C.6 – Groundwater conceptualization of the ARM model

1. Because ARM was not developed in a top-down fashion, code modification and verification would be difficult.
2. Many components in both models (ARM and the unsaturated groundwater model) would be the same (e.g., evapotranspiration, infiltration, and computations). This similarity would create problems with mass balance in one of the models (depending on which components of which models were used).
3. The level of conceptualization of the models would be inconsistent, because ARM is a lumped parameter model in which variables describe uniform conditions over time periods. Unsaturated groundwater models are normally more sophisticated.

More information about these characteristics of ARM can be found in Vol. 2 of Onishi et al. (1982).

These considerations imply that ARM would have to be combined with the saturated groundwater code. Groundwater modeling algorithms describing the hydrodynamics could take the place of those in ARM in the groundwater zone (see Fig. C.6). Because the groundwater zone represents the last subsurface block in ARM, coupling both models here would not mean a major revision of ARM.

In ARM the flux of contaminants and water into the groundwater zone is not a function of the parameters in the groundwater zone. Therefore, the net fluxes from the lower zone would act as boundary conditions for the groundwater code, VTT. In this way, mass balance will be conserved.

During periods of precipitation, the computational increments are 15-min intervals. Between precipitation events, time intervals are daily. This allows for continuous simulation with reasonable computer costs.

As mentioned previously, several conditions limit the application of the overland modeling. All overland simulation areas must have a surface water body as their down-gradient boundary. ARM cannot simulate the migration of flow, sediment, or contaminants from one overland segment to another overland land segment. In addition, all contamination is assumed to be uniformly distributed over the land surface and within the subsurface layers. The uniform areal

distribution of contamination can be assured by reducing the size of the ARM simulation area.

The initial contamination of an area has to exist before the overland simulation. Interactions between models at each time interval in the modeling scenario are not possible at this time and may prove insignificant, time consuming, and costly. However, it is possible to update surface contamination levels at specified time intervals. Sinks and sources cannot be considered at this time and a unidirectional modeling pathway is required during the actual simulation between the overland model and models representing the other environments.

COUPLING WITH GROUNDWATER MODELS

Groundwater codes selected for the methodology are the one-dimensional UNSAT and the two-dimensional VTT for unsaturated and saturated groundwater. Chemical transport in a groundwater system is simulated by the MMT code. As shown in Figs. C.2 through C.5, the groundwater pathway appears for all four sources. Coupling of the groundwater pathway with other media is through creation of output files that define needed inputs to other models lower in the hierarchy. The coupling considerations for the selected groundwater codes, UNSAT, VTT, and MMT, are illustrated in Tables C.3 to C.5.

Unsaturated zone flow modeling is at the top of the hierarchy for the coal pile, landfill, and sludge pond cases. Inputs to UNSAT are from the source characterization data as well as the feedback from VTT, as discussed in Vol. 2 of Onishi et al. (1982). The saturated zone model for flow, VTT, can be a primary pathway code or a secondary pathway code. As a primary pathway model, VTT uses source characterization data as input. As the secondary pathway code, VTT is fed via output files produced by UNSAT and other supplying models handling the land surface and surface water portions. MMT then uses the output files made by UNSAT and VTT, together with chemical distributions used as boundary and initial conditions to simulate chemical transport and fate in the groundwater system.

Table C.3

COUPLING REQUIREMENTS FOR UNSAT MODEL(a)
(UNSATURATED GROUNDWATER)

Coupling Model	Spatial Resolution	Time Resolution	Unit Conversion
Saturated groundwater flow (MMI)	Map from regular grid nodes to one-dimensional columns	Simple extrapolation, as both models assume values constant over the time interval (large steps to small steps)	Simple adjustment for surface water table elevation
(a) UNSAT model output: (1) potential distribution; (2) moisture content distribution; (3) mass balance information; (4) instantaneous and cumulative fluxes. UNSAT model output for coupling: (1) water flux for VIT model; (2) time-varying velocity field for MMI.			

Table C.4

COUPLING CONSIDERATIONS FOR VIT MODEL(a)
(SATURATED GROUNDWATER FLOW)

Coupling Model	Spatial Resolution	Time Resolution	Unit Conversion
Overland (ARM)	Irregular grid (sq mi) to regular grid (acres to hundreds of acres)	Simple averaging, as both models assume values constant over the time interval (small steps to large steps)	Simple unit conversion for mass flux
Unsaturated groundwater flow (UNSAT)	Map from one-dimensional columns to regular grid nodes	Simple averaging, as both models assume values constant over the time interval (small steps to large steps)	Simple unit conversion for mass flux
Surface water hydrodynamic models	Map from river segments to regular grid locations and intercept fractions	Simple averaging, as both models assume values constant over the time interval (small steps to large steps)	Simple adjustment for reference datum (water elevation)
(a) VIT model output: (1) potential; (2) velocities; and (3) flow paths or streamlines with travel time. VIT model output for coupling: (1) water table elevations versus time for the UNSAT model; (2) mass flux of water to river nodes for input to the surface water submodels; and (3) time-varying groundwater velocity fields for input to the MMI model.			

Table C.5

COUPLING REQUIREMENTS FOR MMT MODEL (a)
(GROUNDWATER TRANSPORT)

Coupling Model	Spatial Resolution	Time Resolution	Unit Conversion
Unsaturated groundwater flow (UNSAT)	Utilizes same grid	Averaging or interpolation depending on the contaminant and its retardation	None, same units
Saturated groundwater flow (VTT)	Utilizes same grid	Averaging or interpolation depending on the contaminant and its retardation	None, same units
Unsaturated groundwater transport (MMT)	Map from 1-dimensional columns to regular grid nodes	Averaging or interpolation depending on the contaminant and its retardation	None, same units
Surface water transport models	Map from river segments to regular grid locations and intercept fractions	Simple averaging, as both models assume values constant over the time interval (small steps to large steps)	Simple unit conversion (concentration to mass flux)

(a) MMT model output: (1) concentration; and (2) fluxes across outflow boundaries. MMT model output for coupling: (1) contaminant mass flux from unsaturated zone transport for input to the MMT code; and (2) contaminant mass flux from saturated zone transport for input to the surface water model.

COUPLING WITH SURFACE WATER MODELS

In the modeling scenarios presented in Figs. C.2 through C.5, the surface water phase appears in various positions of the modeling sequence. Each of the other pathway models (air, overland, and groundwater transport) is coupled as input into the surface water model in one or more of the scenarios. In turn, the surface water model transmits information to the groundwater and overland transport models in each of the four pathways presented. A summary of the coupling requirements between the surface water models and other pathway models is shown in Tables C.6 and C.7, with more detailed discussion following.

The surface water sediment-contaminant transport code, SERATRA, must be supplied with hydrodynamic data before its execution. Therefore, all contributions of water from the various media pathways will be combined into composite discharge hydrographs to be routed by a hydrodynamic model before the surface water simulation. With a complete hydrodynamic data set, SERATRA will use the contaminant and sediment information from the other pathway models as boundary conditions. SERATRA can receive this information as depth-dependent influx or in a depth-averaged form. Upstream boundary conditions are input as concentrations, kg/m^3 , at each time level and lateral boundary conditions are input as mass fluxes, kg/s .

TODAM is a one-dimensional sediment-contaminant transport code with attributes similar to SERATRA. TODAM must also be supplied with hydrodynamic data before its execution. Although not capable of depth-dependent resolution, TODAM otherwise has data requirements identical to SERATRA.

Like SERATRA, EXAMS requires data from the other media models in terms of loading rates. Water is input as m^3/hr and the sediment and contaminant data enter as kg/hr . One of the major differences between the other two surface water codes and EXAMS is that EXAMS uses constant loading rates, whereas SERATRA and TODAM update time-varying loading rates at each time interval. An averaging scheme must therefore be used on time-dependent information supplied to EXAMS by the other media models. EXAMS does not simulate sediment transport. We recommend using water-sediment routing models to assist EXAMS in determining loading

Table C.6

COUPLING REQUIREMENTS FOR EXAMS(a)
(SURFACE WATER)

Coupling Model	Spatial Resolution	Time Resolution	Unit Conversion
RAPT (air)	Convert from NMC to Cartesian grid system (50 km to 1 km)	Average input data to steady-state loading (month to day)	Convert deposition ($\mu\text{g}/\text{m}^2$) to loading rate (kg/hr)
STRAM (air)	Convert from NMC to Cartesian grid system (1 km to 1 km)	Average input data to steady-state loading (month to day)	Convert deposition ($\mu\text{g}/\text{m}^2$) to loading rate (kg/hr)
ANDEP (air)	Simple interpolation (1 km to 1 km)	Average input data to steady-state loading (month to day)	Convert deposition ($\mu\text{g}/\text{m}^2$) to loading rate (kg/hr)
ISC (air)	Simple interpolation (1 km to 1 km)	Average input data to steady-state loading (month to day)	Convert deposition ($\mu\text{g}/\text{m}^2$) to loading rate (kg/hr)
ARM (overland)	Simple interpolation (1 km to 1 km)	Average input data to steady-state loading (hour to day)	None, same units for loading rate
MMT (ground-water)	Simple interpolation (10 km to 1 km)	Average input data to steady-state loading (day to day)	Convert flux from kg/s to kg/hr

(a) EXAMS output: (1) spatial distribution of dissolved contaminant concentrations (mg/l); (2) spatial distribution of contaminant sorbed to sediment (mg/kg); and (3) contaminant sorbed to biota ($\mu\text{g}/\text{g}$). EXAMS output for coupling: (1) spatial distribution of dissolved contaminant concentrations (mg/l); (2) spatial distribution of contaminant sorbed to sediment (mg/kg); and (3) contaminant sorbed to biota ($\mu\text{g}/\text{g}$).

Table C.7
COUPLING REQUIREMENTS FOR SERATRA AND TODAM(a)
(SURFACE WATER)

Coupling Model	Spatial Resolution	Time Resolution	Unit Conversion
RAPT (air)	Convert from NMC to Cartesian grid system (50 km to 1 km)	Simple interpolation (month to hour)	Convert deposition ($\mu\text{g}/\text{m}^2$) to loading rate (kg/hr)
STRAM (air)	Convert from NMC to Cartesian grid system (1 km to 1 km)	Simple interpolation (month to hour)	Convert deposition ($\mu\text{g}/\text{m}^2$) to loading rate (kg/hr)
ANDEP (air)	Simple interpolation (1 km to 1 km)	Simple interpolation (month to hour)	Convert deposition ($\mu\text{g}/\text{m}^2$) to loading rate (kg/hr)
ISC (air)	Simple interpolation (1 km to 1 km)	Simple interpolation (month to hour)	Convert deposition ($\mu\text{g}/\text{m}^2$) to loading rate (kg/hr)
ARM (overland)	Simple interpolation (1 km to 1 km)	Simple interpolation (hour to hour)	loading rate (kg/min) to (kg/s) and distribute bulk sediment and particulate contaminant into size fractions
MMT (ground-water)	Simple interpolation (10 km to 1 km)	Simple interpolation (day to hour)	Convert flux from kg/s to kg/hr

(a) SERATRA and TODAM output: (1) sediment concentrations in water column (kg/m^3); (2) dissolved contaminant concentration in water column (kg/m^3); (3) particulate contaminant concentrations in water column (kg/m^3); (4) bed elevation change due to sediment erosion and deposition (m); (5) sediment size distributions in river bed (pct); and (6) particulate contaminant concentrations in river bed (kg/kg). SERATRA and TODAM output for coupling: (1) spatial and temporal distributions of contaminant concentrations (kg/m^3); and (2) spatial and temporal distributions of contaminant sorbed to sediment (kg/kg).

rates for each affected compartment. Water and sediment conveyed by the overland, air, and groundwater transport models will be input into the routing model as a boundary condition, with execution occurring independently from EXAMS in a decoupled mode.

Coupling with Air Models

The atmospheric medium imparts information to the surface water only in the stack and coal pile source scenarios. In general, coupling between the air and surface water models is necessary if one of the following is true:

1. The surface area of the water body is large enough for contaminant deposition to be significant.
2. The power plant in question is situated near the water body.

As discussed before, we selected four atmospheric models for this study: RAPT, ANDEP, STRAM, and ISC. The outputs of these models are similar, typically contaminant mass deposited per unit area at various locations per unit time interval. Although some of these models report wet and dry deposition fractions separately, this is not important to the surface water. The bulk of total deposition is the result that will be passed to the surface water. Total deposition rate is passed to the surface water model.

In the first scenario, where contaminants enter the atmosphere via stacking, the applicable atmospheric codes are RAPT, ANDEP, and STRAM. These three models output the contaminant deposition patterns at user-specified intervals of time in units of $\mu\text{g}/\text{m}^2$. The ANDEP code has an option that allows results to be converted to a Cartesian coordinate system. The largest compatibility problem will occur with the RAPT and STRAM models that have no provision for Cartesian coordinate conversion. These two models generate contaminant depositions on the NMC octagonal northern hemisphere grid. A processing program will have to be used on the RAPT and STRAM outputs to prepare the data for surface water input. Basically, this processing program (described earlier) entails the identification of output grid points in terms of NMC grid points,

conversion to latitude and longitude nodes, and the application of a map scale factor to produce the Cartesian equivalent grid.

Once the results from the atmospheric models are converted to the appropriate coordinate system, the depositional values must be changed to the required input loading rates, using

$$\text{(SERATRA/TODAM)} \quad L_{st} = (1.0 \times 10^{-9})XW/T_{st}$$

$$\text{(EXAMS)} \quad L_e = (1.0 \times 10^{-9})XW/T_e$$

where L_{st} = loading rate for SERATRA/TODAM (kg/s),

L_e = loading rate for EXAMS (kg/hr),

X = deposition rate ($\mu\text{g}/\text{m}^2$),

W = water surface area (m^2)

T_{st} = time increment for SERATRA/TODAM (s),

T_e = time increment for EXAMS (hr).

For the second scenario, where the contaminant source is a coal pile or landfill, the ISC atmospheric model will be used. Like ANDEP, ISC will produce results on a Cartesian grid system. Coupling difficulties between the surface water models and ISC, therefore, are minimal. The contaminant deposition units of the ISC model are g/m^2 . To convert to the loading rate, one should use

$$\text{(SERATRA/TODAM)} \quad L_{st} = (1.0 \times 10^{-3})YW/T_{st}$$

$$\text{(EXAMS)} \quad L_e = (1.0 \times 10^{-3})YW/T_e$$

where L_{st} = loading rate for SERATRA/TODAM (kg/s),

L_e = loading rate for EXAMS (kg/hr),

Y = deposition rate (g/m^2),

W = water surface area (m^2)

T_{st} = time increment for SERATRA/TODAM (s),

T_e = time increment for EXAMS (hr).

Coupling with the Overland Model

In many cases, the overland pathway will make the largest contribution of contaminant to the surface water. Inputs provided by overland modeling include water, sediment, and contaminant contained in the runoff that reaches the surface water.

In three of the four modeling scenarios, the overland component has direct contact with the surface water. For stack releases and coal-pile emissions, overland contaminant transport may furnish information to the surface water models. When the emission source is direct discharge to surface waters, the overland model will receive information from the surface water simulation, if surface water is used to irrigate nearby crops.

ARM provides loading rates for water, sediment, and contaminant at the edge of the water body being modeled. ARM produces a discharge hydrograph in m^3/s , distributed along the water's edge as a lateral inflow boundary condition. When coupling with SERATRA or TODAM, this hydrograph will be used as an input to the hydrodynamic modeling effort, which is performed separately from the contaminant transport modeling.

The loading rates for sediment and contaminant supplied by ARM are bulk rates (not distributed by sediment size fraction) in units of kg/s . Dissolved contaminants are reported as loading (or flux) in kg/min . All values are updated continuously at each time step. In spite of the general compatibility of units (kg-m-s) between the ARM model and the SERATRA and TODAM models, SERATRA and TODAM have requirements that are more detailed than the information received from ARM. A sediment size distribution and a corresponding contaminant distribution must be synthesized from the ARM results using existing analyses of runoff sediment size distributions. Particle diameters are important in calculating sediment transport and the chemical affinity between contaminant and sediment.

To summarize, overland modeling results should be prepared for use in SERATRA and TODAM as follows:

1. Use the discharge hydrograph as lateral inflow to the hydrodynamic model.
2. Distribute bulk sediment loading rates among desired size fractions.
3. Distribute bulk contaminant loading rates among desired size fractions.
4. Use the dissolved contaminant loading as a lateral influx.

EXAMS requires only that the ARM data be averaged to yield steady-state loading rates.

Coupling with Groundwater Models

The groundwater phase of the multimedia simulation is the least dynamic of the various modeling components. This is caused by the strong damping effect provided by porous media flow. The relatively long travel times associated with groundwater often delay the appearance of contamination but do not necessarily attenuate the severity of the problem. Interflow between surface water and groundwater is usually not significant in a surface water modeling time frame. However, the transfer of contaminants between the two media is often important. Therefore, in most cases, only contaminant loadings will be provided from the groundwater model to the surface water model.

The surface water model is directly coupled with the groundwater model in all four modeling scenarios. For stack, coal pile, or ash pond contaminant sources, the groundwater model is expected to furnish contaminant information to the surface water model. The surface water will act as a supplier to the groundwater pathway when the contaminant source is a stack, coal pile, ash pond, or liquid effluent.

A pair of complementary groundwater codes, UNSAT and VTT, have been selected to describe the travel of water through the porous media. UNSAT handles the unsaturated zone from the ground surface to the water table, and VTT models the saturated zone below the water table. All inputs to the surface water model will be from the saturated phase (VTT).

MMT provides dissolved contaminant loading rates in units of kg/s. These loading rates are specified at nodes adjacent to the water body at each time interval. There is thus excellent compatibility with the loadings required by the surface water codes. Coincidence of time and space intervals between media models is not essential, but deviations in the computational grids will require the use of interpolation to couple the models accurately.

Appendix D

POWER PLANT EFFLUENT STREAMS

CONDENSER COOLING SYSTEM

During plant operation, steam leaving the final turbine is cooled and condensed to be returned to the boiler. The condenser cooling water system must be able to remove large quantities of heat from this steam. Cooling water systems may be either once-through or recirculating. Once-through systems are used where large quantities of high quality water are available. Recirculating systems are more appropriate where water is more expensive or thermal discharges must be controlled.

In once-through systems the total cooling water flow is withdrawn from the source, passed through the condenser system once, then discharged as effluent to the receiving waters. About 65 percent of existing steam electric power plants use once-through cooling systems (EPA, 1980a). Approximate flow rates can be calculated assuming 0.1 liter of cooling water per kilocalorie of heat removed for each 10°C increase in cooling water temperature. This corresponds to 12 gallons per thousand Btu removed per 10°F.

The chemical composition of the effluent from once-through systems is similar to that of the intake water. Generally, the only necessary water treatment is the addition of biocides to control growth of algae and slime on condenser surfaces. Most plants use chlorine or sodium hypochlorite as biocides. These chemicals contain no priority pollutants (Colley et al., 1978), although chlorine discharges must be controlled (EPA, 1982). In some plants, waterside condenser corrosion may be a problem, although it should be minimized by proper design. When it does occur, corrosion inhibitors may be added to the cooling water. Some of the potential inhibitors contain priority pollutants (EPA, 1980a). In general, corrosion and its control chemicals can contribute chromium, copper, zinc, and other metals to cooling system effluents.

In recirculating systems the cooling water passes through the condenser several times. After each pass, the water is sent to a spray pond, canal, or cooling tower where heat is removed through evaporative cooling. These evaporative losses concentrate dissolved nonvolatile pollutants and suspended material in the remaining water. Water is also lost to recirculating systems through drift--the entrainment of water droplets in the cooling tower air stream--but this process does not increase pollutant concentrations.

The quality of recirculating cooling water must be kept high, to prevent biofouling, corrosion, and the formation of scale deposits in the condenser. The concentration of dissolved and suspended materials in the cooling water can be controlled by withdrawing a blowdown stream and replacing this loss (as well as evaporation and drift losses) with better quality makeup water. The quantity of blowdown is set by the maximum concentration of limiting impurities that can be tolerated in the system, or by the solubility limit of scaling salts. Typically blowdown rates are between 0.5 and 3.0 percent of total cooling water flow (Colley et al., 1978). Depending on the plant, blowdown may be (1) discharged directly to surface waters or publicly owned treatment works, (2) used in other plant processes, or (3) treated within the plant before discharge or reuse.

In typical power plants, cooling tower blowdown, drift, evaporation, and the number of cycles of concentration in the system are related by the following expression (EPA, 1974):

$$c = (b + v + d)/(b + d) \quad (D.1)$$

where c = cycles of concentration (dimensionless),

b = blowdown rate (percent),

v = evaporation rate (percent),

d = drift rate (percent).

Normally, drift rates vary from 0.002 percent for modern natural draft cooling towers to 0.2 percent for old cooling towers, with a typical value of 0.005 percent for modern mechanical draft towers (Rice

and Strauss, 1977). We can assume evaporation losses to be about 1.0 percent per 10°F temperature change in the cooling water (CH2M, 1980; EPA, 1974). For a plant operating nominally at five cycles of concentration and a 20°F temperature drop, this gives a blowdown rate of approximately 0.5 percent of the flow rate. Under these conditions, blowdown represents about 20 percent of the makeup water flow. This is typical for fresh water systems, although blowdown can be reduced to as little as 3 to 5 percent of makeup flow by means of sidestream treatment--continuously treating a fraction of the recirculating water flow to remove deposit-forming substances (Rice and Strauss, 1977).

The quality of blowdown discharges from a power plant will be the quality of the circulating cooling water. As described by Colley et al. (1978), it will be influenced by design factors and environmental conditions, including:

- Makeup water quality.
- Number of cycles of concentration.
- Air-water contact in the cooling device.
- Design of condenser cooling system.
- Chemical treatment of recirculating cooling water.

Pollutants entering the system in the makeup water will be concentrated in the circulating stream and discharged in the blowdown. Air-water contact within a cooling tower may either add or remove particulates and soluble gases from the water, depending on local air quality and other conditions. Moreover, in power plants using cooling towers, cooling waters can leach preservatives from the wooden tower structures or asbestos from tower fill material (EPA, 1980a). Priority pollutants commonly used in the preservatives include copper, chromium, arsenic, and phenols (Colley et al., 1978).

The final source of priority pollutants is the chemicals used to treat recirculating cooling water to control corrosion, scaling, and biological fouling of condenser surfaces. Corrosion and scale control chemicals can include inhibitors, dispersing agents, and pH control compounds (Hart and DeLaney, 1978). Some priority pollutants, such as

chromium and zinc, may be found in these chemicals. Although chlorine and related oxidizing biocides are most frequently used in power plants, other priority pollutants may be found in some commonly used nonoxidizing biocides, including copper, chromium, arsenic, phenol, and zinc (EPA, 1980a). All of these pollutants including chlorine may appear in the blowdown, as will corrosion products such as copper, which is extensively used in condenser tubes.

ASH HANDLING AND DISPOSAL

Coal-fired power plants produce large quantities of fly ash and bottom ash that must be handled within the plant and removed to treatment and disposal facilities. Ash collection and disposal schemes are basically of two types--wet and dry--although many variations exist within the utility industry. Completely dry systems are pneumatic, vacuum, or pressure, and produce no wastewater. Wet handling systems can produce waste effluent of varying quantity and quality, depending on the coal characteristics and plant design.

Fly ash can be collected in a dry state from cyclones, fabric filters, or dry electrostatic precipitators, or in a wet state from wet scrubbers or wet electrostatic precipitators. If collected and handled dry, it is pneumatically conveyed to a dry storage silo where it accumulates for a few days. Subsequently it may be wetted to 10 or 20 percent moisture content (to minimize dust) and transported to a landfill for disposal. If collected and handled wet, it will be mixed with water to 5 to 10 percent solids by weight and sluiced to a dewatering system, normally a settling pond. This sluicing and settling may be done independently or in a combined fly ash and bottom ash system. The settling ponds can be dredged, with the ash removed to a landfill for final disposal, or filled and used as a final disposal site themselves (CH2M, 1980).

Bottom ash is normally collected in a quench pit under the furnace. In dry handling systems, the ash is dewatered and conveyed to a storage bin for subsequent sale or disposal in a landfill. In wet handling systems, the ash is ground or crushed, mixed with water, and sluiced to a dewatering system. This may be a dewatering bin or settling pond,

where the ash can be combined with fly ash for settling and ultimate disposal.

Bottom ash consists generally of fused clinker-type material which is a vitreous combination of silica and metal oxides. It settles well and has low solubility, so the sluice water in a properly designed system can be relatively free of suspended solids. Fly ash, on the other hand, settles poorly but adequately, if given enough time. It consists of fine particles, some hollow, and contains many soluble inorganic salts that appear in the sluice water.

Wet handling systems can produce one of two types of wastewater. This waste effluent may be overflow--direct discharge of once-through sluice water--or blowdown from a recirculating system. The quantity and quality of the effluent is a function of many influences (Teknekron, 1976; Colley et al., 1978), including:

- Coal ash content and characteristics.
- Intake water quality.
- Distribution of bottom ash and fly ash.
- Boiler furnace size and design.
- Air pollution control equipment.
- Sluice water requirements.
- Type of long-term disposal.
- Design of the ash handling system.
- Other wastewater combined in the system.

Bottom ash systems normally require minimum flows of from 10 to 18 tons of water per ton of ash, but may use up to 165 tons/ton (Colley et al., 1978). For a nominal 1 percent solids concentration, 109 tons/ton is required. Fly ash systems require minimum flows of from 5 to 12.5 tons/ton but can also use up to 165 tons/ton. Here, a nominal 5 percent concentration would require about 21 tons/ton of ash.

The effluent from wet ash handling systems will contain pollutants from the intake or makeup water and dissolved and suspended materials from the coal ash. The range of priority pollutant concentrations and

typical values for the concentrations are shown in Table D.1 for the discharges from the three types of ash ponds.

MAINTENANCE CLEANING WASTES

To maintain plant performance and equipment reliability and life, plant operators must periodically clean the heat transfer surfaces in (1) boilers, (2) steam generators, (3) feedwater heaters, (4) condenser systems, and (5) air preheaters. Additionally, such cleaning is also required for stacks, cooling tower basins, and auxiliary plant equipment. Cleaning removes scale, oil, grease, and metal oxides and other corrosion deposits (Teknekron, 1976; Hart and DeLaney, 1978; Babcock and Wilcox, 1978). Cleaning procedures, solutions, and frequency depend on power plant equipment and design, raw materials, and

Table D.1
PRIORITY POLLUTANT CONCENTRATIONS IN ASH POND DISCHARGES
(ppm)

Substance	Fly Ash Pond			Bottom Ash Pond			Combined Ash Pond		
	Min.	Typical	Max.	Min.	Typical	Max.	Min.	Typical	Max.
Antimony	0.005	0.006	0.006	0.012	0.012	0.012	0.002	0.015	0.029
Arsenic	0.005	0.100	1.100	0.002	0.015	0.070	0.004	0.050	0.416
Beryllium	0.001	0.010	0.020	0.001	0.002	0.002	{a}	{a}	0.020
Cadmium	0.004	0.015	0.052	0.002	0.010	0.016	0.000	0.015	0.082
Chromium	0.005	0.025	0.170	0.004	0.015	0.042	0.003	0.020	0.084
Copper	0.020	0.165	2.400	0.005	0.035	0.200	0.000	0.050	0.730
Cyanide	{a}	{a}	0.022	{a}	{a}	{a}	0.010	0.025	0.080
Lead	0.003	0.125	0.910	0.005	0.025	0.060	0.000	0.030	0.100
Mercury	0.000	0.001	0.006	0.000	0.001	0.004	0.000	0.002	0.065
Nickel	0.033	0.050	0.130	0.013	0.100	1.345	0.000	0.050	0.190
Phenol	0.004	0.025	0.040	0.011	0.011	0.011	0.001	0.025	0.130
Selenium	0.001	0.050	0.330	0.001	0.005	0.056	0.002	0.020	0.170
Silver	0.003	0.005	0.006	0.004	0.010	0.019	0.001	0.010	0.052
Thallium	--	--	--	--	--	--	0.008	0.008	0.008
Vanadium	0.010	0.018	0.027	0.005	0.005	0.005	0.010	0.020	0.043
Zinc	0.050	0.500	2.700	0.010	0.100	0.550	0.010	0.075	0.293

SOURCES: EPA (1980a); Hart and DeLaney (1978); Colley et al. (1978); Rice and Strauss (1977); Bahor et al. (1981).
{a} Less than 0.010.

intake water (Teknekron, 1976). In particular, cleaning frequency and procedures depend on the following:

- Reliability requirements.
- Plant materials.
- Chemical additives.
- Mode of operation.
- Costs of cleaning.
- Allowable performance degradation.
- Permissible cleaning chemicals.
- Compliance with water quality regulations.

The quality of cleaning effluents will be affected by the chemicals used in cleaning and the materials removed from cleaned surfaces.

The primary effluent sources are cleaning of boiler tubes, boiler fireside surfaces, and air preheater. The other cleaning operations mentioned above should be secondary sources of water pollution. For these secondary sources, cleaning is done generally with high pressure water sprays, detergents and wetting agents, or hydrochloric acid (HCl) solutions, depending on the specific equipment. Data are limited on the quantity and quality of effluent from these sources. The volumes are normally small, and cleaning operations are relatively infrequent, although some metals may be present in the discharges (Hart and DeLaney, 1978).

The waterside of steam generators (boiler tubes and often feedwater heaters) must be periodically cleaned with chemicals, the frequency depending on boiler characteristics and operating conditions. This cleaning removes scale and corrosion products and typically occurs every 3 to 5 years for large boilers, although the interval can vary between 7 and 100 months (Colley et al., 1978; Hart and DeLaney, 1978).

Waste effluent from this process consists of the cleaning and rinse solutions. The volume depends on the fill volume of the boiler and the specific cleaning process. Boiler volumes typically range from 20 to 100 thousand gallons (Rice and Strauss, 1977). Cleaning may be done using soaking or circulating processes, and may involve from one to five

boiler volumes, including the cleaning solution, rinses, and neutralizing treatments. As discussed in EPA (1980a) and Colley et al. (1978), solutions may be acid or alkaline with chelating or complexing agents, and may include the following:

- HCl with or without copper complexing agents.
- Ammoniated EDTA.
- Ammonium bromates solutions.
- Organic solvents and proprietary processes.
- Hydroxyacetic/formic acid solutions.

Acid cleaning solutions will frequently be followed by alkaline neutralizing and passivating rinses.

The effluent of boiler fireside cleaning consists of cleaning agents and rinses and various boiler metals. These metals include chromium, copper, nickel, zinc, cadmium, and arsenic, many of which may be in complexes or chelates, so difficult to remove through treatment processes. Because these wastes are intermittent, they are frequently stored for equalization, neutralization, and later release.

Boiler firesides are normally cleaned by spraying high-pressure alkaline solutions against boiler tubes while they are still hot (EPA, 1980a). This cleaning should be done from 2 to 12 times per year depending on the specific plant maintenance problems. Typical effluent volumes range from 90 to 2725 thousand liters per cleaning. Priority pollutants in the cleaning effluent include boiler metals, coal contaminants, and additives to reduce ash corrosion of boiler surfaces. Contaminants often found are chromium, nickel, vanadium, and zinc.

Air preheaters are normally cleaned anywhere from weekly to semiannually to remove soot, fly ash, rust, and other corrosion deposits. As with boiler firesides, preheaters are generally cleaned with high-pressure sprays. The effluent may include copper, nickel, and chromium, and zinc from corrosion products.

Various studies have tried to estimate flow rates for many of the effluent streams discussed above. These are summarized in Table D.2.

Table D.2

TYPICAL VOLUMES OF LOW-VOLUME WASTES
(1/day/MWe)

Effluent Stream	Waste Volume
Boiler cleaning	4-15
Boiler fireside	11-19
Air preheater	55-380
Boiler blowdown	379-560

SOURCES: EPA (1980a); Hart and DeLaney (1978);
Rice and Strauss (1977).

BOILER BLOWDOWN

Coal-fired power plants use either once-through or drum-type boiler designs. Most once-through boilers, found in high-pressure, supercritical systems, produce little or no wastewater. Drum-type boilers, however, operate at subcritical conditions where the steam is in equilibrium with the boiler water. As a consequence, impurities concentrate in the water, and a boiler blowdown stream is necessary to control the concentrations of dissolved and suspended solids (Colley et al., 1978).

The quality of boiler blowdown depends on the age, size, and design of the boiler, the quality of makeup water, and chemicals additives to the system. Chemicals may be added to prevent corrosion and the formation of scale. These chemicals include oxygen scavenging additives, pH control compounds, and dispersing agents. Some of the chemicals may contain priority pollutants such as chromium, copper, phenol, or EDTA (EPA, 1980a). These materials may appear in the waste effluent, with corrosion products such as copper, nickel, and zinc. In general, however, blowdown has very high quality and may be suitable for internal reuse in other plant water systems (EPA, 1980a).

Boiler blowdown may be discharged either continuously or intermittently. Flow rates vary significantly but generally range from 0 to 2 percent of the steam generation rate (Colley et al., 1978). Typical rates for modern high-pressure boilers are about 0.1 percent,

but blowdown rates as high as 10 to 30 percent can be found in low-pressure systems without demineralization of makeup water (Hart and DeLaney, 1978; Colley et al., 1978). Sidestream treatment can be used for a fraction of the normal boiler blowdown. Using this fraction as makeup water may be economical, because it means having to treat a smaller flow volume with higher pollutant concentrations. Any of the processes described below may be used in this sidestream treatment.

WATER TREATMENT WASTES

Power plants normally may treat boiler feedwater, condenser cooling water, other makeup water supplies, and regenerative or cleaning waters to remove undesirable materials. These materials include suspended and dissolved solids, and dissolved gases. In addition to adding chemicals to prevent scaling, corrosion, foaming, priming, caustic embrittlement, and microbial fouling, many types of treatment techniques and processes can be used (Hart and DeLaney, 1978). Selection of specific processes will depend on (1) raw water supplies, (2) plant equipment, (3) operating processes, (4) waste disposal techniques and requirements, and (5) plant economics. The available and commonly used techniques include:

- Screening.
- Sedimentation.
- Clarification.
- Filtration.
- Lime softening.
- Ion exchange.
- Reverse osmosis.
- Electrodialysis.
- Distillation or evaporation.

These processes can be used alone or in combination, depending on plant requirements. Typical treatment might include screening, alum flocculation and clarification, and anthracite filtration. This could be followed optionally by demineralization (with ion exchange, reverse

osmosis, or electrodialysis) and treatment with biocides or corrosion depend on the specific techniques used as well as the raw water inhibitors (Teknekron, 1976). The wastes from water treatment processes characteristics and the extent of treatment required.

Screening and sedimentation are techniques that usually precede other processes. Screening physically removes bulk solids from the intake water. Sedimentation uses gravity separation to remove the larger fractions of suspended organic and inorganic materials. Neither of these processes generates priority pollutants in their effluent other than those present in the suspended material in the influent.

In clarification processes, a coagulant (such as alum or copperas) and coagulant aids may be added to the influent to promote agglomeration of suspended particles. These larger particles are then removed in settling tanks. The clarified water may then be filtered and used in the power plant or else passed to another treatment stage. The settled solids are generally concentrated or dewatered (sometimes in plant ash ponds) and taken to disposal sites. Commonly used clarification chemicals do not contain any priority pollutants and do not add to the pollutants removed from the intake water in the suspended materials.

Filtration processes are also used to remove suspended materials from water, frequently in conjunction with other treatment processes. It commonly follows clarification and lime softening operations to remove residual fine particles from the process effluents. It may also be used to precede some processes (such as ion exchange, reverse osmosis, electrodialysis, and distillation) to remove suspended materials that might otherwise interfere with process efficiency. Filtration media include silica, crushed anthracite, activated carbon, and other materials. The waste discharge from filtration processes comes from the backwash step used to remove the accumulated solids from the filter media. This effluent may be concentrated as described above before discharge and disposal.

Intake water is commonly softened to remove calcium and magnesium ions that can cause scaling inside power plant boilers. These materials are removed by adding calcium hydroxide and sodium carbonate at ambient or elevated temperatures. Softening produces precipitates of calcium

carbonate and magnesium hydroxide but does not add priority pollutants to the intake water.

Ion exchange processes are used to soften or demineralize water supplies used for condenser cooling systems, boiler feedwater, and other plant processes. Because of its flexibility and reliability, ion exchange is the most common technique for treating supply water. Ion exchange resins can be either cationic or anionic, depending on whether they are designed to remove positive or negative ions. The exchange material is generally a natural or manufactured organic resin in bead form.

Intake water is passed through a bed of the resin until all exchange sites are saturated and adsorption decreases. At this time the resin must be regenerated. Regeneration is a three-step process, consisting of (1) backwashing to expand the bed and remove solids, (2) removal of ions from the resin with a regenerant solution, and (3) rinsing excess regenerant solution and ions from the bed. All three steps contribute to the waste effluent from ion exchange operations. Regeneration frequency depends on the influent quality, resin characteristics, and the treatment cycle, but normally falls between daily and weekly.

Backwashing is normally done with raw influent to the process but does not add any significant water pollution. Regeneration or elution of collected ions is done with either acid or basic solution, depending on whether the resin is cationic or anionic. The effluent from this step contains the excess regenerant solution and the ions collected by the resin. Rinsing removes the remaining regenerant solution from the resin bed. The discharged rinse water can vary in quality between spent regenerant solution and treated water (Colley et al., 1978).

Wastes from ion exchange regeneration processes are frequently collected in equalization basins and neutralized with chemicals or other nonprocess wastes before treatment (EPA, 1980a). Treated wastes may be used as makeup water for other plant processes, such as ash transport. The untreated wastewaters will contain the priority pollutants in the influent water, either intake water or wastes from other plant processes. For cationic resins, the trace metals will frequently be present. Regeneration processes should not add priority pollutants.

Reverse osmosis and electrodialysis are demineralization processes that use semipermeable membranes to remove dissolved salts from intake water. These processes create a concentrated brine and a relatively pure solution using either pressure or electric fields to perform the separation. They are always used in conjunction with demineralizers and frequently with clarification, filtration, and ion exchange softening. The waste effluent from reverse osmosis and electrodialysis processes is the brine, essentially the raw influent water with higher concentrations of dissolved solids. The separation processes do not add priority pollutants to the wastewaters produced.

Distillation and evaporation processes remove dissolved and suspended solids by evaporating and subsequently condensing part of the influent. The processes are sometimes used to demineralize boiler feedwater. Impurities are concentrated in the influent remaining in the evaporator. The waste effluent is a blowdown stream for controlling impurity concentrations in the evaporator solution. Pollutant concentrations in the wastewater depend on intake concentrations and the scaling potential of the influent. Typical blowdown rates range from 10 to 40 percent of the influent rate (Colley et al., 1978).

Evaporator brine is normally alkaline but can be neutralized and used for other plant purposes not requiring low dissolved solids concentrations (Hart and DeLaney, 1978). Priority pollutants present in the influent will be concentrated in the effluent, but the process should add no pollutants.

Treatment processes can be used either on raw intake water or on plant boiler blowdown. Using this process--sidestream treatment--a power plant can treat a smaller volume of water with higher pollutant concentrations. This reduces treatment difficulty and costs but does have some problems. It can lead to increased concentrations of some priority pollutants, such as copper and zinc, and those found in chemical additives used in plant water systems.

Typical discharge rates for some of the treatment processes described above can be found in Table D.3.

Table D.3

TYPICAL DISCHARGE RATES FOR WATER TREATMENT PROCESSES
(gpd/MWe)

Process	Flow
Clarification	65
Ion exchange	79
Filtration	71
Lime softening	56
Evaporation	126
Reverse osmosis	31
Typical process	40-80

SOURCES: Teknekron (1976); EPA (1980a).

COAL-PILE AND PLANT DRAINAGE

Coal-fired power plants maintain two types of coal storage, live and reserve. Live or active storage contains sufficient coal to fuel the plant between coal deliveries. Reserve or inactive storage insures a coal supply if scheduled deliveries are interrupted. Precipitation runoff and drainage from these storage piles may generate an effluent containing some of the priority pollutants.

The size and characteristics of storage piles depend on the generating capacity and design of the power plant and the moisture and heating value of the coal (Hart and DeLaney, 1978). The size of active storage piles will depend on the arrival frequency of coal shipments. They are normally kept as small as possible, to minimize oxidation losses and the possibility of spontaneous combustion. Reserve storage piles generally contain a 90 to 100 day supply and are compacted to eliminate air spaces and minimize oxidation (Dvorak et al., 1978; Hart and DeLaney, 1978). Reserve or inactive coal piles may often be sealed with tar or other impervious spray for protection from weather (Colley et al., 1978).

Runoff and percolation of precipitation through unprotected storage piles can leach pollutants from the coal. The discharge can closely resemble acid mine drainage, containing sulfuric and humic acids,

dissolved solids, and suspended materials. According to a recent study reported by the EPA (1980a), the quantity of runoff can be predicted by

$$R = 0.855F + 0.0082 \quad (D.2)$$

where R = runoff (acre-in./acre),

F = precipitation (in.).

Discharge quality will be influenced by many factors, particularly (1) rainfall volume, (2) coal characteristics (such as pyritic sulfur content and alkalinity), (3) coal preparation and cleanliness, (3) storage pile dimensions and design, (4) pollution and dust controls, and (5) rainfall residence time within the coal pile. The precipitation percolating through the pile reacts with atmospheric oxygen and pyritic sulfur in the coal to produce sulfuric acid. The acid, if not neutralized by natural coal alkalinity, can dissolve inorganic salts in the coal (Teknekron, 1976; Hart and DeLaney, 1978; Colley et al., 1978). This process can generate effluent containing dissolved and suspended priority pollutants such as arsenic, beryllium, cadmium, chromium, copper, lead, mercury, nickel, selenium, silver, vanadium, and zinc.

Chemical characteristics of coal pile drainage will vary over the duration of a rainfall event, as pollutants are flushed out of the pile. Before flushing is complete, the concentration of pollutants will vary inversely with the runoff flow rate. After flushing is complete, the concentration of pollutants is independent of the flow rate. The time required to complete flushing depends on the precipitation rate and storm duration (EPA, 1980a). Typical concentrations of trace elements in coal pile drainage are shown in Table D.4.

Plant drainage consists of precipitation runoff from general plant floor and yard drains. It will generally contain suspended and dissolved solids in highly variable concentrations, depending on plant conditions and local climate. Priority pollutants found in this effluent will be those coming from coal dusts and fines, fly ash, and local soil, among other sources. Virtually no data are available to characterize this runoff, but it is unlikely to be an important source

Table D.4
COMPOSITION OF COAL-PILE DRAINAGE
(mg/l)

Constituent	Concentration		
	Minimum	Typical	Maximum
Arsenic	0.005	0.100	0.600
Beryllium	<0.010	0.025	0.070
Cadmium	<0.001	0.002	0.006
Chromium	0.000	0.100	15.700
Copper	0.010	1.000	6.100
Lead	<0.010	0.020	0.200
Mercury	0.0002	0.002	0.027
Nickel	0.200	1.500	4.500
Selenium	<0.001	0.003	0.030
Zinc	0.006	2.500	16.000

SOURCES: DOE (1979); Braunstein et al. (1977);
EPA (1980a); Hart and DeLaney (1978); Colley et al. (1978).

of pollution. Typical flow rates could be about 10 gallons per day per megawatt of capacity (Teknekron, 1976).

PROCESS SPILLS AND LEAKS

These discharges may be associated with many plant problems, such as overfilling, tank or pipe leaks and ruptures, valve and seal failures, and accidental spills. The characteristics of the discharge will depend on the particular materials involved. These are likely to include regenerant solutions for ion exchange processes, water and waste treatment chemicals, oils, and plant makeup and process waters. Again, it is unlikely that this would be an important source of priority pollutant discharges.

AIR POLLUTION CONTROL SYSTEM WASTES

Processes for removing sulfur dioxide from flue gas streams generally use wet scrubbing with alkaline slurries to absorb and react with the sulfur compounds in exiting gases. These processes can be classified either as nonregenerable (throwaway) or regenerable,

depending on whether they produce a solid waste stream for disposal, or a marketable sulfur product and reusable sorbent.

The throwaway processes have lower capital costs and are relatively simple, using readily available sorbents. However, they generate large volumes of waste (including trace elements) that must be treated and sent to disposal sites. The most common nonregenerable processes include (1) lime, (2) limestone, (3) double alkali, and (4) lime-alkaline fly ash scrubbing. In these processes, the flue gas may first be treated to remove particulates, using either an ESP or baghouse. The gas is then passed through the absorbing system, where sulfur dioxide is removed by absorption into a circulating slurry. The effluent from the absorber is sent to a reaction tank or crystallizer where sulfite and sulfate solids can be precipitated. Material from the reaction tank is sent either to a pond or solid/liquid separator. The sludge from the separator may be further dewatered before being sent to a pond or landfill for disposal. Liquid effluent from the separator returns to the slurry cycle (CH2M, 1980; Knight et al., 1980; EPA, 1980a).

Although these systems may be designed to maximize water recirculation, there may be discharges to surface water bodies. Holding tanks for dewatering the sludge may have bleed streams. Moreover, these systems often need blowdown streams to control buildup of dissolved solids, particularly soluble sodium sulfate concentrations in double alkali scrubbers. Finally, the EPA has found that many power plants do not completely recirculate FGD water but rather discharge some disposal pond overflow (EPA, 1980a). Typical flow rates for this system blowdown and pond overflows are 811 gpd/MW and 3,973 gpd/MW (EPA, 1980a).

Another source of wastewater in these systems is mist eliminator wash. Demisters are used in scrubbers to catch entrained scrubber solution in exiting flue gas. The demister vanes must be washed periodically to remove accumulated solids. Wash water from this operation can be directed either to the thickener, recycle tank, or disposal pond in the system.

In regenerable processes, the sorbent is regenerated and reused. The sulfur dioxide is converted to hydrogen sulfide or further processed

into elemental sulfur, sulfuric acid or concentrated sulfur dioxide gas (CH2M, 1980). The most common processes are the Wellman-Lord and magnesium oxide scrubbing processes. Although these systems optimally recirculate all liquids, they do have some liquid effluents. These waste streams include both intermittent and continuous discharges.

Both regenerable processes must pass the flue gas through a prescrubber. This prescrubber is needed to cool and humidify the gases and to remove residual fly ash and chlorides. This process prevents excessive evaporation in the absorber and reduces the potential for scaling and corrosion within the system (CH2M, 1980). The prescrubber will have a blowdown stream that must be treated and discharged. Also, the regeneration loop in the Wellman-Lord process requires a condenser, which will also have a blowdown stream.

These effluent streams and those discussed previously for the throwaway processes may be joined by intermitted discharges from all processes. These intermittent discharges will include bleed streams from the magnesium oxide and double alkali processes and purge streams (during process upset) from the lime and limestone scrubbing systems.

Wastewater from both regenerable and nonregenerable systems could contain up to 50 of the priority pollutants (EPA, 1980a). In general, however, the waste composition will depend on the FGD process, fly ash collection system, composition of plant coal and raw materials, and the specific stream under analysis. Prescrubber blowdown will be high in suspended solids and chloride concentration, with priority pollutants leached from the collected fly ash. In most respects, except for the chloride concentration, it should be similar to ash transport water and can be dealt with in a similar manner. Cooling system blowdown in the Wellman-Lord process will be similar to, and can be treated as, the blowdown from other cooling systems within the power plant. The purge streams from other processes would vary in content, depending on the process, but would in general contain high levels of dissolved salts and possibly significant concentrations of many trace elements. Finally, overflow from sludge disposal ponds could contain significant amounts of priority pollutants, particularly the trace metals. Typical concentration ranges for these metals are shown in Table D.5.

Table D.5

TRACE ELEMENT CONCENTRATIONS IN FGD SLUDGE POND OVERFLOW
(mg/l)

Element	Liquor Concentration		
	Minimum	Maximum	Typical
Antimony	0.090	2.90	0.400
Arsenic	0.004	1.80	0.001
Beryllium	0.001	0.18	0.013
Cadmium	0.002	0.11	0.030
Chromium	0.001	0.50	0.060
Copper	0.002	0.56	0.100
Lead	0.001	0.52	0.050
Mercury	0.0004	0.07	0.005
Nickel	0.005	1.50	0.100
Selenium	0.0006	2.70	0.120
Silver	0.005	0.60	{a}
Vanadium	0.001	0.67	{a}
Zinc	0.005	1.50	0.100

SOURCES: CH2M (1980); Knight et al. (1980).

{a} Insufficient information.

MISCELLANEOUS WASTE STREAMS

The remaining miscellaneous waste streams vary considerably in importance between plants. These streams vary in composition and volume but can contain priority pollutants. They include:

- Laboratory and sampling streams.
- Auxiliary cooling water systems.
- Water intake screen backwashing.
- Sanitary wastes.
- Floor drains.
- Construction wastes.

Flow and concentration data are limited for these streams, except for auxiliary cooling systems. In general, auxiliary cooling water blow-down or discharges are similar to primary cooling water discharges. Discharge rates for some miscellaneous streams are shown in Table D.6.

Table D.6

DISCHARGE RATES FOR MISCELLANEOUS WASTE STREAMS
(gpd/MWe)

Waste Stream	Discharge Rates
Auxiliary cooling (once-through)	750 - 50,000{a}
Auxiliary cooling (blowdown)	0 - 5
Floor drains	10 - 30
Laboratory streams	10
Total miscellaneous streams	0 - 70

SOURCES: Teknekron (1976); Colley et al. (1978); Rice and Strauss (1977).

{a} A typical value for this stream would be 15,000 gpd/MWe.

Appendix E

WASTEWATER TREATMENT AND CONTROL PROCESSES

A number of wastewater treatment processes and plant modifications have been suggested as ways of controlling priority pollutant emissions from coal-fired power plants. In this appendix we briefly discuss these processes and their application, emphasizing the waste streams for which they may be appropriate.

INCINERATION

As applied to the electric utility industry, incineration is defined as the controlled injection of wastewaters into the firebox of an operational power plant boiler. Organic materials in the waste are combusted as the injected water vaporizes. Metals in the waste stream are converted to oxides and either deposit in the bottom ash or leave with the flue gas stream. Injection rates are variable, depending on the fan and fuel capacity of the boiler, but typically range from 20 to 180 gallons per minute (EPA, 1980a).

This process has been used for disposal of spent boiler waterside cleaning wastes, particularly ammoniated EDTA solutions. Hydrochloric acid cleaning wastes can also be incinerated, if they are first neutralized and injection rates are carefully monitored (Rice and Strauss, 1977). No cost information for this process has been located, but we expect costs would be low in comparison with other methods of disposal of such wastes.

SUBSTITUTION OF ALTERNATIVE CHEMICALS

As discussed in Chap. 3, power plants generally use various combinations of chemicals to control biofouling, scale formation, and corrosion in cooling water, boiler water, and other miscellaneous systems. Many of these chemicals contain priority pollutants that may

appear in the plant waste discharges. An obvious method of controlling these pollutants is to avoid using chemicals that contain them. According to the EPA (1980a), in most cases alternative biofouling, scale, and corrosion inhibition chemicals can be obtained at similar cost. In its specification of best available technology economically achievable, new source performance standards, and pretreatment standards for new and existing sources, the EPA has prohibited discharge in cooling tower blowdown of priority pollutants resulting from such additives.

REVERSE OSMOSIS

Reverse osmosis uses pressure to drive water through a semipermeable membrane to remove dissolved solids and other impurities. The process thus produces a deionized product stream and a waste brine stream. Membranes are made of inorganic and synthetic organic materials, primarily cellulose acetate and nylon aromatic polyamide. Membranes are classified in terms of their maximum water transfer rates and salt rejection abilities. Flow rates are measured in m^3/day per m^2 of membrane area. At typical rates of 0.0035 to 0.0088 $\text{m}^3/\text{day}/\text{m}^2$, reverse osmosis units can achieve 90 to 98 percent rejection of salt when operating on wastewater with total dissolved solids (TDS) of 5000 mg/l (Colley et al., 1978).

The membranes are subject to fouling from biological growths, suspended solids, scaling, organic materials, and iron or manganese. As a consequence, extensive pretreatment of feedwater may be necessary. In typical processes, feedwater may undergo pH adjustment, lime softening, filtration, and scale removal, depending on feedwater quality. During operation, membranes are periodically backflushed to remove plugging or fouling materials.

Reverse osmosis generally provides its 90 to 98 percent salt rejection with a water recovery rate of about 75 percent. The 25 percent brine stream is often combined with the backflush water and sent to a disposal pond or for further treatment. Multiple-stage units can achieve much higher salt removal rates at additional cost. Metallic priority pollutants are normally reduced at the same rate as dissolved solids. Organic and colloidal concentrations can be reduced by up to 95 percent (Colley et al., 1978).

This process could theoretically be applied to all utility waste streams, except cleaning water (because of pH) and laboratory drains (because of organic materials). In FGD systems, it could be used on prescrubbed blowdown, cooling tower blowdown, and various purge streams (CH2M, 1980). However, because of its high volume waste stream, it would most likely be used as a pretreatment step to be used in conjunction with other more expensive processes. Reverse osmosis units are commercially available from a number of companies and have been used worldwide for desalting domestic water supplies. They have seen only limited use in industrial wastewater treatment, and then only at small scale (less than 180,000 gallons per day) (CH2M, 1980). Performance data in power plant applications are limited to bench-scale tests (EPA, 1981), and reverse osmosis has not been demonstrated in the electric utility or closely related industry (UWAG, 1982).

Costs of reverse osmosis treatment are influenced primarily by flow rates, membrane replacement costs, and the number of stages used. Capital and operating costs for this process can be found in Colley et al. (1978).

CHEMICAL PRECIPITATION

Chemical precipitation treatment of wastewater removes soluble metallic ions and other undesirable anions by converting them to insoluble forms. This is accomplished by altering the ionic equilibrium to produce insoluble precipitates that can subsequently be removed by a solids separation process. These separation procedures may include coagulation, sedimentation, or filtration (EPA, 1981).

A number of alternative processes have been used in industry, but only hydroxide precipitation (lime-soda softening) has been practiced by electric utilities. Lime (as CaO or Ca(OH)_2) addition to wastewaters raises the pH, causing calcium, magnesium, and many heavy metal species to precipitate out of the solution as hydroxides. Power plants frequently use lime-soda softening to (1) treat metal cleaning wastes, (2) remove scale-forming species in sidestream treatment of recirculating ash transport or cooling system waters, and (3) remove dissolved and suspended materials from feedwater supplies before using reverse osmosis or ion exchange.

In general, lime-soda softening consists of the following stages:

- Lime addition and mixing to adjust pH.
- Flocculation and clarification (with coagulant aids as necessary).
- Deep bed filtration.
- Acid addition and mixing to adjust pH.

The sludge from the clarifier may be sent to a settling pond or thickened and dewatered for disposal in a landfill. Other steps may be incorporated during the process, depending on wastewater characteristics. These include (1) initial mixing with other wastes for equalization and neutralization, (2) oxidation or aeration, (3) addition of sulfides and coagulants, and (4) final activated carbon adsorption. It has been reported (CH2M, 1980) that lime-soda sidestream softening is most effective when the lime and soda ash are added in separate sequential clarifiers, followed by water conditioning and filtration.

Lime precipitation can remove many toxic materials, including arsenic, cadmium, trivalent chromium, copper, cyanide, lead, nickel, silver, and zinc. Removal efficiencies vary significantly with pollutant and wastewater characteristics (CH2M, 1980; EPA, 1981), and can be reduced, for instance, by various complex processes associated with organic material concentrations.

Lime-soda treatment has long been in use for commercial water treatment and municipal and industrial wastewater treatment. However, lime treatment has not been used extensively for wastewater treatment in power plants. Although the EPA has suggested that this process could be used to treat fly ash transport wastewaters (EPA, 1980a; EPA, 1981), it may not be either practical or economical for power plants to do so (UWAG, 1981b). Because of uncertainties associated with removal mechanisms and treatment effectiveness, more information and experience with this process are needed. Compared to alternative processes, such as reverse osmosis, electrodialysis, or ion exchange, chemical precipitation is relatively inexpensive. Information about capital and operating costs can be found in Colley et al. (1978).

DEEP BED FILTRATION

Deep bed filtration processes remove suspended solids from wastewaters by passing the water through a bed of one or more layers of granulated filter medium. Suspended particulate material is trapped within the filter, accumulating both on the surface of the grains in the media and in pore spaces between grains. Filter media may be any of a number of materials, but sand, gravel, and anthracite are most common. If the filtration has been preceded by settling, filtration can often be improved by addition of a coagulant to the feedwater. Filters may be either gravity driven (open to the air) or pressure driven (enclosed in a pressure vessel).

The accumulated suspended material reduces filter porosity and increases the pressure drop across the filter. When the pressure drop reaches unacceptable levels, the filter must be regenerated by backwashing to remove the solids. In backwashing, wash water is sent through the filter in the reverse direction at sufficient velocity to fluidize the filter bed and dislodge the solids. The backwash stream can then be sent to a settling pond or other effluent treatment and disposal process. Filtration generally removes suspended materials in the range of from 0.1 to 50 microns.

Deep bed filtration has been used extensively for municipal and industrial wastewater treatment. It is a proven technology that can be obtained commercially. In power plants, it can be used to remove suspended solids either as a pretreatment step for priority pollutant control technologies or following clarification. A recent study has found that sand filtration can remove some arsenic, copper, nickel, and zinc from ash pond effluents, but that removal efficiencies vary dramatically between wastewaters (UWAG, 1979). To date filtration has seen only limited use in the electric utility industry.

COAGULATION AND FLOCCULATION

Coagulation and flocculation are standard wastewater treatment processes to remove suspended colloidal materials. These are materials intermediate between dissolved and suspended solids and not easily removed by techniques applicable to either. In coagulation, chemicals

are dispersed in the wastewater to destabilize the colloidal particles and permit aggregation. In the flocculation process, the particles are aggregated and bound together to form suspended materials that can be removed by sedimentation or filtration. Chemicals commonly used to cause or aid in coagulation include (1) metallic salts such as aluminum sulfate, ferric chloride, and ferric sulfate, and (2) synthetic or natural organic polymers.

A typical coagulation and flocculation process would include the following steps (EPA, 1981)

- Addition of the coagulant and coagulant aids to the wastewater.
- Rapid mixing to disperse the coagulant.
- Slow mixing to facilitate contact between particles and aggregation.

Coagulation is frequently used before normal sedimentation or filtration processes to improve solids removal efficiencies. In many cases, some dissolved metals may also be removed as hydroxides adsorbed and absorbed by the settled flocs, although removal efficiencies depend strongly on the characteristics of the wastewater and coagulation process.

Normally, power plants use coagulation and flocculation in treating makeup water supplies rather than wastewaters. When used in wastewater treatment, coagulation would be one step in a more complex treatment procedure.

WASTE TREATMENT AND DISPOSAL PONDS

Ponds or basins are traditionally used by power plants for handling and treating wastewater streams. They are a simple and inexpensive means to collect, concentrate, and treat a wide variety of liquid wastes, including sluiced bottom and fly ash, FGD sludge, coal-pile runoff, and most other waste streams. The primary requirement for treatment ponds is availability of inexpensive and appropriate land. Ponds may be used either for containing and settling suspended solids or for precipitating dissolved solids through solar evaporation. They may also serve solely as treatment facilities or as combined treatment and

disposal sites. When ponds become the final disposal site for solid wastes, they are normally reclaimed at the end of their active lives.

Ponds used to settle suspended solids must be designed to have adequate retention for both coarse and colloidal material to be removed. These ponds are normally used for treatment of sluiced bottom ash and fly ash. If lime is added to the influent water, dissolved solids may also be precipitated and removed in settling ponds. Pond size requirements depend on plant coal ash content, plant life, combustion processes, weather conditions, and the number of ponds used. If accumulated solids are periodically removed for off-site disposal, pond land requirements are much smaller. Useful pond life ends when pond overflow occurs too quickly to allow adequate retention time.

Evaporation ponds use solar energy to evaporate water and dry or stabilize solids in wastes. They are a primary means of treating blowdown streams and other wastes at zero-discharge plants. In these ponds, precipitated solids may either be allowed to accumulate on the pond bottom or be periodically removed for disposal. Evaporation ponds require large land areas and positive effective net evaporation of at least 50 cm per year. The effective rate depends on waste characteristics, but is normally from 50 to 70 percent of annual net evaporation rate (Colley et al., 1978). Area requirements are given by

$$A = .19.5G/V$$

where A = pond area (acres),

G = flow rate (gpm),

V = effective net evaporation rate (in./yr).

Treatment ponds can be designed to operate either in series or in parallel. When settling ponds are used in series, primary settling occurs in the first pond. The second and subsequent ponds are used for polishing pond effluent. Ponds are normally used in parallel when they contain combined ash and FGD sludges (CH2M, 1980). The ponds are operated as a batch system in which one pond stabilizes while the other is being filled.

Pond operation and design will be significantly affected by environmental constraints. Treatment and disposal ponds may have to be lined to minimize seepage and leaching of pond effluent into soils and groundwater. Lining requirements will depend on pond waste characteristics, the nature of underlying soil and groundwater, and applicable regulations. Available lining materials include soil cements, clay, asphalt, concrete, and plastic or rubber sheeting. No matter what material is used, however, corrosion, deterioration, and cracking or rupture of liners can occur over time, so monitoring of pond leaching may be necessary. Pond overflow or effluent can be discharged or returned to the plant for reuse. In either case, effluent treatment will probably be necessary to meet water quality regulations or requirements.

Although all waste streams can potentially be treated with ponds, land requirements may limit the treatment of high volume wastes. In addition, it can be difficult to control the conditions within a pond, and treatment effectiveness may vary over time. Additional information about the use of ponds for treatment and disposal of coal ash can be found in Bahor et al. (1981). Treatment and disposal of FGD sludge is discussed in Knight et al. (1980). Capital and operating cost information about ponds and basins can be found in these two references as well as in Colley et al. (1978).

DRY ASH HANDLING SYSTEMS

Fly Ash Handling

Although not strictly a treatment technology, dry fly ash handling systems have been proposed as an alternative means of eliminating ash pond discharges from power plants (EPA, 1980a). Dry handling systems are pneumatic, and may be either vacuum or pressure operated. These systems are frequently used in coal-fired power plants and generate no appreciable quantities of wastewater.

In these systems, fly ash collected in the electrostatic precipitator hoppers is pneumatically conveyed through segregating valves to a storage silo. The ash can then be unloaded from the silo,

wetted, and transported by truck or train to a disposal site or commercial market. Vacuum systems use primary and secondary ash collectors above the storage silo. The dust-free air from these collectors passes through a filter and blowers to return to the atmosphere. Pressure-operated systems do not need these cyclone collectors at the storage silo. Displaced air leaves the silo through a vent filter.

Although these systems do not generate wastewater, they may have significant problems with fugitive dust emissions unless properly maintained. They are generally difficult to retrofit to a power plant with wet ash handling, as virtually no equipment is common to the two types of systems. Under normal circumstances, such replacement would be done only if (1) sluice water were difficult to obtain, (2) government regulations prohibited or limited pond discharges, or (3) commercial markets for fly ash improved (EPA, 1980a). Capital and operating cost information for dry ash handling and disposal systems can be found in Bahor et al. (1981) and EPA (1980a).

Bottom Ash Handling

Dry bottom ash handling systems are normally found only on stoker-fired boilers. Stoker-fired boilers are generally used in older, small coal-fired power plants. Because such dry handling systems are not appropriate for modern large power plants, we will not address them further. However, we describe below wet bottom ash handling systems that use dewatering bins.

RECIRCULATING ASH HANDLING SYSTEMS

Partial or complete recirculation of transport water can be used to limit discharges from wet ash handling systems. In recirculating systems, the sluiced ash is pumped to primary and secondary settling ponds. The effluent flows through another "clear" pond which serves as a holding basin for the recirculation water. From this pond it is returned to the ash handling system for reuse as sluice water.

A fraction of the clear pond overflow can be discharged to control buildup of dissolved solids in the recirculating transport water. Before discharge to the receiving waters, this blowdown would probably

require clarification and filtration to meet effluent standards. The amount of blowdown, and the specific treatment required, would be determined by ash characteristics--specifically the dissolved solids content and the corrosiveness or scaling tendency of the recirculated transport water.

As an alternative to discharging a blowdown stream, scale formation could be controlled in ash transport water by means of lime-soda softening or other sidestream treatment. This process, however, would produce another waste stream for treatment or disposal, although it would be of smaller volume.

As with dry handling systems, recirculation would only be retrofitted onto an existing plant if sluice water became scarce or effluent regulations severely restricted discharge of pond overflows. Unlike dry handling, however, retrofit would not require replacement of the entire ash handling system. Instead, only additional pumps and piping would likely be necessary. Capital and operating costs of recirculating ash handling systems can be found in Bahor et al. (1981).

DEWATERED BOTTOM ASH HANDLING

In most modern furnaces, bottom ash and slag are collected and quenched in water-filled hoppers below the furnace. The collected ash and slag may then be crushed and sluiced to a treatment or disposal pond. As an alternative, however, the ash may be sluiced to dewatering bins, where it is dried to about 20 percent moisture (EPA, 1980a). In this condition it can be loaded into trucks, trains, or conveyors for transport to a storage or disposal site.

The water recovered from the dewatering bins is recirculated to the ash collection and transport system. As with recirculating fly ash systems, lime-soda softening or other sidestream treatment may be required for the recirculating transport water to control solids buildup and scale formation. Such treatment would again create an additional waste stream for treatment or disposal. Because such sidestream softening is not a proven technology in power plants, a blowdown stream might be used to control dissolved solids and scale formation. This blowdown stream would require further treatment before it could be

discharged to receiving waters. Depending on plant and waste stream characteristics, such treatment might include routing to the fly ash sluice water system, evaporation ponds, or physical-chemical treatment as described earlier.

Capital and operating cost information for bottom ash handling and disposal systems can be found in Bahor et al. (1981).

Appendix F

EXPOSURE PATHWAY CALCULATIONS

In Chap. 5 we discussed generally how to calculate exposure rates for the different pathways. In this appendix we present the specific equations that are used to make these calculations in the exposure program. In general, the user of this risk methodology will not have to apply any of these equations, only supply the essential parameters and input data. The equations express exposure rates as a function of pollutant species i , population group m , and subregion r . In making risk calculations for a particular health effect, the analyst would add the exposure rates by subregion for the appropriate exposure intake modes and pollutant species. It may also be necessary to combine exposure rates for the different population groups, if dose-response data do not distinguish between them.

INHALATION

Inhalation exposure rates are expressed in terms of total pollutant concentration in the atmosphere. The concentration is the sum of the gaseous and particulate fractions, and will be a function of the pollutant species i and population subregion r . Accordingly, exposure rates are given by

$$I_h(i,m,r) = C(i,1,r) + C(i,2,r) \quad (F.1)$$

where $I_h(i,m,r)$ = inhalation exposure of pollutant i by individual of population group m in subregion r (mg/m^3),

$C(i,1,r)$ = concentration of pollutant i as gas in air of subregion r (mg/m^3),

$C(i,2,r)$ = concentration of pollutant i as particulates in air of subregion r (mg/m^3).

This expression is independent of population group m . The pollutant concentrations are average values over the subregion and can be determined directly from the atmospheric transport model output.

DERMAL CONTACT

Dermal contact exposure can arise from contact with atmospheric pollutants or contaminated waters. As discussed earlier, exposure rates are expressed as mean pollutant concentrations in the appropriate medium. These concentrations are adjusted to account for fractional body area and duration of exposure.

Atmospheric Pollutants

Because they depend on atmospheric gas and particulate concentrations, exposure rates for atmospheric contact vary with subregion. Although they also vary with population subgroup, it is unlikely that this would be a significant consideration. Exposure rates may be calculated from

$$D_a(i,m,r) = \epsilon_a t_a(m) [C(i,1,r) + C(i,2,r)] / 8760.0 \quad (F.2)$$

where $D_a(i,m,r)$ = dermal exposure rate from atmospheric pollution (mg/m^3),

ϵ_a = fraction of body area exposed to the atmosphere
(dimensionless),

$t_a(m)$ = time of dermal exposure to atmosphere (hr/yr).

The atmospheric pollutant concentrations used for inhalation exposure should also be applied to calculating this type of exposure. Values for the remaining parameters must be estimated from other information.

Water Pollutants

For dermal contact with surface and groundwater, the exposure equations can be divided into recreational exposure to untreated surface water and domestic exposure to treated surface or groundwater. The equations for determining exposure from these pathways are

$$D_{wr}(i,m,r) = \epsilon_{wr} t_r(m) C_r(i) / 8.76 \quad (F.3)$$

$$D_{wd}(i,m,r) = \epsilon_{wd} t_d(m) \{ C(i,4,r) T_f(i,4,r) v_f(r) + C(i,5,r) T_f(i,5,r) [1.0 - v_f(r)] \} / 8.76 \quad (F.4)$$

where $D_{wr}(i,m,r)$ = recreational dermal exposure rate (mg/m³),

$D_{wd}(i,m,r)$ = domestic dermal exposure rate (mg/m³),

ϵ_{wr} = fraction of body area exposed during water recreation (dimensionless),

ϵ_{wd} = fraction of body area exposed during domestic water use (dimensionless),

$t_r(m)$ = time of recreational exposure (hr/yr),

$t_d(m)$ = time of exposure to domestic water (hr/yr),

$C_r(i)$ = mean concentration of pollutant species i in surface water used for recreation (mg/l),

$C(i,4,r)$ = pollutant concentration in surface water used for domestic purposes (mg/l),

$C(i,5,r)$ = pollutant concentration in groundwater used for domestic purposes (mg/l),

$T_f(i,4,r)$ = fraction of pollutant remaining in surface water after treatment (dimensionless),

$T_f(i,5,r)$ = fraction of pollutant remaining in groundwater after treatment (dimensionless),

$v_f(r)$ = fraction of domestic water supply derived from surface sources (dimensionless).

Pollutant concentrations for domestic water supplies must be calculated from the surface and groundwater transport models and information about the water supply or intake points for each subregion. Concentrations in recreational water must also be determined from the surface water model output and the locations of local recreational sites. Water treatment efficiencies can be estimated from sources such as Sorg (1978), Sorg and Logsdon (1978), Sorg et al. (1978), Sorg (1979), and Patterson (1975) and descriptions of local treatment processes. Exposure times and fractions must be estimated from available local information.

INGESTION

Exposure can originate from numerous sources that we have classified and described as ingestion of (1) atmospheric particulates, (2) drinking water, (3) aquatic organisms, (4) vegetable products, and (5) animal products. We will consider each of these exposure pathways separately. In making ingestion calculations, it may be necessary to know mean human weights as a function of sex and age group. Because this information may be difficult to locate, we have provided it in Table F.1.

Table F.1
TYPICAL POPULATION WEIGHTS
(kg)

Age Group	Sex	Weight
Infant	M	7.5
	F	6.9
Child	M	21.9
	F	21.0
Teenager	M	52.9
	F	48.4
Adult	M	78.0
	F	64.9

SOURCES: NHS (1979); NHS (1977);
Finch and Hayflick (1971), Gehan and
George (n.d.).

Atmospheric Particulates

We can calculate ingestion of atmospheric particulates from the air pollution concentrations used in the inhalation exposure calculations. The exposure rate is given by

$$I_p(i,m,r) = C(i,2,r)\alpha U_i(m) \quad (F.5)$$

where $I_p(i,m,r)$ = ingestion exposure rate for atmospheric particulates (mg/day),

α = fraction of inhaled particulates ingested
(dimensionless),

$U_i(m)$ = specific inhalation rate (m^3/day).

These exposure rates again vary with subregion, pollutant species, and population group. The fraction of inhaled particulates depends on the pollutant and particle size distribution in the air. We have noted that a nominal default value of 0.25 can be assumed in the absence of more specific information. If this is an important exposure source, however, this default value should certainly be investigated. Mean specific inhalation rates have been presented in Table 5.1.

Drinking Water

Exposure through consumption of drinking water can be calculated from the concentrations used in the dermal contact analysis. If dermal contact does not constitute an important pathway, the surface and groundwater concentrations for each subregion should be derived from the surface and groundwater transport model outputs and data describing the locations of withdrawal points for domestic water supplies. Such information, as well descriptions of water treatment processes and water supplies, should be available from local public works agencies. Treatment efficiencies for the water treatment processes used in the region can be estimated as described earlier. Given these considerations, the exposure from consumption of drinking water can be calculated from

$$I_{dw}(i,m,r) = U_w(m)\{C(i,4,r)v_f(r)T_f(i,4,r) + C(i,5,r)[1.0 - v_f(r)]T_f(i,5,r)\} \quad (F.6)$$

where $I_{dw}(i,m,r)$ = ingestion exposure rate for drinking water consumption (mg/day),

$U_w(m)$ = specific drinking water consumption rate (l/day).

$C(i,4,r)$ = pollutant concentration in surface water used for domestic purposes (mg/l),

$C(i,5,r)$ = pollutant concentration in groundwater used for domestic purposes (mg/l),

$T_f(i,4,r)$ = fraction of pollutant remaining in surface water after treatment (dimensionless),

$T_f(i,5,r)$ = fraction of pollutant remaining in groundwater after treatment (dimensionless),

$v_f(r)$ = fraction of domestic water supply derived from surface sources (dimensionless).

Estimates of drinking water consumption have been presented in Tables 5.2 and 5.3. We should probably consider these rates to be high, in light of the growing consumption of bottled beverages, most of which probably originate outside the power plant region. The analyst may calculate a better estimate of exposure by using some fraction of the nominal rates and adding pollutant as background in the diet.

Aquatic Organisms

Consumption of aquatic organisms, primarily fish and shellfish, could be an important pathway for some pollutants in some regions. The problem has two additional complications not present in the drinking water calculations. First, the pollutant concentrations in different fish and shellfish species will not be constant, although they may have reasonably constant ratios to the pollutant concentration in surface waters. We deal with this problem by using bioaccumulation factors as described in Chap. 5 and presented in Table 5.5. Second, fish consumed in the region may originate from outside the area and will not be contaminated by plant emissions. Accordingly, we must consider both the production and consumption rates for local aquatic species.

Production rates (annual catches) may be available from state wildlife or fisheries agencies. Consumption rates may be more difficult to obtain, but national averages can be used as default values if no other data are available. To simplify the problem and provide an upper bound on effects, we assume that all regional production is used first to meet regional consumption. When production exceeds consumption, we assume that all consumption is locally produced and exposure rates can be determined from

$$I_q(i,m,s,r) = C_q(i,s)\beta_q(i,s)U_q(m,s) \quad (F.7)$$

where $I_q(i,m,s,r)$ = ingestion exposure rate for aquatic organism s (mg/day),

$C_q(i,s)$ = mean surface water pollutant concentration for organism s (mg/l),

$\beta_q(i,s)$ = bioconcentration factor for organism s (mg/kg per mg/l),

$U_q(m,s)$ = specific consumption rate for organism s (kg/day).

If regional consumption exceeds production for a specific organism, we must again include the pollutant contribution from externally produced supply. Under these circumstances, the exposure rate expression becomes

$$I_q(i,m,s,r) = [U_q(m,s)/L_q(s)][C_q(i,s) - B_q(i,s)] + B_q(i,s)U_q(m,s) \quad (F.8)$$

where $B_q(i,s)$ = background pollutant concentration in aquatic organism s from outside region (mg/kg),

$L_q(s)$ = ratio of regional production to regional consumption of aquatic organism (dimensionless).

Pollutant concentrations in the surface water are obtained from the surface water transport model outputs and information (or assumptions) about the location of aquatic species. Background pollutant concentrations in aquatic organisms must be obtained from the literature or calculated using the bioaccumulation factors and background pollutant concentrations. Potential sources for this information include Braunstein et al. (1977), Heit (1977), Jelinek and Corneliussen (1977), Mahaffey et al. (1975), and Leland and Luoma (1977).

Vegetable Products

The pollutant concentration associated with vegetable products can be divided into two components: (1) pollutant deposited on the plant from atmospheric processes or irrigation, and (2) pollutant taken up

from the soil. We calculate these two components separately for each product and add them. From NRC (1977) and WPPSS (1973b), we can calculate the concentration from deposition processes as

$$V_{sc}(i,v,r) = \{f_c(v)T_1(v)/[\delta_d(v)w_1]\}[J_a(i,r) + J_d(i,1,v,r) + J_d(i,2,v,r)] \quad (F.9)$$

where $V_{sc}(i,v,r)$ = contribution to pollutant concentration on vegetable product v from deposition (mg/kg),

$f_c(v)$ = capture fraction (dimensionless),

$\delta_d(v)$ = vegetative density for product v (kg/m²),

w_1 = weathering loss rate (/day),

$T_1(v)$ = translocation factor to edible parts of vegetable product v (dimensionless),

$J_a(i,r)$ = atmospheric pollutant deposition flux (mg/m²/day),

$J_d(i,1,v,r)$ = pollutant deposition flux from surface water irrigation (mg/m²/day),

$J_d(i,2,v,r)$ = pollutant deposition flux from groundwater irrigation (mg/m²/day).

The NRC (1977) and WPPSS (1973b) have suggested values of 0.2 to 0.25 for the capture fraction. The translocation factor can normally be assigned a value of 1.0 for leafy vegetables and 0.1 for other plant species. A standard weathering loss rate of 0.0495 per day (corresponding to a halflife of two weeks) can be used in the absence of specific data. The atmospheric deposition flux is specified as an average value over the subregion and should come directly from the output of the atmospheric transport model. Pollutant deposition fluxes from irrigation with surface and groundwater can be calculated from

$$J_d(i,1,v,r) = C_i(i,4,r)i(1,v,r) \quad (F.10)$$

$$J_d(i,2,v,r) = C_i(i,5,r)i(2,v,r) \quad (F.11)$$

where $C_i(i,4,r)$ = pollutant concentration in surface water used for
irrigation of vegetable product v in subregion r (mg/l),
 $C_i(i,5,r)$ = pollutant concentration in groundwater used for
irrigation of vegetable product v in subregion r (mg/l),
 $i(1,v,r)$ = irrigation rate with surface water (l/day),
 $i(2,v,r)$ = irrigation rate with groundwater (l/day).

The pollutant concentrations in surface and groundwater used for irrigation can be determined from the output of the appropriate environmental transport models and data or assumptions about the sources of irrigation water in each subregion. Information about the extent and amount of irrigation should be available from county agricultural agents and U.S. Water Resources Council (WRC, 1978).

The contribution to plant pollutant concentrations from pollutant taken up from the soil is given by

$$V_c(i,v,r) = C(i,3,r)\beta_v(i,v) \quad (F.12)$$

where $V_c(i,v,r)$ = contribution to pollutant in edible parts of
vegetable product v from soil uptake (mg/kg),
 $\beta_v(i,v)$ = bioconcentration factor (dimensionless),
 $C(i,3,r)$ = surface soil concentration of pollutant (mg/kg).

Surface soil pollutant concentrations should be subregion average values taken from the overland transport model. Bioconcentration factors must be measured or found in the literature. These factors and the problems associated with determining them have been discussed in Chap. 5.

For each vegetable product, we must determine pollutant concentrations in all appropriate subregions then calculate a mean concentration for all of the product produced in the region. This process will also be necessary for each of the animal products to be discussed in the next section. In general terms, this averaging process can be expressed mathematically as

$$C_p(i,x) = [1/P(x)] \sum_r C_p(i,r,x)P_r(r,x) \quad (F.13)$$

where $C_p(i,x)$ = mean concentration of pollutant species i in product x (mg/kg),

$P(x)$ = total regional production of product x (kg),

$C_p(i,r,x)$ = concentration of pollutant i , in product x , produced in subregion r (mg/kg),

$P_r(r,x)$ = production of product x in subregion r (kg).

In this equation, the units of concentration and production will be those most appropriate for the specific commodity under consideration.

After determining mean pollutant concentrations by product, we must compare local production with local consumption, as discussed earlier for aquatic organisms. We follow the same general process for vegetable products. If total regional production equals or exceeds regional consumption, we assume that all consumption comes from local sources. Exposure can then be found from

$$I_v(i,m,v,r) = U_v(m,v)C_v(i,v) \quad (F.14)$$

where $I_v(i,m,v,r)$ = ingestion exposure rate from vegetable product v (mg/day),

$U_v(m,v)$ = specific consumption rate for product v (kg/day),

$C_v(i,v)$ = mean pollutant concentration in product v (mg/kg).

Specific consumption rates should be determined from local information, when available. Otherwise, national average default values can be used. Some of these consumption rates are presented in Tables 5.2 and 5.3. Other data on consumption rates can be found in USDOA (1980).

If regional production does not exceed regional consumption, we must include externally grown produce with an assumed background pollutant concentration. The expression for exposure rate then becomes

$$I_v(i,m,v,r) = [U_v(m,v)/L_v(v)][C_v(i,v) - B_v(i,v)] + B_v(i,v)U_v(m,v) \quad (F.15)$$

where $B_v(i,v)$ = background pollutant concentration in vegetable product from outside region (mg/kg),

$L_v(v)$ = ratio of regional production to regional consumption of vegetable product (dimensionless).

Background pollutant concentrations for outside products must either be measured or taken from literature sources. Some potential sources were listed earlier in the discussion of aquatic organisms.

Animal Products

Animals take up toxic pollutants primarily from their feed and water. These pollutants may subsequently appear in products consumed by the regional population. As with vegetable products, we must determine the pollutant concentrations in each important product by subregion, then average these to calculate a mean concentration for local production of each product.

In general terms, the concentration of pollutant in an animal product (including meats, milk, and eggs) can be calculated from

$$C_m(i,a,r) = m_f(i,a)[C_{af}(i,a,f,r)Q_{af}(a,f,r) + C_{aw}(i,a,r)Q_{aw}(a,r)] \quad (F.16)$$

where $C_m(i,a,r)$ = pollutant concentration in animal product a (mg/kg),

$m_f(i,a)$ = fraction of daily intake of pollutant i found in animal product a (mg/kg per mg/day),

$C_{af}(i,a,f,r)$ = pollutant concentration in feed f for animal product a (mg/kg),

$C_{aw}(i,a,r)$ = pollutant concentration in drinking water for animal product a (mg/l),

$Q_{af}(a,f,r)$ = consumption rate of polluted feed (kg/day),

$Q_{aw}(a,r)$ = consumption rate of polluted water (l/day).

The fraction of daily pollutant intake found in each product must come from the literature. Some default values can be found in NRC (1977).

The quantities of feed and water consumed by animals must also come from the literature. Some default values from NRC (1977) and WPPSS (1973b) are given in Table 5.6. Pollutant concentrations in water can be determined from the concentrations calculated by the environmental transport models and information or assumptions about the sources of animal water in each subregion. Pollutant concentrations in feed must come from (1) earlier calculations of concentrations in vegetable products (grass, corn, and other feed crops) in the subregion, (2) regional data, or (3) literature default values. Some sources for these default values include Braunstein et al. (1977), Lisk (1972), and Dvorak et al. (1978), although a limited literature search should be able to locate reasonable data for many pollutants.

After determining the concentration by subregion, we must again calculate the mean concentration for all regional production of each product. This can be done using Eq. (F.13) as described earlier. Next, as with vegetable products and aquatic organisms, we must compare total regional production with total consumption to determine if external supplies of any product must be included. If total regional production equals or exceeds regional consumption, we assume all consumption comes from regional sources. Exposure rates can be found from

$$I_a(i,m,a,r) = U_a(m,a)C_a(i,a) \quad (F.17)$$

where $I_a(i,m,a,r)$ = ingestion exposure rate from animal product a (mg/day),

$U_a(m,a)$ = specific consumption rate for product a (kg/day),

$C_a(i,a)$ = mean pollutant concentration in product a (mg/kg).

Default values for specific consumption rates have been given for some products in Tables 5.2 and 5.3. Other values can be calculated from data in USDA (1980).

If regional production does not exceed regional consumption, we must include externally grown produce with an assumed background pollutant concentration. The expression for exposure rate becomes

$$I_a(i,m,a,r) = [U_a(m,a)/L_a(a)][C_a(i,a) - B_a(i,a)] \\ + B_a(i,a)U_a(m,a) \quad (F.18)$$

where $B_a(i,a)$ = background pollutant concentration in animal product
from outside region (mg/kg),

$L_a(a)$ = ratio of regional production to regional consumption of
animal product (dimensionless).

Background concentrations in animal products must either be measured or
estimated from literature sources pertaining to the pollutant of
interest or from those mentioned earlier in this appendix.

Appendix G

DESCRIPTION OF PROGRAM TO CALCULATE EXPOSURE RATES AND HEALTH EFFECTS

PROGRAM VARIABLES--DESCRIPTION, LIMITS, AND UNITS

The primary variables used in the program that calculates exposure rates and health effects are shown in Table G.1. These calculations are discussed in detail in Chaps. 5 and 6 and App. F.

PROGRAM INPUTS AND FORMATS

General Considerations

In the description of program inputs below, we have used certain conventions to describe the information requirements. All information must be entered in the order indicated, and in the appropriate formats. Where more than one variable is required on a given input line, these variables are described in the order in which they must appear on the line. Field widths indicate the number of consecutive spaces occupied by the variable, beginning the first field in the first space of the line. Numbers following slashes (/) in field widths indicate the number of blank characters that must follow the indicated field. Variable names are taken from the list described earlier in this appendix. The variable or field types are defined as follows:

- A--alphabetical or numeric characters used as a name or indicator; entered left-justified within the field.
- E--exponential number; entered as a floating point number (see below) with or without an exponent. The exponent must be a signed one- or two-digit integer, right-justified within the field, and may be preceded by an 'E'.

Table G.1

PROGRAM VARIABLES

Variable	Description
a	Index for animal products (0-10)
AAC(h,p)	Crump dose-response model parameters (-)
AAD(h,p)	Multistage dose-response model parameters (-)
ACB(a)	Background pollutant concentration in animal products from outside the region (mg/kg)
AFD(r)	Atmospheric particulate deposition flux (mg/m ² /day)
ANIMAL(a)	Animal products (-)
AM(h,p)	Multihit dose-response model parameters (-)
AO(h)	One-hit dose-response model parameters (-)
AP(h,p)	Probit dose-response model parameters (-)
AQUAT(s)	Aquatic organisms (-)
AW(h,p)	Weibull dose-response model parameters (-)
B(s)	Bioconcentration factor for aquatic organisms (-)
BAFD(r)	Background atmospheric particulate deposition flux (mg/m ² /day)
BC(k,r)	Background pollutant concentrations in media (see below)
BCF(a,r,f)	Background pollutant concentration in animal food (mg/kg)
BCI(j,r)	Background pollutant concentration in irrigation water (mg/l) (j = 1--surface water; 2--groundwater)
BCM(a,r)	Background pollutant concentration in animal products (mg/kg)
BCQ(s)	Background pollutant concentration in surface waters for aquatic organisms (mg/l)
BCR(l)	Background pollutant concentration in recreational water (mg/l)
BCW(a,r)	Background pollutant concentration in animal drinking water (mg/l)
BDA(i,m,r)	Dermal exposure rate from background levels in the atmosphere (mg/m ³)
BDT(i,m,r)	Total dermal exposure rate from background concentrations (mg/m ³)
BDTW(i,r)	Total background dermal dose--weighted (mg/m ³)
BDTWC(i,r)	Total background dermal dose--weighted/converted (mg/m ³)
BDWD(i,m,r)	Dermal exposure rate from background concentrations in domestic water (mg/m ³)
BDWR(i,m,r)	Dermal exposure rate from background concentrations in recreational water (mg/m ³)
BFD(k,v,r)	Background pollutant deposition flux from irrigation water (mg/m ² /day)
BIG(m)	Background exposure rate from food consumption not considered in analysis (mg/day)

Table G.1 (Cont.)

Variable	Description
BIGA(i,a,r)	Background exposure rate from ingestion of animal products (mg/day)
BIGQ(i,m,r)	Background exposure rate from ingestion of aquatic organisms (mg/day)
BIGP(i,m,r)	Background exposure rate from ingestion of atmospheric particulates (mg/day)
BIGT(i,m,r)	Total background exposure rate from ingestion (mg/day)
BIGTC(i,m,r)	Total background exposure rate from ingestion--converted (mg/kg)
BIGTW(i,r)	Total background ingestion exposure rate--weighted (mg/day)
BIGTWC(i,r)	Total background ingestion dose--weighted/converted (mg/kg)
BIGV(i,m,r)	Background exposure rate from ingestion of vegetable products (mg/day)
BIGW(i,m,r)	Background exposure rate from ingestion of drinking water (mg/day)
BIH(i,m,r)	Background inhalation exposure rate (mg/m ³)
BIHW(i,r)	Background inhalation dose--weighted (mg/m ³)
BIHWC(i,r)	Background inhalation dose--weighted/converted (mg/m ³)
BMCM(a)	Mean background pollutant concentration in animal products (mg/kg)
BMF	Background pollutant concentration in remainder of diet not considered in analysis (mg/kg)
BMVC(v)	Mean background pollutant concentration in vegetable products (mg/kg)
BR(h,d,e)	Health risk from background exposure levels (cases)
BRW(h)	Health risk from background exposure levels for Weibull model with plant lifetime exposure (cases)
BSIGA(m,a)	Background ingestion exposure rate by type of animal product (mg/day)
BSIGQ(m,s)	Background ingestion exposure rate by type of aquatic organism (mg/day)
BSIGV(m,v)	Background ingestion exposure rate by type of vegetable product (mg/day)
BTMD(r)	Total background dermal contact dose for health effects (mg/m ³)
BTMG(r)	Total background ingestion dose for health effects (mg/kg)
BTMH(r)	Total background inhalation dose for health effects (mg/m ³)
BVC(v,r)	Background contribution to pollutant concentration in vegetable products from soil uptake (mg/kg)

Table G.1 (Cont.)

Variable	Description
BVSC(v,r)	Background contribution to pollutant concentration in vegetable products from surface deposition (mg/kg)
C(k,r)	Pollutant concentrations in media (k = 1--atmospheric gases (mg/m ³); 2--atmospheric particulates (mg/m ³); 3--surface soil (mg/kg); 4--surface water (mg/l); 5--groundwater (mg/l))
CASE	Description of case (-)
CF(a,r,f)	Pollutant concentration in animal feed (mg/kg)
CI(j,r)	Pollutant concentration in irrigation water (mg/l)
CM(a,r)	Pollutant concentration in animal products (mg/kg)
CQ(s)	Mean surface water pollutant concentration for aquatic organisms (mg/l)
CR(l)	Pollutant concentration in recreational waters (mg/l)
CV(v)	Bioconcentration factor for vegetable product (-)
CW(a,r)	Pollutant concentration in animal drinking water (mg/l)
d	Index for dose-response relationships (1-6)
DA(i,m,r)	Dermal exposure rate from atmospheric pollution (mg/m ³)
DR(h,d)	Dose-response relationship
DT(i,m,r)	Total dermal exposure rate (mg/m ³)
DTW(i,r)	Total dermal dose--weighted (mg/m ³)
DTWC(i,r)	Total dermal dose--weighted/converted (mg/m ³)
DWD(i,m,r)	Dermal exposure rate from domestic water (mg/m ³)
DWR(i,m,r)	Dermal exposure rate from recreational water (mg/m ³)
EDA	Fraction of body area exposed to atmosphere (-)
EDAD	Fraction of body area exposed to domestic water (-)
EDAR	Fraction of body area exposed during recreation (-)
EFFECT(h)	Health effects
e	Index for exposure period (plant/human lifetime) (-)
f	Index for animal product feed types (-)
FC(v)	Capture fraction for vegetable product (-)
FD(j,v,r)	Pollutant deposition flux from irrigation water (mg/m ² /day)
FI(k,v,r)	Fraction of vegetable product irrigated (-)
FEED(a,r,f)	Product animal feeds (-)
FS	Specific inhalation rate coefficient (-)
GS	Coefficient of dermal mass transfer per unit area (-)
h	Index for health effects (1-10)
HLIFE	Human lifetime (yr)
HS	Specific ingestion rate coefficient (-)
i	Index for pollutant species (1-5)
IERR	Indicator to eliminate underflow error messages
IGA(i,m,r)	Total ingestion exposure rate for animal products (mg/day)

Table G.1 (Cont.)

Variable	Description
IGP(i,m,r)	Ingestion exposure rate for atmospheric particulates (mg/day)
IGQ(i,m,r)	Total ingestion exposure rate for aquatic organisms (mg/day)
IGT(i,m,r)	Total ingestion exposure rate (mg/day)
IGTC(i,m,r)	Total ingestion exposure rate--converted (mg/day)
IGTW(i,r)	Total ingestion exposure rate--weighted (mg/kg)
IGTWC(i,r)	Total ingestion dose--weighted/converted (mg/kg)
IGV(i,m,r)	Ingestion exposure rate for vegetable products (mg/day)
IGW(i,m,r)	Ingestion exposure rate for drinking water (mg/day)
IH(i,m,r)	Inhalation exposure rate (mg/m ³)
IHW(i,r)	Inhalation dose--weighted (mg/m ³)
IHWC(i,r)	Inhalation dose--weighted/converted (mg/m ³)
IMODE	Exposure modes (-)
IRR(v)	Irrigation rate (l/m ² /day)
IWAT	Types of domestic water supply (-)
IWT(j,r)	Type of domestic water treatment process (-)
j	Index for water types (0-2)
k	Index for environmental media (1-5)
l	Index for recreational locations (0-5)
LA(a)	Ratio of local consumption to local production of animal products (-)
LQ(s)	Ratio of local consumption to local production of aquatic organisms (-)
LV(v)	Ratio of local consumption to local production of vegetable products (-)
m	Index for population group (1-4)
MCM(a)	Mean pollutant concentration in animal products (mg/kg)
MF(a)	Fraction of daily pollutant intake in animal product (-)
MFC	Mean capture fraction for plants (-)
MVC(v)	Mean pollutant concentration in vegetable products (-)
n	Index for chemical species in health effect calculations (-)
NAO	Number of aquatic organisms (0-5)
NAP	Number of animal products (0-10)
NAPR(a)	Number of subregions producing animal products (-)
NDR(h)	Number of dose-response relationships (-)
NHE	Number of health effects (1-10)
NPG	Number of population groups (1-4)
NPR	Number of population subregions (1-100)
NPS	Number of pollutant species (1-5)
NRL	Number of recreation locations (0-5)
NSC(h)	Number of chemical species determining health effect (-)

Table G.1 (Cont.)

Variable	Description
NTF(a,r)	Number of types of feed consumed by product animals in each population subregion (-)
NTP	Number of water treatment processes (1-10)
NVS	Number of vegetation species (0-10)
NVSR(v)	Number of subregions producing vegetable products (-)
P	Index for dose-response model parameters
P(m,r)	Population at risk (-)
PA(a)	Regional production of animal products (kg/day)
PIG	Fraction of atmospheric particulates ingested
PLIFE	Power plant lifetime (yr)
POLLUT(i)	Pollutant species (-)
POPGRP(m)	Population groups (-)
PQ(s)	Regional production of aquatic organisms (kg/day)
PSC(h,n)	Chemical species producing health effects (-)
PV(v)	Regional production of vegetable products (kg/day)
QCB(s)	Background pollutant concentration in aquatic organisms from outside region (mg/kg)
QF(a,r,f)	Product animal feed consumption rate (kg/day)
QW(a)	Animal product water consumption rate (l/day)
r	Index for population subregion (1-100)
R(h,d,e)	Health risk with plant in place (cases)
RISK(h,d,e)	Net health risk (plant less background) (cases)
RPA(a,r)	Subregion production of animal products (kg/day)
RPV(v,r)	Subregion production of vegetable products (kg/day)
RSG	Ingestion exposure absorption fraction (-)
RSI	Inhalation exposure absorption fraction (-)
RUF(l)	Relative use of recreational location (-)
s	Index for aquatic organisms (0-5)
SD	Mean effective surface soil density (kg/m ²)
SIGA(m,a)	Ingestion exposure rate by animal product (mg/day)
SIGQ(m,s)	Ingestion exposure rate by aquatic organism (mg/day)
SIGV(m,v)	Ingestion exposure rate by vegetable product (mg/day)
TDA(m)	Time of dermal exposure to atmosphere (hr/yr)
TDD(m)	Time of dermal exposure to domestic water (hr/yr)
TDR(m)	Time of dermal exposure to recreational water (hr/yr)
TF(n)	Fraction of pollutant remaining after water treatment
TL(v)	Translocation factor to edible parts of vegetation (-)
TMD(r)	Total dermal contact dose for health effect (mg/m ³)
TMG(r)	Total ingestion dose for health effect (mg/kg)
TMH(r)	Total inhalation dose for health effects (mg/m ³)
TP(m)	Total population by group (-)
TPR(r)	Total population by subregion (-)
TPS	Total population for region (-)

Table G.1 (Cont.)

Variable	Description
TS	Pollutant transfer loss rate through soil (/day)
TU(m)	Total food consumption rate (kg/day)
TUA(a)	Regional consumption of animal products (kg/day)
TUQ(s)	Regional consumption of aquatic organisms (kg/day)
TUT(m)	Food consumption rate for products considered (kg/day)
TUV(v)	Regional consumption of vegetable products (kg/day)
UA(m,a)	Specific consumption rate for animal products (kg/day)
UI(m)	Specific inhalation rate (m ³ /day)
UQ(m,s)	Specific consumption rate for aquatic organisms (kg/day)
UV(m,v)	Specific consumption rate for vegetable products (kg/day)
UW(m)	Specific consumption rate for drinking water (l/day)
v	Index for vegetable products (0-10)
VC(v,r)	Contribution to pollutant concentration in vegetable products from soil uptake (mg/kg)
VCB(v)	Background pollutant concentration in vegetable products from outside region (mg/kg)
VEGET(v)	Vegetable products (-)
VF(r)	Fraction of domestic water supply by surface sources (-)
VG(v)	Vegetative surface density (kg/m ²)
VSC(v,r)	Contribution to pollutant concentration in vegetable products from surface deposition (mg/kg)
W(m)	Human weight (kg)
WL	Weathering loss rate from vegetation surface (/day)

- F--floating point number entered anywhere within the field with a decimal point.
- I--integer number; entered right-justified within the field without a decimal point.
- X--blank spaces.

When specific names are required in a field, these have been designated by single quotation marks ('). Required blanks within a field are shown by underscores (_). Where information may be optional, we have indicated this with brackets ([]). When an entire group of cards must be repeated, we have used asterisks (*) to identify the group

and specify the number of times this type of information must be entered.

Information must be entered in consistent order. In other words, once an order for subregions has been determined, this order must be followed throughout the program. This consistency also holds for information relating to aquatic organisms, animal products, vegetable products, and population groups.

The input information for the program has been separated into four major sections: (1) general case description; (2) data describing the initial pollutant species; (3) data describing other pollutant species; and (4) information specifying health effects calculations. We discuss each of these sections in detail below.

General Case Description

The initial information required by the program defines and describes the case, including intake modes, types of water used for domestic supply, number of chemical species, population groups, and types of exposure pathways considered. The required information is shown in Table G.2.

Initial Pollutant Species

This information specifies environmental concentrations and deposition rates for the initial pollutant species considered in the analysis. These inputs differ from those required for additional species, in that they also include other information describing parameters required for calculating exposure rates for certain pathways. Data requirements and input formats are shown in Table G.3. Note that some data may be omitted, as indicated, depending on the details of the analysis.

Additional Pollutant Species

Basic information describing the case was entered in the previous section. Data entered for subsequent pollutant species relate primarily to environmental concentrations and deposition rates for the additional species. The specific data requirements and input formats for this

Table G.2

INPUTS FOR GENERAL CASE DESCRIPTION

Number of Lines	Field Width	Variable Name	Field Type	Comments
4	80	CASE	A	All four lines will be printed at top of program output
1	3(8)	IMODE	A	'INHALE__', 'INGEST__', and 'DERMAL__' must be used to indicate exposure intake modes to be considered--they may be entered in any order
1	2(8)	IWAT	A	'SURFACE_' and 'GROUND__' must be used to indicate domestic water sources to be considered--they may be entered in any order
1	5/5	NPS	I	
	5(8/2)	POLLUT	A	Up to five species names, occupying a maximum of eight spaces each, padded with blanks to ten-space fields
1	5/5	NPG	I	
	5(8/2)	POPGRP	A	Up to four names, each occupying a maximum of eight spaces with blanks to pad each field to ten spaces
1	5	NPR	I	
	6	PLIFE	F	
	6	HLIFE	F	
NPR	4(12/1)	P	E	Population by group per subregion
1	5	NAP	I	
	5	NAO	I	
	5	NVS	I	

information are shown in Table G.4. All appropriate information shown in the table should be entered for each additional pollutant species, in the order established at the beginning of the program. Note again that some data may be omitted, as indicated, depending on the details of the analysis.

Table G.3
INPUTS FOR INITIAL POLLUTANT SPECIES

Number of Lines	Field Width	Variable Name	Field Type	Comments
NPR	12/1	C(1,r)	E	Plant and background pollutant
	12/1	BC(1,r)	E	concentrations for atmospheric gases
	12/1	C(2,r)	E	and particulates by subregion {a}
	12/1	BC(2,r)	E	
NPR	12/1	C(3,r)	E	Plant and background pollutant
	12/1	BC(3,r)	E	concentrations for surface soil {b}
NPR	12/1	C(4,r)	E	Plant and background pollutant
	12/1	BC(4,r)	E	concentrations for surface water and
	12/1	C(5,r)	E	groundwater {c}
	12/1	BC(5,r)	E	
NPR	12/1	AFD	E	Plant and background pollutant
	12/1	BAFD	E	deposition fluxes by subregion {b}
1	10	RSH	F	{a}
1	6	FS	F	{b}
	6	HS	F	
	6	RSG	F	
1	4(12/1)	UI	E	{b}
	12	PIG	E	
1	4(12/1)	W	E	{b}
1	4(12/1)	TU	E	{b}
	12	BMF	E	
1	4(12/1)	UW	E	{b}
1	5	NTP	I	{b}
	10(6)	TF	F	
NPR	5	VF	F	{b}
	2(5)	IWT	I	Treatment types for surface water and
				groundwater by subregion
*****	NAO {d}			Repeat group for each species of
1	8	AQUAT	A	aquatic organism
	12/1	PQ	E	
	12/1	CQ	E	
	12/1	BCQ	E	
	12/1	B	E	
	12/1	QCB	E	
1	4(12/1)	UQ	E	

*****	NAP {e}			Repeat entire group for each type of
				animal product
1	8	ANIMAL	A	
	5	NAPR	I	

Table G.3 (Cont.)

Number of Lines	Field Width	Variable Name	Field Type	Comments
1	4(12/1)	UA	E	
1	12/1	MF	E	
	12/1	QW	E	
	12/1	ACB	E	
** NAPR				Repeat subgroup for each subregion in which animal product is produced
1	5	r	I	Subregion identifier
	5/1	NTF	I	
	12/1	RPA	E	
1	8/2	FEED	A	Should have NTF pairs of feed types and consumption rates
	12/1	QF	E	
	8/2	FEED	A	
	12/1	QF	E	
	8/2	FEED	A	
	12/1	QF	E	
1	5/1	r	I	
	12/1	CW	E	
	12/1	BCW	E	
1	3(12/1)	CF	E	Should have NTF values of each pollutant concentration
	3(12/1)	BCF	E	

1	12/1	MFC	E	
	12/1	SD	E	
	12/1	TS	E	
	12/1	WL	E	
***** NVS {f}				Repeat group for each type of vegetable product
1	8	VEGET	A	
	5	NVSR	I	
1	4(12/1)	UV	E	
1	12/1	CV	E	
	12/1	FC	E	
	12/1	IRR	E	
	12/1	TL	E	
	12/1	VG	E	
	12/1	VCB	E	

Table G.3 (Cont.)

Number of Lines	Field Width	Variable Name	Field Type	Comments
** NVSR				
				Repeat subgroup for each subregion in which vegetable product is produced
1	5/1	r	I	Subregion identifier
	12	RPV	E	
1	2(5)	FI	F	Surface water, groundwater
1	5/1	r	I	
	12/1	CI	E	Surface water
	12/1	BCI	E	
	12/1	CI	E	Groundwater
	12/1	BCI	E	
**				

1	4(12/1)	TDA	E	{g}
1	4(12/1)	TDD	E	{g}
1	4(12/1)	TDR	E	{g}
1	12/1	EDA	E	{g}
	12/1	EDAD	E	{g}
	12/1	EDAR	E	{g}
	12/1	GS	E	{g}
1	5/1	NRL	E	{g}
	5(12/1)	CR	E	Should have NRL values {g}
1	5(12/1)	BCR	E	Should have NRL values {g}
1	5(5)	RUF	E	Should have NRL values {g}

NOTE: Input data should be omitted as described below.

{a} Inhalation exposure not considered.

{b} Ingestion exposure not considered.

{c} Ingestion and dermal exposure not considered.

{d} Aquatic organisms not included.

{e} Animal products not included.

{f} Vegetable products not included.

{g} Dermal contact not considered.

Health Effects Calculations

This information describes the (1) type of health effects, (2) pollutant species, (3) intake modes, and (4) dose-response relationships to be used in the analysis. For each health effect, the program combines the relevant exposure rates for the specified pollutant

Table G.4

INPUTS FOR ADDITIONAL POLLUTANT SPECIES

Number of Lines	Field Width	Variable Name	Field Type	Comments
NPR	12/1	C(1,r)	E	Plant and background pollutant
	12/1	BC(1,r)	E	concentrations for atmospheric gases
	12/1	C(2,r)	E	and particulates by subregion {a}
	12/1	BC(2,r)	E	
NPR	12/1	C(3,r)	E	Plant and background pollutant
	12/1	BC(3,r)	E	concentrations for surface soil {b}
NPR	12/1	C(4,r)	E	Plant and background pollutant
	12/1	BC(4,r)	E	concentrations for surface water and
	12/1	C(5,r)	E	groundwater {c}
	12/1	BC(5,r)	E	
NPR	12/1	AFD	E	Plant and background pollutant
	12/1	BAFD	E	deposition fluxes by subregion {b}
1	5	NTP	I	{b}
	10(6)	TF	F	Should be NTP values
*****	NAO {d}			Repeat group for each species of
1	8	AQUAT	A	aquatic organism in previously
	12/1	PQ	E	determined order
	12/1	CQ	E	
	12/1	BCQ	E	
	12/1	B	E	
	12/1	QCB	E	

*****	NAP {e}			Repeat entire group for each type of
				animal product in previously
				determined order
1	12/1	MF	E	
	12/1	QW	E	
	12/1	ACB	E	
**	NAPR			Repeat subgroup for each subregion in
				which animal product is produced
1	5/1	r	I	Subregion identifier
	12/1	CW	E	
	12/1	BCW	E	
1	3(12/1)	CF	E	Should have NTF values of each pollutant
	3(12/1)	BCF	E	concentration

Table G.4 (Cont.)

Number of Lines	Field Width	Variable Name	Field Type	Comments
**** NVS {f}				Repeat group for each type of vegetable product in previously determined order
** NVSR				Repeat subgroup for each subregion in which vegetable product is produced
1	5/1	r	I	Subregion identifier
	12/1	CI	E	Surface water
	12/1	BCI	E	
	12/1	CI	E	Groundwater
	12/1	BCI	E	

1	5/1	NRL	E	{g}
	5(12/1)	CR	E	Should have NRL values {g}
1	5(12/1)	BCR	E	Should have NRL values {g}

NOTE: Input data should be omitted as described below.

- {a} Inhalation exposure not considered.
- {b} Ingestion exposure not considered.
- {c} Ingestion and dermal exposure not considered.
- {d} Aquatic organisms not included.
- {e} Animal products not included.
- {f} Vegetable products not included.
- {g} Dermal contact not considered.

species. It then calculates the background, plant, and net risk using the dose-response relationships chosen and supplied by the user. The input data and formats are shown in Table G.5.

ILLUSTRATIVE INPUTS

The inputs from a purely hypothetical data set (used to test the program) are presented below. These data do not represent an actual situation; they only show how the input would appear for an analysis incorporating all intake modes and exposure pathways.

Table G.5

INPUTS FOR HEALTH EFFECTS CALCULATIONS

Number of Lines	Field Width	Variable Name	Field Type	Comments
1 **** NHE	5	NHE	I	Repeat entire group for each health effect
1	8/2	KMODE	A	Modes--'INHALE__', 'INGEST__', and 'DERMAL__'
	8/2	EFFECT	A	
	5	NDR	I	Number of dose-response relationships to be used for this effect
	5	NSC	I	Number of pollutant species combined
1	10(5)	PSC	I	Should be NSC integers indicating pollutant species to be summed
** NDR				For each selected model, enter the name and appropriate parameter information as described below (there should be NDR models with parameters)
1	8/2	DR	A	Relationships--'ONEHIT__', 'MULTIHIT', 'MULTISTA', 'CRUMP__', 'PROBIT__', and 'WEIBULL__'
1	12	AO	E	One-hit model parameter
1	2(12/1)	AM	E	Dose parameter and number of hits in multihit model
1	5	n	I	Number of hits in multistage model
1	6(12/1)	AAD	E	First six parameters of model
1	6(12/1)	AAD	E	Remaining parameters
1	5	n	I	Number of hits in Crump formulation
1	6(12/1)	AAC	E	First six parameters of Crump model
1	6(12/1)	AAC	E	Remaining parameters
1	2(12/1)	AP	E	Intercept/slope of probit model
1	3(12/1)	AW	E	Constant/exponents for dose and time in Weibull model
**				

ILLUSTRATIVE INPUTS FOR EXPOSURE AND HEALTH EFFECTS MODEL

GENERAL DESCRIPTION OF CASE STUDY TO TEST OPERATION OF PROGRAM
HYPOTHETICAL DATA EMPLOYING ALL OPTIONS OF EXPOSURE AND HEALTH ROUTINES
AND TO ILLUSTRATE OUTPUT INFORMATION AND FORMATS

TEST NUMBER- 1 DATE- 3/14/83 2 POLLUTANTS/4 HEALTH EFFECTS

INHALE INGEST DERMAL

SURFACE GROUND

2	SPECIES1	SPECIES2				
4	INFANTS	CHILDREN	TEENS	ADULTS		
8	35.0	72.0				
4.46	E+02 4.771	E+03 3.172	E+03 1.7824	E+04		
1.83	E+02 1.963	E+03 1.305	E+03 7.334	E+03		
1.58	E+02 1.689	E+03 1.123	E+03 6.310	E+03		
1.222	E+03 1.3080	E+04 8.696	E+03 4.8869	E+04		
3.70	E+02 3.956	E+03 2.630	E+03 1.4780	E+04		
4.89	E+02 5.230	E+03 3.477	E+03 1.9541	E+04		
9.90	E+02 1.0597	E+04 7.045	E+03 3.9593	E+04		
1.59	E+02 1.705	E+03 1.134	E+03 6.370	E+03		
2	3	3				
1.00	E-06 1.00	E-07 1.00	E-06 1.00	E-07		
1.50	E-06 1.50	E-07 1.50	E-06 1.50	E-07		
2.00	E-06 2.00	E-07 2.00	E-06 2.00	E-07		
2.50	E-06 2.50	E-07 2.50	E-06 2.50	E-07		
3.00	E-06 3.00	E-07 3.00	E-06 3.00	E-07		
3.50	E-06 3.50	E-07 3.50	E-06 3.50	E-07		
4.00	E-06 4.00	E-07 4.00	E-06 4.00	E-07		
4.50	E-06 4.50	E-07 4.50	E-06 4.50	E-07		
6.10	E-01 6.00	E-01				
6.20	E-01 6.00	E-01				
6.30	E-01 6.00	E-01				
6.40	E-01 6.00	E-01				
6.50	E-01 6.00	E-01				
6.60	E-01 6.00	E-01				
6.70	E-01 6.00	E-01				
6.80	E-01 6.00	E-01				
1.50	E-02 1.00	E-02 4.00	E-02 2.00	E-02		
2.00	E-02 1.00	E-02 5.00	E-02 2.00	E-02		
2.50	E-02 1.00	E-02 6.00	E-02 2.00	E-02		
3.00	E-02 1.00	E-02 7.00	E-02 2.00	E-02		
3.50	E-02 1.00	E-02 8.00	E-02 2.00	E-02		
4.00	E-02 1.00	E-02 9.00	E-02 2.00	E-02		
4.50	E-02 1.00	E-02 1.00	E-01 2.00	E-02		
5.00	E-02 1.00	E-02 1.10	E-01 2.00	E-02		
5.00	E-01 1.00	E-01				
5.50	E-01 1.00	E-01				
6.00	E-01 1.00	E-01				
6.50	E-01 1.00	E-01				
7.00	E-01 1.00	E-01				
7.50	E-01 1.00	E-01				

8.00	E-01	1.00	E-01				
8.50	E-01	1.00	E-01				
1.00							
1.18	.1154	1.00					
3.84	E+00	1.014	E+01	2.192	E+01	2.192	E+01 2.50 E-01
7.20	E+00	2.15	E+01	5.06	E+01	7.15	E+01
5.50	E-01	1.19	E+00	1.50	E+00	1.28	E+00 3.00 E-03
5.50	E-01	7.10	E-01	7.10	E-01	1.01	E+00
4	0.20	0.40	0.60	0.80			
0.50	4	3					
0.75	2	1					
0.25	2	4					
0.60	3	4					
0.80	3	4					
1.00	3	4					
1.00	3	4					
0.50	3	4					
TROUT	1.50	E+03	2.00	E-02	1.00	E-02	2.00 E+01 4.00 E-02
0.00	E+00	1.50	E-03	3.50	E-03	4.70	E-03
BASS	5.00	E+02	3.00	E-02	1.00	E-02	5.00 E+01 8.00 E-02
0.00	E+00	1.80	E-03	4.20	E-03	5.60	E-03
SALMON	5.00	E+03	2.50	E-02	1.00	E-02	1.50 E+01 2.00 E-01
0.00	E+00	6.00	E-04	5.00	E-03	6.60	E-03
BEEF	2						
0.00	E+00	4.00	E-02	6.50	E-02	1.00	E-01
1.00	E-02	6.00	E+01	4.00	E-03		
2	2	6.00	E+05				
GRASS	3.50	E+01	CORN	1.50	E+01		
2	2.00	E-02	1.00	E-02			
3.00	E-02	5.00	E-02	2.00	E-02	2.50	E-02
4	2	8.00	E+05				
GRASS	3.50	E+01	CORN	1.50	E+01		
4	3.00	E-02	1.00	E-02			
3.50	E-02	6.00	E-02	2.00	E-02	2.50	E-02
PORK	1						
0.00	E+00	4.00	E-02	6.00	E-02	6.50	E-02
2.50	E-03	1.50	E+01	5.50	E-03		
5	2	5.60	E+05				
CORN	1.00	E+01	SOYBEANS	5.00	E+00		
5	3.50	E-02	1.00	E-02			
4.00	E-02	5.00	E-02	2.00	E-02	3.00	E-02
2.50	E-01	2.40	E+02	1.00	E-01	5.00	E-02
CORN	1						
0.00	E+00	1.00	E-02	1.25	E-02	1.35	E-02
2.00	E-01	2.50	E-01	1.50	E+00	2.00	E-01 5.00 E+00 4.00 E-02
2	4.00	E+04					
.50	.50						
2	2.50	E-02	1.00	E-02	6.00	E-02	2.00 E-02
WHEAT	2						
0.00	E+00	1.00	E-01	1.60	E-01	1.40	E-01
1.00	E-01	5.00	E-01	3.00	E+00	3.00	E-01 3.00 E+00 3.00 E-02

3	3.50	E+05						
.25	.25							
3	4.00	E-02	1.00	E-02	9.00	E-02	2.00	E-02
6	5.00	E+05						
1.00	0.00							
6	4.50	E-02	1.00	E-02	1.00	E-01	2.00	E-02
SOYBEANS 1								
0.00	E+00	2.00	E-02	3.50	E-02	3.00	E-02	
5.00	E-02	2.00	E-01	2.00	E+00	1.00	E-01	2.50 E+00 2.50 E-02
5	3.50	E+06						
.20	.10							
5	4.00	E-02	1.00	E-02	2.00	E-01	2.00	E-02
5.00	E+03	6.00	E+03	7.00	E+03	6.00	E+03	
5.50	E+01	3.50	E+01	5.00	E+01	6.00	E+01	
1.00	E+00	2.00	E+01	5.00	E+01	2.00	E+01	
9.00	E-01	5.00	E-01	2.00	E-01	1.00	E-02	
3	2.00	E-02	3.00	E-02	4.00	E-02		
1.00	E-02	1.00	E-02	1.00	E-02			
.50	.20	.30						
1.00	E-06	1.00	E-07	1.00	E-06	1.00	E-07	
1.50	E-06	1.50	E-07	1.50	E-06	1.50	E-07	
2.00	E-06	2.00	E-07	2.00	E-06	2.00	E-07	
2.50	E-06	2.50	E-07	2.50	E-06	2.50	E-07	
3.00	E-06	3.00	E-07	3.00	E-06	3.00	E-07	
3.50	E-06	3.50	E-07	3.50	E-06	3.50	E-07	
4.00	E-06	4.00	E-07	4.00	E-06	4.00	E-07	
4.50	E-06	4.50	E-07	4.50	E-06	4.50	E-07	
6.10	E-01	6.00	E-01					
6.20	E-01	6.00	E-01					
6.30	E-01	6.00	E-01					
6.40	E-01	6.00	E-01					
6.50	E-01	6.00	E-01					
6.60	E-01	6.00	E-01					
6.70	E-01	6.00	E-01					
6.80	E-01	6.00	E-01					
1.50	E-02	1.00	E-02	4.00	E-02	2.00	E-02	
2.00	E-02	1.00	E-02	5.00	E-02	2.00	E-02	
2.50	E-02	1.00	E-02	6.00	E-02	2.00	E-02	
3.00	E-02	1.00	E-02	7.00	E-02	2.00	E-02	
3.50	E-02	1.00	E-02	8.00	E-02	2.00	E-02	
4.00	E-02	1.00	E-02	9.00	E-02	2.00	E-02	
4.50	E-02	1.00	E-02	1.00	E-01	2.00	E-02	
5.00	E-02	1.00	E-02	1.10	E-01	2.00	E-02	
5.00	E-01	1.00	E-01					
5.50	E-01	1.00	E-01					
6.00	E-01	1.00	E-01					
6.50	E-01	1.00	E-01					
7.00	E-01	1.00	E-01					
7.50	E-01	1.00	E-01					
8.00	E-01	1.00	E-01					
8.50	E-01	1.00	E-01					
0.10								

	4	0.20	0.40	0.60	0.80				
TROUT	1.50		E+03	2.00	E-02	1.00	E-02	2.00	E+01 4.00 E-02
BASS	5.00		E+02	3.00	E-02	1.00	E-02	5.00	E+01 8.00 E-02
SALMON	5.00		E+03	2.50	E-02	1.00	E-02	1.50	E+01 2.00 E-01
1.00	E-02	6.00		E+01 4.00	E-03				
2	2.00	E-02	1.00	E-02					
3.00	E-02	5.00	E-02	2.00	E-02	2.50	E-02		
4	3.00	E-02	1.00	E-02					
3.50	E-02	6.00	E-02	2.00	E-02	2.50	E-02		
2.50	E-03	1.50	E+01	5.50	E-03				
5	3.50	E-02	1.00	E-02					
4.00	E-02	5.00	E-02	2.00	E-02	3.00	E-02		
2	2.50	E-02	1.00	E-02	6.00	E-02	2.00	E-02	
3	4.00	E-02	1.00	E-02	9.00	E-02	2.00	E-02	
6	4.50	E-02	1.00	E-02	1.00	E-01	2.00	E-02	
5	4.00	E-02	1.00	E-02	2.00	E-01	2.00	E-02	
3	2.00	E-02	3.00	E-02	4.00	E-02			
1.00	E-02	1.00	E-02	1.00	E-02				
4									
INHALE		CANCER-A		3	1				
1									
ONEHIT									
2.00	E-03								
MULTIHIT									
4.00	E+02	2.00	E+00						
PROBIT									
3.50	E+00	1.50	E+00						
INHALE		CANCER-B		3	2				
1	2								
ONEHIT									
2.00	E-03								
MULTIHIT									
4.00	E+02	2.00	E+00						
PROBIT									
3.50	E+00	1.50	E+00						
INGEST		CANCER-C		2	1				
1									
MULTISTAGE									
3									
3.00	E-04	5.00	E-03	7.00	E-04	4.00	E-03		
CRUMP									
2									
2.50	E-03	6.00	E-04	9.50	E-10				
INGEST		CHRONIC		2	1				
2									
ONEHIT									
8.00	E-07								
MULTIHIT									
5.00	E-01	4.00	E+00						

PROGRAM OUTPUTS

Program outputs are determined by the characteristics of the case. The outputs from the purely hypothetical data set presented in the previous section are presented below. As discussed, these results do not represent an actual situation; they only show how the output would appear for an analysis incorporating all intake modes and exposure pathways.

As the results indicate, the output begins with the four-line case description. Next, the exposure pathways considered in the analysis are summarized. This summary includes a listing of the intake modes and the specific pathways under each mode, including names of aquatic, animal, and vegetable species used in ingestion calculations. Following this, the program prints a summary of the regional population distribution used in the calculations.

After printing the above general case information, the program prints intermediate results and descriptive information for each pollutant species considered. This output begins with a summary of pollutant concentrations by subregion and medium. A summary of exposure rates follows. Exposure rates are tabulated by population group and subregion for each intake mode and specific pathway and are presented in the units discussed earlier.

The final information provided by the program is a summary of the health effects calculations. This includes a description of the health effects considered, including intake mode and pollutant species. Final health effect results, or risks to regional population, are summarized in three tables. The first presents the background level of incidence in the region--the risk associated with the nominal case. The second table gives the level of incidence associated with the case under consideration, including background level effects. The third table shows the net risk--the difference between the first two tables. In each table, results are shown as total incidence of the different health effects in the regional population. These incidence results are tabulated under the specific dose-response models used in making the calculations, to facilitate the comparison of models and parameters.

GENERAL DESCRIPTION OF CASE STUDY TO TEST OPERATION OF PROGRAM
 HYPOTHETICAL DATA EMPLOYING ALL OPTIONS OF EXPOSURE AND HEALTH ROUTINES
 AND TO ILLUSTRATE OUTPUT INFORMATION AND FORMATS
 TEST NUMBER- 1 DATE- 3/14/83 2 POLLUTANTS/4 HEALTH EFFECTS

EXPOSURE MODES AND PATHWAYS CONSIDERED

INHALATION OF GASES AND PARTICULATES
 INGESTION OF PARTICULATES, WATER, AND FOOD PRODUCTS
 SURFACE WATER
 GROUNDWATER
 AQUATIC ORGANISMS - TROUT BASS SALMON
 ANIMAL PRODUCTS - BEEF PORK
 VEGETABLE PRODUCTS - CORN WHEAT SOYBEANS
 DERMAL CONTACT WITH WATER AND ATMOSPHERE

SUBREGION	POPULATION DESCRIPTION FOR RISK ASSESSMENT				TOTAL	CUMULATIVE
	INFANTS	CHILDREN	TEENS	ADULTS		
1	4.46000E+02	4.77100E+03	3.17200E+03	1.78240E+04	2.62130E+04	2.62130E+04
2	1.83000E+02	1.56300E+03	1.30500E+03	7.33400E+03	1.07850E+04	3.69980E+04
3	1.58000E+02	1.68900E+03	1.12300E+03	6.31000E+03	9.28000E+03	4.62780E+04
4	1.22200E+03	1.30800E+04	8.69600E+03	4.88690E+04	7.18670E+04	1.18145E+05
5	3.70000E+02	3.95600E+03	2.63000E+03	1.47800E+04	2.17360E+04	1.39881E+05
6	4.89000E+02	5.23000E+03	3.47700E+03	1.9410E+04	2.87370E+04	1.68618E+05
7	9.90000E+02	1.05970E+04	7.04500E+03	3.95930E+04	5.82250E+04	2.26843E+05
8	1.59000E+02	1.70500E+03	1.13400E+03	6.37000E+03	9.36800E+03	2.36211E+05
GROUP TOTAL	4.01700E+03	4.29910E+04	2.85820E+04	1.60621E+05	2.36211E+05	

ENVIRONMENTAL CONCENTRATIONS FOR SPECIES - SPECIES1

POPULATION SUBREGION	GASES (MG/M3)		PARTICULATES		SURFACE SOIL (MG/KG)		SURFACE WATER (MG/L)		GROUNDWATER (MG/L)	
	CASE	BACKGROUND	CASE	BACKGROUND	CASE	BACKGROUND	CASE	BACKGROUND	CASE	BACKGROUND
1	1.00000E-06	1.00000E-07	1.00000E-06	1.00000E-07	6.10000E-01	6.00000E-01	1.50000E-02	1.00000E-02	4.00000E-02	2.00000E-02
2	1.50000E-06	1.50000E-07	1.50000E-06	1.50000E-07	6.20000E-01	6.00000E-01	2.00000E-02	1.00000E-02	5.00000E-02	2.00000E-02
3	2.00000E-06	2.00000E-07	2.00000E-06	2.00000E-07	6.30000E-01	6.00000E-01	2.50000E-02	1.00000E-02	6.00000E-02	2.00000E-02
4	2.50000E-06	2.50000E-07	2.50000E-06	2.50000E-07	6.40000E-01	6.00000E-01	3.00000E-02	1.00000E-02	7.00000E-02	2.00000E-02
5	3.00000E-06	3.00000E-07	3.00000E-06	3.00000E-07	6.50000E-01	6.00000E-01	3.50000E-02	1.00000E-02	8.00000E-02	2.00000E-02
6	3.50000E-06	3.50000E-07	3.50000E-06	3.50000E-07	6.60000E-01	6.00000E-01	4.00000E-02	1.00000E-02	9.00000E-02	2.00000E-02
7	4.00000E-06	4.00000E-07	4.00000E-06	4.00000E-07	6.70000E-01	6.00000E-01	4.50000E-02	1.00000E-02	1.00000E-01	2.00000E-02
8	4.50000E-06	4.50000E-07	4.50000E-06	4.50000E-07	6.80000E-01	6.00000E-01	5.00000E-02	1.00000E-02	1.10000E-01	2.00000E-02

SUMMARY OF EXPOSURE RATES

EXPOSURE THROUGH INHALATION OF GASES AND PARTICULATES

SUBREGION	PLANT		MEAN PLANT		BACKGROUND		MEAN BACKGROUND	
	PLANT	BACKGROUND	PLANT	BACKGROUND	PLANT	BACKGROUND	PLANT	BACKGROUND
1	2.00000E-06	2.00000E-06	2.00000E-06	2.00000E-06	2.00000E-07	2.00000E-07	2.00000E-07	2.00000E-07
2	3.00000E-06	3.00000E-06	3.00000E-06	3.00000E-06	3.00000E-07	3.00000E-07	3.00000E-07	3.00000E-07
3	4.00000E-06	4.00000E-06	4.00000E-06	4.00000E-06	4.00000E-07	4.00000E-07	4.00000E-07	4.00000E-07
4	5.00000E-06	5.00000E-06	5.00000E-06	5.00000E-06	5.00000E-07	5.00000E-07	5.00000E-07	5.00000E-07
5	6.00000E-06	6.00000E-06	6.00000E-06	6.00000E-06	6.00000E-07	6.00000E-07	6.00000E-07	6.00000E-07
6	7.00000E-06	7.00000E-06	7.00000E-06	7.00000E-06	7.00000E-07	7.00000E-07	7.00000E-07	7.00000E-07
7	8.00000E-06	8.00000E-06	8.00000E-06	8.00000E-06	8.00000E-07	8.00000E-07	8.00000E-07	8.00000E-07
8	9.00000E-06	9.00000E-06	9.00000E-06	9.00000E-06	9.00000E-07	9.00000E-07	9.00000E-07	9.00000E-07

EXPOSURE THROUGH INGESTION PATHWAYS

EXPOSURE THROUGH INGESTION OF AQUATIC ORGANISMS

SUBREGION	EXPOSURE RATE (MG/DAY)				
	CHILDREN	TEENS	ADULTS	INFANTS	BACKGROUND
1	2.13409E-03	6.32955E-03	8.42773E-03	0.0	CHILDREN TEENS ADULTS
2	2.13409E-03	6.32955E-03	8.42773E-03	0.0	8.78605E-04 2.59008E-03 3.45011E-03
3	2.13409E-03	6.32955E-03	8.42773E-03	0.0	8.78605E-04 2.59008E-03 3.45011E-03
4	2.13409E-03	6.32955E-03	8.42773E-03	0.0	8.78605E-04 2.59008E-03 3.45011E-03
5	2.13409E-03	6.32955E-03	8.42773E-03	0.0	8.78605E-04 2.59008E-03 3.45011E-03
6	2.13409E-03	6.32955E-03	8.42773E-03	0.0	8.78605E-04 2.59008E-03 3.45011E-03
7	2.13409E-03	6.32955E-03	8.42773E-03	0.0	8.78605E-04 2.59008E-03 3.45011E-03
8	2.13409E-03	6.32955E-03	8.42773E-03	0.0	8.78605E-04 2.59008E-03 3.45011E-03

EXPOSURE THROUGH INGESTION OF ANIMAL PRODUCTS

SUBREGION	EXPOSURE RATE (MG/DAY)				
	CHILDREN	TEENS	ADULTS	INFANTS	BACKGROUND
1	2.08658E-03	3.36955E-03	5.06838E-03	0.0	CHILDREN TEENS ADULTS
2	2.08658E-03	3.36955E-03	5.06838E-03	0.0	1.03592E-03 1.67605E-03 2.53850E-03
3	2.08658E-03	3.36955E-03	5.06838E-03	0.0	1.03592E-03 1.67605E-03 2.53850E-03
4	2.08658E-03	3.36955E-03	5.06838E-03	0.0	1.03592E-03 1.67605E-03 2.53850E-03
5	2.08658E-03	3.36955E-03	5.06838E-03	0.0	1.03592E-03 1.67605E-03 2.53850E-03
6	2.08658E-03	3.36955E-03	5.06838E-03	0.0	1.03592E-03 1.67605E-03 2.53850E-03
7	2.08658E-03	3.36955E-03	5.06838E-03	0.0	1.03592E-03 1.67605E-03 2.53850E-03
8	2.08658E-03	3.36955E-03	5.06838E-03	0.0	1.03592E-03 1.67605E-03 2.53850E-03

EXPOSURE THROUGH INGESTION OF VEGETABLE PRODUCTS

SUBREGION	EXPOSURE RATE (MG/DAY)				
	CHILDREN	TEENS	ADULTS	INFANTS	BACKGROUND
1	9.27925E-02	1.48065E-01	1.30093E-01	0.0	CHILDREN TEENS ADULTS
2	9.27925E-02	1.48065E-01	1.30093E-01	0.0	2.10818E-02 3.33669E-02 2.95368E-02
3	9.27925E-02	1.48065E-01	1.30093E-01	0.0	2.10818E-02 3.33669E-02 2.95368E-02
4	9.27925E-02	1.48065E-01	1.30093E-01	0.0	2.10818E-02 3.33669E-02 2.95368E-02
5	9.27925E-02	1.48065E-01	1.30093E-01	0.0	2.10818E-02 3.33669E-02 2.95368E-02
6	9.27925E-02	1.48065E-01	1.30093E-01	0.0	2.10818E-02 3.33669E-02 2.95368E-02
7	9.27925E-02	1.48065E-01	1.30093E-01	0.0	2.10818E-02 3.33669E-02 2.95368E-02
8	9.27925E-02	1.48065E-01	1.30093E-01	0.0	2.10818E-02 3.33669E-02 2.95368E-02

EXPOSURE THROUGH INGESTION OF DRINKING WATER

SUBREGION	EXPOSURE RATE (MG/DAY)											
	PLANT				BACKGROUND				TEENS			
	INFANTS	CHILDREN	TEENS	ADULTS	INFANTS	CHILDREN	TEENS	ADULTS	INFANTS	CHILDREN	TEENS	ADULTS
1	9.9000E-03	1.2780E-02	1.2780E-02	1.8180E-02	5.5000E-03	7.0999E-03	7.0999E-03	7.0999E-03	5.5000E-03	7.0999E-03	7.0999E-03	7.0999E-03
2	4.6750E-03	6.0349E-03	6.0349E-03	8.5849E-03	2.2000E-03	2.8400E-03	2.8400E-03	2.8400E-03	2.2000E-03	2.8400E-03	2.8400E-03	2.8400E-03
3	2.1175E-02	2.7335E-02	2.7335E-02	3.8885E-02	7.1500E-03	9.2300E-03	9.2300E-03	9.2300E-03	7.1500E-03	9.2300E-03	9.2300E-03	9.2300E-03
4	1.8260E-02	2.3572E-02	2.3572E-02	3.3532E-02	5.5000E-03	7.0999E-03	7.0999E-03	7.0999E-03	5.5000E-03	7.0999E-03	7.0999E-03	7.0999E-03
5	1.6280E-02	2.1016E-02	2.1016E-02	2.9896E-02	4.4000E-03	5.6799E-03	5.6799E-03	5.6799E-03	4.4000E-03	5.6799E-03	5.6799E-03	5.6799E-03
6	1.3200E-02	1.7040E-02	1.7040E-02	2.4240E-02	3.3000E-03	4.2600E-03	4.2600E-03	4.2600E-03	3.3000E-03	4.2600E-03	4.2600E-03	4.2600E-03
7	1.4850E-02	1.9170E-02	1.9170E-02	2.7270E-02	3.3000E-03	4.2600E-03	4.2600E-03	4.2600E-03	3.3000E-03	4.2600E-03	4.2600E-03	4.2600E-03
8	3.2450E-02	4.1890E-02	4.1890E-02	5.9590E-02	6.0500E-03	7.8100E-03	7.8100E-03	7.8100E-03	6.0500E-03	7.8100E-03	7.8100E-03	7.8100E-03

EXPOSURE THROUGH INGESTION OF PARTICULATES

SUBREGION	EXPOSURE RATE (MG/DAY)											
	PLANT				BACKGROUND				ADULTS			
	INFANTS	CHILDREN	TEENS	ADULTS	INFANTS	CHILDREN	TEENS	ADULTS	INFANTS	CHILDREN	TEENS	ADULTS
1	9.6000E-07	2.5350E-06	5.4800E-06	5.4800E-06	9.6000E-08	2.5350E-07	5.4800E-07	5.4800E-07	9.6000E-08	2.5350E-07	5.4800E-07	5.4800E-07
2	1.4400E-06	3.8025E-06	8.2200E-06	8.2200E-06	1.4400E-07	3.8025E-07	8.2200E-07	8.2200E-07	1.4400E-07	3.8025E-07	8.2200E-07	8.2200E-07
3	1.9200E-06	5.0700E-06	1.0960E-05	1.0960E-05	1.9200E-07	5.0700E-07	1.0960E-06	1.0960E-06	1.9200E-07	5.0700E-07	1.0960E-06	1.0960E-06
4	2.4000E-06	6.3375E-06	1.3700E-05	1.3700E-05	2.4000E-07	6.3375E-07	1.3700E-06	1.3700E-06	2.4000E-07	6.3375E-07	1.3700E-06	1.3700E-06
5	2.8800E-06	7.6050E-06	1.6440E-05	1.6440E-05	2.8800E-07	7.6050E-07	1.6440E-06	1.6440E-06	2.8800E-07	7.6050E-07	1.6440E-06	1.6440E-06
6	3.3600E-06	8.8725E-06	1.9180E-05	1.9180E-05	3.3600E-07	8.8725E-07	1.9180E-06	1.9180E-06	3.3600E-07	8.8725E-07	1.9180E-06	1.9180E-06
7	3.8400E-06	1.0140E-05	2.1920E-05	2.1920E-05	3.8400E-07	1.0140E-06	2.1920E-06	2.1920E-06	3.8400E-07	1.0140E-06	2.1920E-06	2.1920E-06
8	4.3200E-06	1.14075E-05	2.4660E-05	2.4660E-05	4.3200E-07	1.14075E-06	2.4660E-06	2.4660E-06	4.3200E-07	1.14075E-06	2.4660E-06	2.4660E-06

SUMMARY CALCULATIONS FOR INGESTION EXPOSURE

SUBREGION	PLANT				EXPOSURE RATE (MG/DAY)				BACKGROUND			
	INFANTS	CHILDREN	TEENS	ADULTS	MEAN	INFANTS	CHILDREN	TEENS	ADULTS	MEAN		
1	1.15510E-02	1.12724E-01	1.74014E-01	1.64518E-01	1.53638E-01	7.15009E-03	3.30248F-02	4.81980E-02	4.83697E-02	4.48547E-02		
2	6.32643E-03	1.05980E-01	1.67271E-01	1.54926E-01	1.44990E-01	3.85014E-03	2.87650E-02	4.39382E-02	4.23100E-02	3.93890E-02		
3	2.28269E-02	1.27281E-01	1.88571E-01	1.85229E-01	1.72322E-01	8.80019E-03	3.30252E-02	5.03285E-02	5.14003E-02	4.75885E-02		
4	1.99124E-02	1.23520E-01	1.84814E-01	1.79879E-01	1.67498E-01	7.15023E-03	3.30252E-02	4.81983E-02	4.83705E-02	4.48560E-02		
5	1.79329E-02	1.20965E-01	1.82261E-01	1.76245E-01	1.64217E-01	6.05028E-03	3.16053E-02	4.67791E-02	4.63508E-02	4.30329E-02		
6	1.48534E-02	1.16990E-01	1.78287E-01	1.70592E-01	1.59118E-01	4.95033E-03	3.01855E-02	4.53593E-02	4.43311E-02	4.12109E-02		
7	1.65038E-02	1.19121E-01	1.80420E-01	1.73625E-01	1.61856E-01	4.95038E-03	3.01856E-02	4.53596E-02	4.43314E-02	4.12116E-02		
8	3.41043E-02	1.41843E-01	2.03143E-01	2.05947E-01	1.91024E-01	7.70042E-03	3.37357E-02	4.89099E-02	4.93816E-02	4.57695E-02		

EXPOSURE THROUGH DERMAL CONTACT PATHWAYS EXPOSURE THROUGH DERMAL CONTACT WITH ATMOSPHERIC PARTICULATES AND GASES

SUBREGION	EXPOSURE RATE (MG/M3)									
	INFANTS	CHILDREN	TEENS	ADULTS	PLANT	TEENS	ADULTS	INFANTS	CHILDREN	TEENS
1	1.02740E-06	1.23288E-06	1.43835E-06	1.23288E-06	1.02740E-07	1.23288E-07	1.43835E-07	1.02740E-07	1.23288E-07	1.43835E-07
2	1.54109E-06	1.84931E-06	2.15753E-06	1.84931E-06	1.54110E-07	1.84931E-07	2.15753E-07	1.54110E-07	1.84931E-07	2.15753E-07
3	2.05479E-06	2.46575E-06	2.87671E-06	2.46575E-06	2.05479E-07	2.46575E-07	2.87671E-07	2.05479E-07	2.46575E-07	2.87671E-07
4	2.56849E-06	3.08219E-06	3.59589E-06	3.08219E-06	2.56849E-07	3.08219E-07	3.59589E-07	2.56849E-07	3.08219E-07	3.59589E-07
5	3.08219E-06	3.69863E-06	4.31507E-06	3.69863E-06	3.08219E-07	3.69863E-07	4.31507E-07	3.08219E-07	3.69863E-07	4.31507E-07
6	3.59589E-06	4.31507E-06	5.03425E-06	4.31507E-06	3.59589E-07	4.31507E-07	5.03425E-07	3.59589E-07	4.31507E-07	5.03425E-07
7	4.10959E-06	4.93151E-06	5.75342E-06	4.93151E-06	4.10959E-07	4.93151E-07	5.75342E-07	4.10959E-07	4.93151E-07	5.75342E-07
8	4.62329E-06	5.54794E-06	6.47260E-06	5.54794E-06	4.62329E-07	5.54794E-07	6.47260E-07	4.62329E-07	5.54794E-07	6.47260E-07

EXPOSURE THROUGH DERMAL CONTACT WITH RECREATIONAL WATER

SUBREGION	EXPOSURE RATE (MG/M3)									
	INFANTS	CHILDREN	TEENS	ADULTS	PLANT	TEENS	ADULTS	INFANTS	CHILDREN	TEENS
1	6.39269E-04	1.27854E-02	3.19634E-02	1.27854E-02	6.39269E-04	1.27854E-02	3.19634E-02	6.39269E-04	1.27854E-02	3.19634E-02
2	6.39269E-04	1.27854E-02	3.19634E-02	1.27854E-02	6.39269E-04	1.27854E-02	3.19634E-02	6.39269E-04	1.27854E-02	3.19634E-02
3	6.39269E-04	1.27854E-02	3.19634E-02	1.27854E-02	6.39269E-04	1.27854E-02	3.19634E-02	6.39269E-04	1.27854E-02	3.19634E-02
4	6.39269E-04	1.27854E-02	3.19634E-02	1.27854E-02	6.39269E-04	1.27854E-02	3.19634E-02	6.39269E-04	1.27854E-02	3.19634E-02
5	6.39269E-04	1.27854E-02	3.19634E-02	1.27854E-02	6.39269E-04	1.27854E-02	3.19634E-02	6.39269E-04	1.27854E-02	3.19634E-02
6	6.39269E-04	1.27854E-02	3.19634E-02	1.27854E-02	6.39269E-04	1.27854E-02	3.19634E-02	6.39269E-04	1.27854E-02	3.19634E-02
7	6.39269E-04	1.27854E-02	3.19634E-02	1.27854E-02	6.39269E-04	1.27854E-02	3.19634E-02	6.39269E-04	1.27854E-02	3.19634E-02
8	6.39269E-04	1.27854E-02	3.19634E-02	1.27854E-02	6.39269E-04	1.27854E-02	3.19634E-02	6.39269E-04	1.27854E-02	3.19634E-02

EXPOSURE THROUGH DERMAL CONTACT WITH DOMESTIC WATER

SUBREGION	EXPOSURE RATE (MG/M3)									
	INFANTS	CHILDREN	TEENS	ADULTS	PLANT	TEENS	ADULTS	INFANTS	CHILDREN	TEENS
1	5.65068E-02	3.59589E-02	5.13628E-02	6.16438E-02	5.65068E-02	3.59589E-02	5.13628E-02	5.65068E-02	3.59589E-02	5.13628E-02
2	6.6838E-02	4.18968E-02	5.62236E-02	6.16438E-02	6.6838E-02	4.18968E-02	5.62236E-02	6.6838E-02	4.18968E-02	5.62236E-02
3	1.20862E-01	7.69120E-02	1.09874E-01	9.10966E-02	1.20862E-01	7.69120E-02	1.09874E-01	1.20862E-01	7.69120E-02	1.09874E-01
4	1.04224E-01	6.63242E-02	9.47488E-02	1.13699E-01	1.04224E-01	6.63242E-02	9.47488E-02	1.04224E-01	6.63242E-02	9.47488E-02
5	9.29223E-02	5.91324E-02	8.44748E-02	1.01370E-01	9.29223E-02	5.91324E-02	8.44748E-02	9.29223E-02	5.91324E-02	8.44748E-02
6	7.53424E-02	4.79452E-02	6.84931E-02	8.21918E-02	7.53424E-02	4.79452E-02	6.84931E-02	7.53424E-02	4.79452E-02	6.84931E-02
7	8.47602E-02	5.39383E-02	7.70547E-02	9.24657E-02	8.47602E-02	5.39383E-02	7.70547E-02	8.47602E-02	5.39383E-02	7.70547E-02
8	1.85217E-01	1.17865E-01	1.68379E-01	2.02055E-01	1.85217E-01	1.17865E-01	1.68379E-01	1.85217E-01	1.17865E-01	1.68379E-01

SUMMARY OF DERMAL CONTACT EXPOSURE RATES

SUBREGION	EXPOSURE RATE (MG/M3)									
	INFANTS	CHILDREN	TEENS	ADULTS	PLANT	TEENS	ADULTS	INFANTS	CHILDREN	TEENS
1	5.71471E-02	4.87455E-02	8.33347E-02	7.44304E-02	5.71471E-02	4.87455E-02	8.33347E-02	5.71471E-02	4.87455E-02	8.33347E-02
2	2.73246E-02	2.97678E-02	5.62236E-02	4.18968E-02	2.73246E-02	2.97678E-02	5.62236E-02	2.73246E-02	2.97678E-02	5.62236E-02
3	1.21503E-01	8.96999E-02	1.41841E-01	1.33906E-01	1.21503E-01	8.96999E-02	1.41841E-01	1.21503E-01	8.96999E-02	1.41841E-01
4	1.04865E-01	7.91126E-02	1.26716E-01	1.26487E-01	1.04865E-01	7.91126E-02	1.26716E-01	1.04865E-01	7.91126E-02	1.26487E-01
5	9.35646E-02	7.19214E-02	1.16443E-01	1.14159E-01	9.35646E-02	7.19214E-02	1.16443E-01	9.35646E-02	7.19214E-02	1.16443E-01
6	7.59853E-02	6.07349E-02	1.00462E-01	9.49814E-02	7.59853E-02	6.07349E-02	1.00462E-01	7.59853E-02	6.07349E-02	1.00462E-01
7	8.54036E-02	6.67287E-02	1.09024E-01	1.05256E-01	8.54036E-02	6.67287E-02	1.09024E-01	8.54036E-02	6.67287E-02	1.09024E-01
8	1.85861E-01	1.30656E-01	2.00349E-01	2.14846E-01	1.85861E-01	1.30656E-01	2.00349E-01	1.85861E-01	1.30656E-01	2.00349E-01

ENVIRONMENTAL CONCENTRATIONS FOR SPECIES - SPECIES2

POPULATION SUBREGION	GASES (MG/M3)		ATMOSPHERIC (MG/M3)		PARTICULATES		SURFACE SOIL (MG/KG)		SURFACE WATER (MG/L)		GROUNDWATER (MG/L)	
	CASE	BACKGROUND	CASE	BACKGROUND	CASE	BACKGROUND	CASE	BACKGROUND	CASE	BACKGROUND	CASE	BACKGROUND
1	1.00000E-06	1.00000E-07	1.00000E-07	1.00000E-06	1.00000E-06	1.00000E-07	6.10000E-01	6.00000E-01	1.50000E-02	1.00000E-02	4.00000E-02	2.00000E-02
2	1.50000E-06	1.50000E-07	1.50000E-07	1.50000E-06	1.50000E-06	1.50000E-07	6.20000E-01	6.00000E-01	2.00000E-02	1.00000E-02	5.00000E-02	2.00000E-02
3	2.00000E-06	2.00000E-07	2.00000E-07	2.00000E-06	2.00000E-06	2.00000E-07	6.30000E-01	6.00000E-01	2.50000E-02	1.00000E-02	6.00000E-02	2.00000E-02
4	2.50000E-06	2.50000E-07	2.50000E-07	2.50000E-06	2.50000E-06	2.50000E-07	6.40000E-01	6.00000E-01	3.00000E-02	1.00000E-02	7.00000E-02	2.00000E-02
5	3.00000E-06	3.00000E-07	3.00000E-07	3.00000E-06	3.00000E-06	3.00000E-07	6.50000E-01	6.00000E-01	3.50000E-02	1.00000E-02	8.00000E-02	2.00000E-02
6	3.50000E-06	3.50000E-07	3.50000E-07	3.50000E-06	3.50000E-06	3.50000E-07	6.60000E-01	6.00000E-01	4.00000E-02	1.00000E-02	9.00000E-02	2.00000E-02
7	4.00000E-06	4.00000E-07	4.00000E-07	4.00000E-06	4.00000E-06	4.00000E-07	6.70000E-01	6.00000E-01	4.50000E-02	1.00000E-02	1.00000E-01	2.00000E-02
8	4.50000E-06	4.50000E-07	4.50000E-07	4.50000E-06	4.50000E-06	4.50000E-07	6.80000E-01	6.00000E-01	5.00000E-02	1.00000E-02	1.10000E-01	2.00000E-02

SUMMARY OF EXPOSURE RATES

EXPOSURE THROUGH INHALATION OF GASES AND PARTICULATES

SUBREGION	PLANT		MEAN PLANT		BACKGROUND		MEAN BACKGROUND	
	CASE	BACKGROUND	CASE	BACKGROUND	CASE	BACKGROUND	CASE	BACKGROUND
1	2.00000E-06	2.00000E-06	2.00000E-06	2.00000E-06	2.00000E-07	2.00000E-07	2.00000E-07	2.00000E-07
2	3.00000E-06	3.00000E-06	3.00000E-06	3.00000E-06	3.00000E-07	3.00000E-07	3.00000E-07	3.00000E-07
3	4.00000E-06	4.00000E-06	4.00000E-06	4.00000E-06	4.00000E-07	4.00000E-07	4.00000E-07	4.00000E-07
4	5.00000E-06	5.00000E-06	5.00000E-06	5.00000E-06	5.00000E-07	5.00000E-07	5.00000E-07	5.00000E-07
5	6.00000E-06	6.00000E-06	6.00000E-06	6.00000E-06	6.00000E-07	6.00000E-07	6.00000E-07	6.00000E-07
6	7.00000E-06	7.00000E-06	7.00000E-06	7.00000E-06	7.00000E-07	7.00000E-07	7.00000E-07	7.00000E-07
7	8.00000E-06	8.00000E-06	8.00000E-06	8.00000E-06	8.00000E-07	8.00000E-07	8.00000E-07	8.00000E-07
8	9.00000E-06	9.00000E-06	9.00000E-06	9.00000E-06	9.00000E-07	9.00000E-07	9.00000E-07	9.00000E-07

EXPOSURE THROUGH INGESTION PATHWAYS

EXPOSURE THROUGH INGESTION OF AQUATIC ORGANISMS

SUBREGION	EXPOSURE RATE (MG/DAY)			
	CHILDREN	TEENS	ADULTS	INFANTS
1	2.13409E-03	6.32955E-03	8.42773E-03	0.0
2	2.13409E-03	6.32955E-03	8.42773E-03	0.0
3	2.13409E-03	6.32955E-03	8.42773E-03	0.0
4	2.13409E-03	6.32955E-03	8.42773E-03	0.0
5	2.13409E-03	6.32955E-03	8.42773E-03	0.0
6	2.13409E-03	6.32955E-03	8.42773E-03	0.0
7	2.13409E-03	6.32955E-03	8.42773E-03	0.0
8	2.13409E-03	6.32955E-03	8.42773E-03	0.0

BACKGROUND		
CHILDREN	TEENS	ADULTS
8.78605E-04	2.59008E-03	3.45011E-03
8.78605E-04	2.59008E-03	3.45011E-03
8.78605E-04	2.59008E-03	3.45011E-03
8.78605E-04	2.59008E-03	3.45011E-03
8.78605E-04	2.59008E-03	3.45011E-03
8.78605E-04	2.59008E-03	3.45011E-03
8.78605E-04	2.59008E-03	3.45011E-03

EXPOSURE THROUGH INGESTION OF ANIMAL PRODUCTS

SUBREGION	EXPOSURE RATE (MG/DAY)			
	CHILDREN	TEENS	ADULTS	INFANTS
1	2.08658E-03	3.36955E-03	5.06838E-03	0.0
2	2.08658E-03	3.36955E-03	5.06838E-03	0.0
3	2.08658E-03	3.36955E-03	5.06838E-03	0.0
4	2.08658E-03	3.36955E-03	5.06838E-03	0.0
5	2.08658E-03	3.36955E-03	5.06838E-03	0.0
6	2.08658E-03	3.36955E-03	5.06838E-03	0.0
7	2.08658E-03	3.36955E-03	5.06838E-03	0.0
8	2.08658E-03	3.36955E-03	5.06838E-03	0.0

BACKGROUND		
CHILDREN	TEENS	ADULTS
1.03592E-03	1.67605E-03	2.53850E-03
1.03592E-03	1.67605E-03	2.53850E-03
1.03592E-03	1.67605E-03	2.53850E-03
1.03592E-03	1.67605E-03	2.53850E-03
1.03592E-03	1.67605E-03	2.53850E-03
1.03592E-03	1.67605E-03	2.53850E-03
1.03592E-03	1.67605E-03	2.53850E-03

EXPOSURE THROUGH INGESTION OF VEGETABLE PRODUCTS

SUBREGION	EXPOSURE RATE (MG/DAY)			
	CHILDREN	TEENS	ADULTS	INFANTS
1	9.27925E-02	1.48065E-01	1.30093E-01	0.0
2	9.27925E-02	1.48065E-01	1.30093E-01	0.0
3	9.27925E-02	1.48065E-01	1.30093E-01	0.0
4	9.27925E-02	1.48065E-01	1.30093E-01	0.0
5	9.27925E-02	1.48065E-01	1.30093E-01	0.0
6	9.27925E-02	1.48065E-01	1.30093E-01	0.0
7	9.27925E-02	1.48065E-01	1.30093E-01	0.0
8	9.27925E-02	1.48065E-01	1.30093E-01	0.0

BACKGROUND		
CHILDREN	TEENS	ADULTS
2.10818E-02	3.33669E-02	2.95368E-02
2.10818E-02	3.33669E-02	2.95368E-02
2.10818E-02	3.33669E-02	2.95368E-02
2.10818E-02	3.33669E-02	2.95368E-02
2.10818E-02	3.33669E-02	2.95368E-02
2.10818E-02	3.33669E-02	2.95368E-02
2.10818E-02	3.33669E-02	2.95368E-02

EXPOSURE THROUGH INGESTION OF DRINKING WATER

SUBREGION	EXPOSURE RATE (MG/DAY)									
	PLANT		CHILDREN		TEENS		ADULTS		BACKGROUND	
	INFANTS	CHILDREN	TEENS	ADULTS	INFANTS	CHILDREN	TEENS	ADULTS	INFANTS	CHILDREN
1	9.9000E-03	1.2780E-02	1.2780E-02	1.8180E-02	5.5000E-03	7.0999E-03	7.0999E-03	1.0100E-02	5.5000E-03	7.0999E-03
2	4.6750E-03	6.0349E-03	6.0349E-03	8.5849E-03	2.2000E-03	2.8400E-03	2.8400E-03	4.0400E-03	2.2000E-03	2.8400E-03
3	2.1175E-02	2.7335E-02	2.7335E-02	3.8885E-02	7.1500E-03	9.2300E-03	9.2300E-03	1.3100E-02	7.1500E-03	9.2300E-03
4	1.8260E-02	2.3572E-02	2.3572E-02	3.3532E-02	5.5000E-03	7.0999E-03	7.0999E-03	1.0100E-02	5.5000E-03	7.0999E-03
5	1.6280E-02	2.1016E-02	2.1016E-02	2.9896E-02	4.4000E-03	5.6799E-03	5.6799E-03	8.0800E-03	4.4000E-03	5.6799E-03
6	1.3200E-02	1.7040E-02	1.7040E-02	2.4240E-02	3.3000E-03	4.2600E-03	4.2600E-03	6.0600E-03	3.3000E-03	4.2600E-03
7	1.4850E-02	1.9170E-02	1.9170E-02	2.7270E-02	3.3000E-03	4.2600E-03	4.2600E-03	6.0600E-03	3.3000E-03	4.2600E-03
8	3.2450E-02	4.1890E-02	4.1890E-02	5.9590E-02	6.0500E-03	7.8100E-03	7.8100E-03	1.1100E-02	6.0500E-03	7.8100E-03

EXPOSURE THROUGH INGESTION OF PARTICULATES

SUBREGION	EXPOSURE RATE (MG/DAY)									
	PLANT		CHILDREN		TEENS		ADULTS		BACKGROUND	
	INFANTS	CHILDREN	TEENS	ADULTS	INFANTS	CHILDREN	TEENS	ADULTS	INFANTS	CHILDREN
1	9.6000E-07	2.5350E-06	5.4800E-06	5.4800E-06	9.6000E-08	2.5350E-07	5.4800E-07	5.4800E-07	9.6000E-08	2.5350E-07
2	1.4400E-06	3.8025E-06	8.2200E-06	8.2200E-06	1.4400E-07	3.8025E-07	8.2200E-07	8.2200E-07	1.4400E-07	3.8025E-07
3	1.9200E-06	5.0700E-06	1.0960E-05	1.0960E-05	1.9200E-07	5.0700E-07	1.0960E-06	1.0960E-06	1.9200E-07	5.0700E-07
4	2.4000E-06	6.3375E-06	1.3700E-05	1.3700E-05	2.4000E-07	6.3375E-07	1.3700E-06	1.3700E-06	2.4000E-07	6.3375E-07
5	2.8800E-06	7.6050E-06	1.6440E-05	1.6440E-05	2.8800E-07	7.6050E-07	1.6440E-06	1.6440E-06	2.8800E-07	7.6050E-07
6	3.3600E-06	8.8725E-06	1.9180E-05	1.9180E-05	3.3600E-07	8.8725E-07	1.9180E-06	1.9180E-06	3.3600E-07	8.8725E-07
7	3.8400E-06	1.0140E-05	2.1920E-05	2.1920E-05	3.8400E-07	1.0140E-06	2.1920E-06	2.1920E-06	3.8400E-07	1.0140E-06
8	4.3200E-06	1.1407E-05	2.4660E-05	2.4660E-05	4.3200E-07	1.1407E-06	2.4660E-06	2.4660E-06	4.3200E-07	1.1407E-06

SUMMARY CALCULATIONS FOR INGESTION EXPOSURE

SUBREGION	EXPOSURE RATE (MG/DAY)									
	PLANT		CHILDREN		TEENS		ADULTS		BACKGROUND	
	INFANTS	CHILDREN	TEENS	ADULTS	INFANTS	CHILDREN	TEENS	ADULTS	INFANTS	CHILDREN
1	1.1551E-02	1.1272E-01	1.7401E-01	1.6451E-01	7.1500E-03	3.3024E-02	4.8198E-02	4.8367E-02	5.5000E-03	7.0999E-03
2	6.3264E-03	1.0598E-01	1.6727E-01	1.5492E-01	3.8501E-03	2.8765E-02	4.3938E-02	4.2310E-02	2.2000E-03	2.8400E-03
3	2.2826E-02	1.2728E-01	1.8857E-01	1.8522E-01	8.8001E-03	3.5155E-02	5.0328E-02	5.1400E-02	7.1500E-03	9.2300E-03
4	1.9912E-02	1.2352E-01	1.8481E-01	1.7987E-01	7.1502E-03	3.3025E-02	4.8198E-02	4.8370E-02	5.5000E-03	7.0999E-03
5	1.7932E-02	1.2096E-01	1.8226E-01	1.7624E-01	6.0502E-03	3.1605E-02	4.6779E-02	4.6350E-02	4.4000E-03	5.6799E-03
6	1.4853E-02	1.1699E-01	1.7828E-01	1.5911E-01	4.9503E-03	3.0185E-02	4.5359E-02	4.4331E-02	3.3000E-03	4.2600E-03
7	1.6503E-02	1.1912E-01	1.8042E-01	1.7362E-01	4.9503E-03	3.0185E-02	4.5359E-02	4.4331E-02	3.3000E-03	4.2600E-03
8	3.4104E-02	1.4184E-01	2.0314E-01	2.0594E-01	7.7004E-03	3.3735E-02	4.8909E-02	4.9381E-02	6.0500E-03	7.8100E-03

EXPOSURE THROUGH DERMAL CONTACT PATHWAYS

EXPOSURE THROUGH DERMAL CONTACT WITH ATMOSPHERIC PARTICULATES AND GASES

SUBREGION	EXPOSURE RATE (MG/M3)				
	INFANTS	CHILDREN	TEENS	ADULTS	PIANT
1	1.02740E-06	1.23288E-06	1.43835E-06	1.23288E-06	1.02740E-07
2	1.54109E-06	1.84931E-06	2.15753E-06	1.84931E-06	1.54109E-07
3	2.05479E-06	2.46575E-06	2.87671E-06	2.46575E-06	2.05479E-07
4	2.56849E-06	3.08219E-06	3.59589E-06	3.08219E-06	2.56849E-07
5	3.08219E-06	3.69863E-06	4.31507E-06	3.69863E-06	3.08219E-07
6	3.59589E-06	4.31507E-06	5.03425E-06	4.31507E-06	3.59589E-07
7	4.10959E-06	4.93151E-06	5.75342E-06	4.93151E-06	4.10959E-07
8	4.62329E-06	5.54794E-06	6.47260E-06	5.54794E-06	4.62329E-07

EXPOSURE THROUGH DERMAL CONTACT WITH RECREATIONAL WATER

SUBREGION	EXPOSURE RATE (MG/M3)				
	INFANTS	CHILDREN	TEENS	ADULTS	PIANT
1	6.39269E-04	1.27854E-02	3.19634E-02	1.27854E-02	6.39269E-04
2	6.39269E-04	1.27854E-02	3.19634E-02	1.27854E-02	6.39269E-04
3	6.39269E-04	1.27854E-02	3.19634E-02	1.27854E-02	6.39269E-04
4	6.39269E-04	1.27854E-02	3.19634E-02	1.27854E-02	6.39269E-04
5	6.39269E-04	1.27854E-02	3.19634E-02	1.27854E-02	6.39269E-04
6	6.39269E-04	1.27854E-02	3.19634E-02	1.27854E-02	6.39269E-04
7	6.39269E-04	1.27854E-02	3.19634E-02	1.27854E-02	6.39269E-04
8	6.39269E-04	1.27854E-02	3.19634E-02	1.27854E-02	6.39269E-04

EXPOSURE THROUGH DERMAL CONTACT WITH DOMESTIC WATER

SUBREGION	EXPOSURE RATE (MG/M3)				
	INFANTS	CHILDREN	TEENS	ADULTS	PIANT
1	5.65068E-02	3.59589E-02	5.13698E-02	6.16438E-02	5.65068E-02
2	6.66838E-02	4.19806E-02	5.92580E-02	7.01096E-02	6.66838E-02
3	1.20862E-01	7.69120E-02	1.09874E-01	1.31849E-01	1.20862E-01
4	1.04224E-01	6.63242E-02	9.47488E-02	1.13699E-01	1.04224E-01
5	9.29223E-02	5.91324E-02	8.44748E-02	1.01370E-01	9.29223E-02
6	7.53424E-02	4.79452E-02	6.84931E-02	8.21918E-02	7.53424E-02
7	8.47602E-02	5.39383E-02	7.70347E-02	9.24657E-02	8.47602E-02
8	1.85217E-01	1.17865E-01	1.68379E-01	2.02055E-01	1.85217E-01

SUMMARY OF DERMAL CONTACT EXPOSURE RATES

SUBREGION	EXPOSURE RATE (MG/M3)				
	INFANTS	CHILDREN	TEENS	ADULTS	PIANT
1	5.71471E-02	4.87455E-02	8.33347E-02	7.44304E-02	5.71471E-02
2	2.73246E-02	2.97678E-02	5.62236E-02	4.18968E-02	2.73246E-02
3	1.21503E-01	8.96999E-02	1.41841E-01	1.44637E-01	1.21503E-01
4	1.04865E-01	7.91126E-02	1.26716E-01	1.37525E-01	1.04865E-01
5	9.35646E-02	7.19214E-02	1.16443E-01	1.14159E-01	9.35646E-02
6	7.59853E-02	6.07349E-02	1.00462E-01	9.49814E-02	7.59853E-02
7	8.54036E-02	6.67287E-02	1.09024E-01	1.05256E-01	8.54036E-02
8	1.85861E-01	1.30656E-01	2.00349E-01	2.14846E-01	1.85861E-01

SUMMARY OF HEALTH EFFECTS

HEALTH EFFECT	INTAKE MODE		CHEMICAL SPECIES			
	INITIALE	INITIALE	SPECIES1	SPECIES2		
CANCER-A	INITIALE		SPECIES1			
CANCER-B	INGEST		SPECIES1			
CANCER-C	INGEST		SPECIES2			
CHRONIC			SPECIES1			
			SPECIES2			
			SPECIES2			
BACKGROUND LEVEL OF INCIDENCE IN REGIONAL POPULATION--EXPOSURE OVER PLANT LIFETIME						
HEALTH EFFECT	ONE HIT	MULTIHIT	DOSE-RESPONSE RELATIONSHIPS		PROBIT	WEIBULL
			MULTISTAGE			
			CRUMP			
CANCER-A	0.32165E-03	0.98258E-02	0.0	0.0	0.19315E-02	0.0
CANCER-B	0.35381E-03	0.11889E-01	0.0	0.0	0.27239E-02	0.0
CANCER-C	0.0	0.0	0.11811E+02	0.59011E+03	0.0	0.0
CHRONIC	0.42611E-03	0.16110E-07	0.0	0.0	0.0	0.0
LEVEL OF INCIDENCE IN REGIONAL POPULATION WITH PLANT--EXPOSURE OVER PLANT LIFETIME						
HEALTH EFFECT	ONE HIT	MULTIHIT	DOSE-RESPONSE RELATIONSHIPS		PROBIT	WEIBULL
			MULTISTAGE			
			CRUMP			
CANCER-A	0.17289E-02	0.28360E+00	0.0	0.0	0.53445E+00	0.0
CANCER-B	0.19018E-02	0.34312E+00	0.0	0.0	0.71336E+00	0.0
CANCER-C	0.0	0.0	0.11811E+02	0.59054E+03	0.0	0.0
CHRONIC	0.10011E-02	0.48983E-06	0.0	0.0	0.0	0.0
INCREASED INCIDENCE OF HEALTH EFFECTS IN REGIONAL POPULATION--EXPOSURE OVER PLANT LIFETIME						
HEALTH EFFECT	ONE HIT	MULTIHIT	DOSE-RESPONSE RELATIONSHIPS		PROBIT	WEIBULL
			MULTISTAGE			
			CRUMP			
CANCER-A	0.14072E-02	0.27378E+00	0.0	0.0	0.53251E+00	0.0
CANCER-B	0.15479E-02	0.33123E+00	0.0	0.0	0.71063E+00	0.0
CANCER-C	0.0	0.0	0.37193E-03	0.43018E+00	0.0	0.0
CHRONIC	0.57496E-03	0.47371E-06	0.0	0.0	0.0	0.0

BACKGROUND LEVEL OF INCIDENCE IN REGIONAL POPULATION--EXPOSURE OVER HUMAN LIFETIME

HEALTH EFFECT	ONE HIT	MULTIHIT	DOSE-RESPONSE RELATIONSHIPS MULTISTAGE	GRUMP	PROBIT	WEIBULL
CANCER-A	0.32165E-03	0.98258E-02	0.0	0.0	0.19315E-02	0.0
CANCER-B	0.35381E-03	0.11889E-01	0.0	0.0	0.27239E-02	0.0
CANCER-C	0.0	0.0	0.11811E+02	0.59011E+03	0.0	0.0
CHRONIC	0.42611E-03	0.16110E-07	0.0	0.0	0.0	0.0

LEVEL OF INCIDENCE IN REGIONAL POPULATION WITH PLANT--EXPOSURE OVER HUMAN LIFETIME

HEALTH EFFECT	ONE HIT	MULTIHIT	DOSE-RESPONSE RELATIONSHIPS MULTISTAGE	GRUMP	PROBIT	WEIBULL
CANCER-A	0.32165E-02	0.98065E+00	0.0	0.0	0.32976E+01	0.0
CANCER-B	0.35381E-02	0.11863E+01	0.0	0.0	0.43044E+01	0.0
CANCER-C	0.0	0.0	0.11812E+02	0.59099E+03	0.0	0.0
CHRONIC	0.16089E-02	0.32725E-05	0.0	0.0	0.0	0.0

INCREASED INCIDENCE OF HEALTH EFFECTS IN REGIONAL POPULATION--EXPOSURE OVER HUMAN LIFETIME

HEALTH EFFECT	ONE HIT	MULTIHIT	DOSE-RESPONSE RELATIONSHIPS MULTISTAGE	GRUMP	PROBIT	WEIBULL
CANCER-A	0.28948E-02	0.97083E+00	0.0	0.0	0.32957E+01	0.0
CANCER-B	0.31843E-02	0.11744E+01	0.0	0.0	0.43017E+01	0.0
CANCER-C	0.0	0.0	0.71716E-03	0.88477E+00	0.0	0.0
CHRONIC	0.11828E-02	0.32564E-05	0.0	0.0	0.0	0.0

PROGRAM CODE

The computer program that calculates exposure rates and health effects has been written in standard FORTRAN. A copy of the source code for the program is given below.

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C      TITLE (MAIN PROGRAM)
C
C      PROGRAM CALCULATES EXPOSURE RATES FOR INHALATION, INGESTION, AND
C      DERMAL CONTACT WITH POLLUTANT SPECIES, GIVEN CONCENTRATIONS IN
C      THE ENVIRONMENT AND OTHER DESCRIPTIVE INFORMATION.  THE EXPOSURE
C      RATES ARE USED WITH DOSE-RESPONSE RELATIONSHIPS AND POPULATION
C      INFORMATION TO CALCULATE THE ASSOCIATED CHRONIC HEALTH RISK.
C
C      COMMON      DESCRIPTION
C      CASE        DESCRIPTION OF CASE (2-DIM INTEGER ARRAY)
C      HLFIE       LENGTH OF HUMAN LIFETIME
C      NPG         NUMBER OF POPULATION GROUPS (INTEGER)
C      NPR         NUMBER OF POPULATION SUBREGIONS (INTEGER)
C      PL          RATIO OF POWER PLANT OPERATING LIFE TO HUMAN LIFE
C      PLIFE       SERVICE LIFE OF POWER PLANT
C      POLLUT      NAME OF POLLUTANT SPECIES (2-DIM REAL ARRAY)
C      POPGRP      NAME OF POPULATION GROUPS (2-DIM REAL ARRAY)
C      P           POPULATION BY SUBREGION AND GROUP (2-DIM REAL ARRAY)
C      TPR         TOTAL POPULATION BY SUBREGION (1-DIM REAL ARRAY)
C      TPS         TOTAL POPULATION
C      TP          TOTAL POPULATION BY GROUP (1-DIM REAL ARRAY)
C      RSH         INHALATION EXPOSURE ABSORPTION FRACTION
C      QINIT       LOGICAL INITIALIZATION INDICATOR--.TRUE. FOR THE
C                  INITIAL POLLUTANT SPECIES THEN .FALSE. THEREAFTER
C      QINHLE      LOGICAL INHALATION EXPOSURE INDICATOR--.TRUE. IF
C                  INHALATION EXPOSURE IS TO BE CALCULATED
C      QINGST      LOGICAL INGESTION EXPOSURE INDICATOR
C      QDERML      LOGICAL DERMAL EXPOSURE INDICATOR
C      QGRW        LOGICAL GROUNDWATER USE INDICATOR
C      QSURFW      LOGICAL SURFACE WATER USE INDICATOR
C
C      EXTERNALS   DESCRIPTION
C      RDCONC      SUBROUTINE TO READ POLLUTANT CONCENTRATIONS
C      RDINGS      SUBROUTINE TO READ BASIC INGESTION EXPOSURE DATA
C      RDAQUA      SUBROUTINE TO READ DATA FOR CONSUMPTION OF AQUATIC
C                  ORGANISMS
C      RDANIM      SUBROUTINE TO READ DATA FOR CONSUMPTION OF ANIMAL
C                  PRODUCTS
C      RDVEGT      SUBROUTINE TO READ DATA FOR CONSUMPTION OF VEGETABLE
C                  PRODUCTS
C      RDDERM      SUBROUTINE TO READ DATA FOR DERMAL EXPOSURE
C      WRCASE      SUBROUTINE TO WRITE GENERAL CASE DESCRIPTION
C      WRPOLL      SUBROUTINE TO WRITE POLLUTANT CONCENTRATIONS
C      CALINH      SUBROUTINE TO CALCULATE INHALATION EXPOSURE
C      CALING      SUBROUTINE TO CALCULATE INGESTION EXPOSURE
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C      CALDER      SUBROUTINE TO CALCULATE DERMAL CONTACT EXPOSURE
C      EXSUMC      SUBROUTINE TO CONVERT AND SUM EXPOSURE RATES BY
C                  MODE AND CALCULATE MEAN VALUES
C      WREXP      SUBROUTINE TO WRITE EXPOSURE INFORMATION
C      HEALTH      SUBROUTINE TO CALCULATE HEALTH EFFECTS
C      WRHLTH      SUBROUTINE TO WRITE RESULTS OF HEALTH CALCULATIONS
C
C      VARIABLE    DESCRIPTION
C      IMODE       LIST OF EXPOSURE MODES CONSIDERED (2-DIM REAL ARRAY)
C      JMODE       REFERENCE LIST OF EXPOSURE MODES (2-DIM REAL ARRAY)
C      IWAT        LIST OF TYPES OF WATER CONSIDERED (2-DIM REAL ARRAY)
C      JWAT        REFERENCE LIST OF WATER TYPES (2-DIM REAL ARRAY)
C      NPS         NUMBER OF POLLUTANT SPECIES (INTEGER)
C      NAP         NUMBER OF ANIMAL PRODUCTS (INTEGER)
C      NAO         NUMBER OF SPECIES OF AQUATIC ORGANISMS (INTEGER)
C      NVS         NUMBER OF VEGETABLE PRODUCTS (INTEGER)
C      II          INDEX VARIABLE FOR POLLUTANT SPECIES (INTEGER)
C      NHE         NUMBER OF HEALTH EFFECTS CONSIDERED (INTEGER)
C
C      INTEGER CASE(4,18)
C      REAL IWAT(2,2), POLLUT(2,5), POPGRP(2,4), IMODE(2,3), JMODE(2,3),
1      JWAT(2,2), P(4,100), TP(4), TPR(100)
C      LOGICAL QINIT, QINHLE, QINGST, QDERML, QGRW, QSURFW
C      COMMON /L/HLIFE, PLIFE, FS, HS, RSG, RSH
C      COMMON /N/NPG, NPR, PL
C      COMMON /NAME/CASE, AQUAT(2,5), ANIMAL(2,10), VEGET(2,10)
C      COMMON /POL/POLLUT
C      COMMON /POP/POPGRP, P, TPR, TPS, TP
C      COMMON /Q/QINIT, QINHLE, QINGST, QDERML, QGRW, QSURFW
C      DATA JWAT(1,1), JWAT(2,1), JWAT(1,2), JWAT(2,2)/4HSURF, 4HACE ,
1      4HGROU, 4HND /
C      DATA JMODE(1,1), JMODE(2,1), JMODE(1,2), JMODE(2,2), JMODE(1,3),
1      JMODE(2,3)/4HINHA, 4HLE , 4HINGE, 4HST , 4HDERM, 4HAL /
C
C      SECTION I    READ BASIC INPUT DATA, SET INDICATOR VARIABLES TO
C                  DETERMINE PATH OF PROGRAM, AND CALCULATE BASIC
C                  POPULATION INFORMATION.
C
C      STEP 1      READ GENERAL DESCRIPTION OF CASE
C
C      DO 20 I = 1,4
C      20 READ (5,5005) (CASE(I,J), J=1,18)
C
C      STEP 2      READ LIST OF EXPOSURE MODES TO BE CONSIDERED AND
C                  COMPARE THIS TO THE STORED LIST. SET LOGICAL
C                  INDICATOR VARIABLES TO .TRUE. FOR ALL SELECTED MODES.
C
C      READ (5,5001) ((IMODE(J,I), J=1,2), I=1,3)
C      QINHLE = .FALSE.
C      QINGST = .FALSE.
C      QDERML = .FALSE.
C      DO 30 J = 1,3

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      IF (IMODE(1,J) .EQ. JMODE(1,1))   QINHLE = .TRUE.
      IF (IMODE(1,J) .EQ. JMODE(1,2))   QINGST = .TRUE.
      IF (IMODE(1,J) .EQ. JMODE(1,3))   QDERML = .TRUE.
30  CONTINUE
C
C      STEP 3      READ LIST OF WATER TYPES TO BE CONSIDERED AND COMPARE
C                  THIS TO THE STORED LIST.  SET LOGICAL INDICATOR
C                  VARIABLES TO .TRUE. FOR ALL SELECTED TYPES.
C
      READ (5,5001) ((IWAT(J,I),J=1,2),I=1,2)
      QGRW = .FALSE.
      QSURFW = .FALSE.
      DO 40 J = 1,2
      IF (IWAT(1,J) .EQ. JWAT(1,1))   QSURFW = .TRUE.
      IF (IWAT(1,J) .EQ. JWAT(1,2))   QGRW = .TRUE.
40  CONTINUE
C
C      STEP 4      READ NUMBER AND NAME OF POLLUTANT SPECIES AND
C                  POPULATION GROUPS AND NUMBER OF SUBREGIONS.  READ
C                  HUMAN AND POWER PLANT LIVES AND CALCULATE RATIO.
C
      READ (5,5006) NPS,((POLLUT(I,J),I=1,2),J=1,NPS)
      READ (5,5006) NPG,((POPGRP(J,I),J=1,2),I=1,NPG)
      READ (5,5004) NPR,PLIFE,HLIFE
      PL = PLIFE/HLIFE
C
C      STEP 5      READ POPULATION BY SUBREGION AND GROUP.  SUM OVER
C                  SUBREGION AND GROUP SEPARATELY, AND ALSO OVER BOTH
C                  TO DETERMINE TOTAL REGIONAL POPULATION.
C
      DO 50 J = 1,NPR
50  READ (5,5003) (P(I,J),I=1,NPG)
      TPS = 0.
      DO 70 I = 1,NPG
      TP(I) = 0.
      DO 60 J = 1,NPR
      IF (I .EQ. 1)   TPR(J) = 0.
      TPR(J) = TPR(J)+P(I,J)
60  TP(I) = TP(I)+P(I,J)
70  TPS = TPS+TP(I)
C
C      STEP 6      READ NUMBER OF ANIMAL PRODUCTS, AQUATIC ORGANISMS,
C                  AND VEGETABLE PRODUCTS IN ANALYSIS.
C
      READ (5,5002) NAP,NAO,NVS
C
C      SECTION II  CALCULATE EXPOSURE RATES BY SELECTED PATHWAYS FOR
C                  EACH POLLUTANT SPECIES.
C
C      STEP 1      SET INITIAL SPECIES INDICATOR TO .TRUE. AND BEGIN
C                  LOOP THROUGH POLLUTANT SPECIES.  READ POLLUTANT
C                  CONCENTRATIONS AND DEPOSITION RATES FOR EACH SPECIES.

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C          READ INHALATION EXPOSURE ABSORPTION FRACTION.
C
    QINIT = .TRUE.
    DO 100 II = 1,NPS
    IF (II .GT. 1) QINIT = .FALSE.
    CALL RDCONC
    IF (QINHLE) READ (5,5007) RSH
C
C    STEP 2      IF INGESTION EXPOSURE WILL BE CALCULATED, READ
C                BASIC DESCRIPTIVE INFORMATION FOR ALL APPROPRIATE
C                TYPES OF PRODUCTS.
C
    IF (QINGST) CALL RDINGS
    IF (NAO .NE. 0) CALL RDAQUA(NAO)
    IF (NAP .NE. 0) CALL RDANIM(NAP)
    IF (NVS .NE. 0) CALL RDVEGT(NVS)
C
C    STEP 3      IF DERMAL EXPOSURE WILL BE CALCULATED, READ BASIC
C                DESCRIPTIVE INFORMATION.
C
    IF (QDERML) CALL RDDERM
C
C    STEP 4      IF THIS IS THE INITIAL POLLUTANT SPECIES, WRITE THE
C                GENERAL CASE DESCRIPTION. FOR ALL SPECIES, WRITE
C                THE POLLUTANT CONCENTRATIONS USED IN THE ANALYSIS.
C
    IF (QINIT) CALL WRCASE(NAO,NAP,NVS)
    CALL WRPOLL(II)
C
C    STEP 5      CALCULATE EXPOSURE RATES FOR THIS POLLUTANT SPECIES
C                FOR EACH SELECTED INTAKE MODE. CONVERT THESE RATES
C                AND SUM THEM BY INTAKE MODE, CALCULATE MEAN VALUES,
C                WRITE THE RESULTS, AND RETURN TO STEP 1 TO REPEAT
C                FOR THE NEXT POLLUTANT SPECIES.
C
    IF (QINHLE) CALL CALINH(II)
    IF (QINGST) CALL CALING(II,NAO,NAP,NVS)
    IF (QDERML) CALL CALDER(II)
    CALL EXSUMC(II)
    CALL WREXP(II,NAO,NAP,NVS)
100 CONTINUE
C
C    SECTION III CALCULATE HEALTH EFFECTS FROM EXPOSURE INFORMATION
C
C    STEP 1      INTEGRATE EXPOSURE CALCULATIONS WITH POPULATION
C                INFORMATION AND DOSE-RESPONSE MODELS TO CALCULATE
C                HEALTH EFFECTS FOR THIS CASE.
C
    CALL HEALTH(NHE)
C
C    STEP 2      WRITE THE RESULTS OF THE HEALTH EFFECTS CALCULATIONS.
C

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      CALL WRHLTH(NHE)
C
C   FORMAT STATEMENTS
C
5001 FORMAT (3(2A4))
5002 FORMAT (16I5)
5003 FORMAT (6(E12.5,1X))
5004 FORMAT (I5,10F6.0)
5005 FORMAT (20A4)
5006 FORMAT (I5,5X,5(2A4,2X))
5007 FORMAT (F10.0)
      STOP
      END

      SUBROUTINE RDCONC
C
C   TITLE (READ CONCENTRATIONS)
C
C   SUBROUTINE RDCONC READS POLLUTANT CONCENTRATIONS AND DEPOSITION
C   RATES IN THE ENVIRONMENT FOR ONE POLLUTANT SPECIES
C
C   COMMON      DESCRIPTION
C   C           POLLUTANT CONCENTRATIONS BY SUBREGION AND MEDIUM FOR
C   BC          BACKGROUND POLLUTANT CONCENTRATIONS BY SUBREGION AND
C   AFD         ATMOSPHERIC PARTICULATE DEPOSITION RATES BY SUBREGION
C   BAFD        BACKGROUND PARTICULATE DEPOSITION RATES BY SUBREGION
C   NPR         NUMBER OF POPULATION SUBREGIONS (INTEGER)
C   QINHLE      LOGICAL INHALATION EXPOSURE INDICATOR--.TRUE. IF
C   QINGST      LOGICAL INGESTION EXPOSURE INDICATOR
C   QDERML      LOGICAL DERMAL EXPOSURE INDICATOR
C
C   CALLED BY   MAIN PROGRAM
C
      REAL C(5,100),BC(5,100),AFD(100),BAFD(100)
      LOGICAL QINIT,QINHLE,QINGST,QDERML,QGRW,QSURFW
      COMMON /CON/C,BC
      COMMON /DEP/AFD,BAFD
      COMMON /N/NPG,NPR,PL
      COMMON /Q/QINIT,QINHLE,QINGST,QDERML,QGRW,QSURFW
C
C   STEP 1      INITIALIZE CONCENTRATION AND DEPOSITION ARRAYS
C
      DO 10 J = 1,100
      AFD(J) = 0.0
      BAFD(J) = 0.0
      DO 10 I = 1,5
      BC(I,J) = 0.0

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10 C(I,J) = 0.0
C
C STEP 2 IF INHALATION EXPOSURE IS CONSIDERED, READ GASEOUS
C AND PARTICULATE POLLUTANT CONCENTRATIONS IN THE
C ATMOSPHERE. OTHERWISE, BRANCH TO STEP 3.
C
IF (.NOT. QINHLE) GO TO 30
DO 20 J = 1,NPR
20 READ (5,5001) C(1,J),BC(1,J),C(2,J),BC(2,J)
C
C STEP 3 IF INGESTION EXPOSURE IS CONSIDERED, READ POLLUTANT
C CONCENTRATIONS IN SURFACE SOIL LAYERS. OTHERWISE,
C BRANCH TO STEP 4.
C
30 IF (.NOT. QINGST) GO TO 50
DO 40 J = 1,NPR
40 READ (5,5001) C(3,J),BC(3,J)
C
C STEP 4 IF INGESTION OR DERMAL EXPOSURE IS CONSIDERED, READ
C POLLUTANT CONCENTRATIONS IN SURFACE AND GROUNDWATER.
C OTHERWISE, BRANCH TO STEP 5.
C
50 IF ((.NOT. QINGST) .AND. (.NOT. QDERML)) GO TO 70
DO 60 J = 1,NPR
60 READ (5,5001) C(4,J),BC(4,J),C(5,J),BC(5,J)
C
C STEP 5 IF INGESTION EXPOSURE IS CONSIDERED, READ ATMOSPHERIC
C DEPOSITION RATES. OTHERWISE, RETURN TO MAIN PROGRAM.
C
70 IF (.NOT. QINGST) RETURN
DO 80 J = 1,NPR
80 READ (5,5001) AFD(J),BAFD(J)
RETURN
C
C FORMAT STATEMENTS
C
5001 FORMAT (6(E12.5,1X))
END

SUBROUTINE RDINGS
C
C TITLE (READ INGESTION DATA)
C
C SUBROUTINE RDINGS READS BASIC INFORMATION NEEDED FOR ALL
C CALCULATIONS OF INGESTION EXPOSURE RATES, INCLUDING EXPOSURE FROM
C CONSUMPTION OF DRINKING WATER AND ATMOSPHERIC PARTICULATES.
C
C COMMON DESCRIPTION
C NTP NUMBER OF DRINKING WATER TREATMENT PROCESSES USED
C IN THE REGION (INTEGER)
C TF FRACTION OF POLLUTANT SPECIES REMAINING AFTER
C DRINKING WATER TREATMENT (1-DIM REAL ARRAY)

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C      VF      FRACTION OF DOMESTIC WATER CONSUMED BY SUBREGION THAT
C              COMES FROM SURFACE WATER SOURCES (1-DIM REAL ARRAY)
C      IWT     DRINKING WATER TREATMENT PROCESSES USED WITHIN THE
C              SUBREGION FOR SURFACE AND GROUNDWATER SOURCES (2-DIM
C              INTEGER ARRAY)
C      UI      INHALATION RATES BY POPULATION GROUP (1-DIM REAL
C              ARRAY)
C      W       HUMAN WEIGHTS BY POPULATION GROUP (1-DIM REAL ARRAY)
C      TU      TOTAL FOOD CONSUMPTION RATES BY POPULATION GROUP
C              (1-DIM REAL ARRAY)
C      UW      DRINKING WATER CONSUMPTION RATES BY POPULATION GROUP
C              (1-DIM REAL ARRAY)
C      PIG     FRACTION OF INHALED PARTICULATES THAT IS INGESTED
C      BMF     MEAN BACKGROUND POLLUTANT CONCENTRATION IN FOOD FROM
C              OUTSIDE THE POWER PLANT REGION
C      FS      SPECIFIC INHALATION RATE COEFFICIENT
C      HS      SPECIFIC INGESTION RATE COEFFICIENT
C      RSG     INGESTION EXPOSURE ABSORPTION FRACTION
C      NPR     NUMBER OF POPULATION SUBREGIONS (INTEGER)
C      QINIT   LOGICAL INITIALIZATION INDICATOR--.TRUE. FOR THE
C              INITIAL POLLUTANT SPECIES, THEN .FALSE. THEREAFTER
C
C      CALLED BY  MAIN PROGRAM
C
C      REAL UI(4),W(4),TU(4),UW(4),TF(10),VF(100)
C      INTEGER IWT(2,100)
C      LOGICAL QINIT,QINHLE,QINGST,QDERML,QGRW,QSURFW
C      COMMON /DW/TF,VF,IWT,NTP
C      COMMON /IGD/UI,W,TU,UW,PIG,BMF
C      COMMON /L/HLIFE,PLIFE,FS,HS,RSG,RSH
C      COMMON /N/NPG,NPR,PL
C      COMMON /Q/QINIT,QINHLE,QINGST,QDERML,QGRW,QSURFW
C
C      STEP 1    IF THIS IS THE INITIAL POLLUTANT SPECIES, PROCEED TO
C              STEP 2 TO READ BASIC INGESTION DATA.  OTHERWISE,
C              BRANCH TO STEP 3.
C
C      IF (.NOT. QINIT)  GO TO 10
C
C      STEP 2    READ BASIC HUMAN DESCRIPTIVE DATA REQUIRED FOR ANY
C              INGESTION EXPOSURE CALCULATIONS.
C
C      READ (5,5001) FS,HS,RSG
C      READ (5,5002) (UI(I),I=1,NPG),PIG
C      READ (5,5002) (W(I),I=1,NPG)
C      READ (5,5002) (TU(I),I=1,NPG),BMF
C      READ (5,5002) (UW(I),I=1,NPG)
C
C      STEP 3    READ DESCRIPTION OF WATER TREATMENT PROCESSES.
C
C      10 READ (5,5003) NTP,(TF(I),I=1,NTP)
C

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C      STEP 4      IF THIS IS THE INITIAL POLLUTANT SPECIES, READ THE
C                  DESCRIPTION OF THE DOMESTIC WATER USE AND TREATMENT
C                  PROCESSES BY SUBREGION.  OTHERWISE, RETURN.
C
C                  IF (.NOT. QINIT)  RETURN
C                  DO 20 J = 1,NPR
C                  READ (5,5004) VF(J),IWT(1,J),IWT(2,J)
20    CONTINUE
C                  RETURN
C
C      FORMAT STATEMENTS
C
C      5001 FORMAT (16F6.0)
C      5002 FORMAT (6(E12.5,1X))
C      5003 FORMAT (I5,10F6.0)
C      5004 FORMAT (F5.0,2I5)
C      END
C
C      SUBROUTINE RDAQUA(NAO)
C
C      TITLE (READ AQUATIC ORGANISM DATA)
C
C      SUBROUTINE RDAQUA READS INFORMATION NEEDED TO CALCULATE EXPOSURE
C      RATES ASSOCIATED WITH THE CONSUMPTION OF AQUATIC ORGANISMS.
C
C      PARAMETER      DESCRIPTION
C      NAO             NUMBER OF SPECIES OF AQUATIC ORGANISMS (INTEGER)
C
C      COMMON          DESCRIPTION
C      PQ              REGIONAL PRODUCTION OF ORGANISMS (1-DIM REAL ARRAY)
C      CQ              MEAN SURFACE WATER POLLUTANT CONCENTRATION WHERE THE
C                     ORGANISMS ARE FOUND (1-DIM REAL ARRAY)
C      BCQ             MEAN SURFACE WATER BACKGROUND POLLUTANT CONCENTRATION
C                     WHERE ORGANISMS ARE FOUND (1-DIM REAL ARRAY)
C      B               BIOCONCENTRATION FACTOR FOR AQUATIC ORGANISMS (1-DIM
C                     REAL ARRAY)
C      QCB             POLLUTANT CONCENTRATION IN SUPPLY OF ORGANISMS FROM
C                     OUTSIDE POWER PLANT REGION (1-DIM REAL ARRAY)
C      NPG             NUMBER OF POPULATION GROUPS (INTEGER)
C      AQUAT           NAMES OF AQUATIC ORGANISMS (2-DIM REAL ARRAY)
C      QINIT           LOGICAL INITIALIZATION INDICATOR--.TRUE. FOR THE
C                     INITIAL POLLUTANT SPECIES THEN .FALSE. THEREAFTER
C      UQ              CONSUMPTION OF ORGANISMS BY POPULATION GROUP (2-DIM
C                     REAL ARRAY)
C
C      CALLED BY      MAIN PROGRAM
C
C      REAL AQUAT(2,5),CASE(4,18),ANIMAL(2,10),VEGET(2,10),PQ(5),CQ(5),
1    BCQ(5),B(5),QCB(5),UQ(4,5)
C      LOGICAL QINIT,QINHLE,QINGST,QDERML,QGRW,QSURFW
C      COMMON /AQ/PQ,CQ,BCQ,B,QCB
C      COMMON /N/NPG,NPR,PL

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COMMON /NAME/CASE,AQUAT,ANIMAL,VEGET
COMMON /Q/QINIT,QINHLE,QINGST,QDERML,QGRW,QSURFW
COMMON /U/UA(4,10),UQ,UV(4,10)

C
C STEP 1      FOR EACH AQUATIC ORGANISM, READ DESCRIPTIVE DATA
C              ABOUT ITS PRODUCTION AND CHARACTERISTICS.  IF THIS
C              IS THE INITIAL POLLUTANT SPECIES, READ CONSUMPTION
C              RATES.
C
DO 10 I = 1,NAO
READ (5,5001) (AQUAT(J,I),J=1,2),PQ(I),CQ(I),BCQ(I),B(I),QCB(I)
IF (.NOT. QINIT) GO TO 10
READ (5,5002) (UQ(J,I),J=1,NPG)
10 CONTINUE
RETURN

C
C FORMAT STATEMENTS
C
5001 FORMAT (2A4,5(E12.5,1X))
5002 FORMAT (6(E12.5,1X))
END

SUBROUTINE RDANIM(NAP)

C
C TITLE (READ ANIMAL PRODUCT INFORMATION)
C
SUBROUTINE RDANIM READ INFORMATION NEEDED TO CALCULATE EXPOSURE
RATES ASSOCIATED WITH THE CONSUMPTION OF ANIMAL PRODUCTS.

C
C PARAMETER    DESCRIPTION
C NAP          NUMBER OF ANIMAL PRODUCTS (INTEGER)
C
C COMMON       DESCRIPTION
C FEED         TYPES OF FEED FOR ANIMAL PRODUCTS (3-DIM REAL ARRAY)
C MF          FRACTION OF DAILY POLLUTANT INTAKE REACHING THE
C              ANIMAL PRODUCT (1-DIM REAL ARRAY)
C QW          WATER CONSUMPTION RATE FOR ANIMAL PRODUCTS (1-DIM
C              REAL ARRAY)
C QF          FEED CONSUMPTION RATE FOR ANIMAL PRODUCTS (3-DIM
C              REAL ARRAY)
C ACB         POLLUTANT CONCENTRATION IN ANIMAL PRODUCTS PRODUCED
C              OUTSIDE THE REGION (1-DIM REAL ARRAY)
C RPA         SUBREGIONAL PRODUCTION OF ANIMAL PRODUCTS (2-DIM
C              REAL ARRAY)
C CW          POLLUTANT CONCENTRATION IN ANIMAL DRINKING WATER
C              (2-DIM REAL ARRAY)
C BCW        BACKGROUND POLLUTANT CONCENTRATION IN ANIMAL DRINKING
C              WATER (2-DIM REAL ARRAY)
C CF         POLLUTANT CONCENTRATION IN ANIMAL FEED (3-DIM REAL
C              ARRAY)
C BCF        BACKGROUND POLLUTANT CONCENTRATION IN ANIMAL FEED
C              (3-DIM REAL ARRAY)

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C   NAPR          NUMBER OF SUBREGIONS PRODUCING ANIMAL PRODUCTS
C                   (1-DIM INTEGER ARRAY)
C   NTF           NUMBER OF TYPES OF FEED CONSUMED BY ANIMALS (2-DIM
C                   INTEGER ARRAY)
C   NPG           NUMBER OF POPULATION GROUPS (INTEGER)
C   ANIMAL        NAMES OF ANIMAL PRODUCTS (2-DIM REAL ARRAY)
C   QINIT         LOGICAL INITIALIZATION INDICATOR--.TRUE. FOR THE
C                   INITIAL POLLUTANT SPECIES THEN .FALSE. THEREAFTER
C   UA            CONSUMPTION OF ANIMAL PRODUCTS BY POPULATION GROUP
C                   (2-DIM REAL ARRAY)
C
C   VARIABLE      DESCRIPTION
C   KU            CURRENT SUBREGION FOR ANIMAL PRODUCTION (INTEGER)
C   JU            CURRENT NUMBER OF TYPES OF ANIMAL FEED (INTEGER)
C
C   CALLED BY     MAIN PROGRAM
C
C
C   REAL ANIMAL(2,10),UA(4,10),MF(10),QW(10),ACB(10),RPA(10,100),
1     FEED(10,100,3),QF(10,100,3),CW(10,100),BCW(10,100),
2     CF(10,100,3),BCF(10,100,3),CASE(4,18),AQUAT(2,5),VEGET(2,10)
C   INTEGER NAPR(10),NTF(10,100)
C   LOGICAL QINIT,QINHLE,QINGST,QDERML,QGRW,QSURFW
C   COMMON /AN/FEED,MF,QW,ACB,RPA,QF,CW,BCW,CF,BCF,NAPR,NTF
C   COMMON /N/NPG,NPR,PL
C   COMMON /NAME/CASE,AQUAT,ANIMAL,VEGET
C   COMMON /Q/QINIT,QINHLE,QINGST,QDERML,QGRW,QSURFW
C   COMMON /U/UA,UQ(4,5),UV(4,10)
C
C   STEP 1        INITIALIZE PRODUCTION LEVEL ARRAY ON THE INITIAL
C                   PASS THROUGH THIS ROUTINE.  ON SUBSEQUENT PASSES,
C                   BRANCH TO STEP 2.
C
C   IF (.NOT. QINIT) GO TO 20
C   DO 10 J = 1,100
C   DO 10 I = 1,10
C   RPA(I,J) = 0.
10  CONTINUE
C
C   STEP 2        BEGIN TO READ INPUT DATA FOR EACH ANIMAL PRODUCT.
C                   IF THIS IS THE INITIAL POLLUTANT SPECIES, READ
C                   BASIC INFORMATION THAT IS INDEPENDENT OF SPECIES.
C                   OTHERWISE, BRANCH TO STEP 3.
C
C   20 DO 60 I = 1,NAP
C   IF (.NOT. QINIT) GO TO 30
C   READ (5,5001) (ANIMAL(J,I),J=1,2),NAPR(I)
C   READ (5,5002) (UA(K,I),K=1,NPG)
C
C   STEP 3        FOR THE INITIAL POLLUTANT SPECIES, BY PRODUCING
C                   SUBREGION, READ DESCRIPTION OF PRODUCTION LEVELS AND
C                   ANIMAL FEEDS.  FOR ALL POLLUTANT SPECIES, ALSO READ

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C          POLLUTANT CONCENTRATIONS IN FEED AND WATER.
C
30 READ (5,5002) MF(I),QW(I),ACB(I)
   KU = NAPR(I)
   DO 50 K = 1,KU
   IF (.NOT. QINIT) GO TO 40
   READ (5,5003) IR,NTF(I,IR),RPA(I,IR)
   JU = NTF(I,IR)
   READ (5,5004) (FEED(I,IR,J),QF(I,IR,J),J=1,JU)
40 READ (5,5005) IR,CW(I,IR),BCW(I,IR)
   JU = NTF(I,IR)
   READ (5,5002) (CF(I,IR,J),J=1,JU),(BCF(I,IR,J),J=1,JU)
50 CONTINUE
60 CONTINUE
   RETURN
C
C   FORMAT STATEMENTS
C
5002 FORMAT (6(E12.5,1X))
5001 FORMAT (2A4,5I5)
5003 FORMAT (2I5,1X,3(E12.5,1X))
5004 FORMAT (3(A4,6X,E12.5,1X))
5005 FORMAT (I5,1X,5(E12.5,1X))
   END

   SUBROUTINE RDVEGT(NVS)
C
C   TITLE (READ VEGETABLE PRODUCT INFORMATION)
C
C   SUBROUTINE RDVEGT READS INFORMATION NEEDED TO CALCULATE EXPOSURE
C   RATES ASSOCIATED WITH THE CONSUMPTION OF VEGETABLE PRODUCTS.
C
C   PARAMETER      DESCRIPTION
C   NVS            NUMBER OF VEGETABLE PRODUCTS (INTEGER)
C
C   COMMON          DESCRIPTION
C   NPG            NUMBER OF POPULATION GROUPS (INTEGER)
C   VEGET          NAME OF VEGETABLE PRODUCTS (2-DIM REAL ARRAY)
C   QINIT          LOGICAL INITIALIZATION INDICATOR--.TRUE. FOR THE
C                 INITIAL POLLUTANT SPECIES THEN .FALSE. THEREAFTER
C   UV            CONSUMPTION OF VEGETABLE PRODUCTS BY POPULATION
C                 GROUP (2-DIM REAL ARRAY)
C   CV            BIOCONCENTRATION FACTOR FOR VEGETABLE PRODUCTS
C                 (1-DIM REAL ARRAY)
C   FC            CAPTURE FRACTION FOR VEGETABLE PRODUCTS (1-DIM REAL
C                 ARRAY)
C   VG            VEGETATIVE SURFACE DENSITY (1-DIM REAL ARRAY)
C   VCB           BACKGROUND POLLUTANT CONCENTRATION IN PRODUCTS FROM
C                 OUTSIDE THE POWER PLANT REGION (1-DIM REAL ARRAY)
C   RPV           SUBREGIONAL PRODUCTION OF VEGETABLE PRODUCTS (2-DIM
C                 REAL ARRAY)
C   FI            FRACTION OF VEGETABLE PRODUCTION IRRIGATED (3-DIM

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C          REAL ARRAY)
C      CI      POLLUTANT CONCENTRATION IN IRRIGATION WATER (2-DIM
C              REAL ARRAY)
C      BCI     BACKGROUND POLLUTANT CONCENTRATION IN IRRIGATION
C              WATER (2-DIM REAL ARRAY)
C      MFC     MEAN CAPTURE FRACTION FOR PLANTS
C      IRR     IRRIGATION RATE FOR VEGETABLE PRODUCTS (1-DIM REAL
C              ARRAY)
C      TL      TRANSLOCATION FACTOR FOR POLLUTANT MOVEMENT TO EDIBLE
C              PARTS OF PRODUCTS (1-DIM REAL ARRAY)
C      SD      MEAN EFFECTIVE SURFACE SOIL DENSITY
C      TS      POLLUTANT TRANSFER LOSS RATE THROUGH SOIL
C      WL      WEATHERING LOSS RATE FROM VEGETATION SURFACES
C      NVSR     NUMBER OF SUBREGIONS PRODUCING VEGETABLE PRODUCTS
C              (1-DIM INTEGER ARRAY)
C
C      VARIABLE  DESCRIPTION
C      JU        CURRENT NUMBER OF SUBREGIONS PRODUCING VEGETABLE
C              PRODUCT (INTEGER)
C
C      CALLED BY  MAIN PROGRAM
C
C      REAL VEGET(2,10),UV(4,10),CV(10),FC(10),VG(10),VCB(10),
1      RPV(10,100),FI(2,10,100),CI(2,100),BCI(2,100),MFC,IRR(10),
2      TL(10),CASE(4,18),ANIMAL(2,10),AQUAT(2,5)
C      INTEGER NVSR(10)
C      LOGICAL QINIT,QINHLE,QINGST,QDERML,QGRW,QSURFW
C      COMMON /N/NPG,NPR,PL
C      COMMON /NAME/CASE,AQUAT,ANIMAL,VEGET
C      COMMON /Q/QINIT,QINHLE,QINGST,QDERML,QGRW,QSURFW
C      COMMON /U/CA(4,10),UQ(4,5),UV
C      COMMON /VG/CV,FC,VG,VCB,RPV,FI,CI,BCI,MFC,IRR,TL,SD,TS,WL,NVSR
C
C      STEP 1      INITIALIZE PRODUCTION LEVEL ARRAY
C
C      IF (.NOT. QINIT)  GO TO 20
C      DO 10 J = 1,100
C      DO 10 I = 1,10
C      RPV(I,J) = 0.
10  CONTINUE
C
C      STEP 2      FOR THE INITIAL POLLUTANT SPECIES, READ BASIC PLANT
C                  INFORMATION.  OTHERWISE, BRANCH TO STEP 3.
C
C      IF (QINIT)  READ (5,5002) MFC,SD,TS,WL
C
C      STEP 3      BEGIN TO READ INPUT DATA FOR EACH VEGETABLE PRODUCT.
C                  IF THIS IS THE INITIAL POLLUTANT SPECIES, READ BASIC
C                  INFORMATION THAT IS INDEPENDENT OF SPECIES.
C                  OTHERWISE, BRANCH TO STEP 4.
C
C      20 DO 60 I = 1,NVS

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      IF (.NOT. QINIT) GO TO 30
      READ (5,5001) (VEGET(J,I),J=1,2),NVSR(I)
      READ (5,5002) (UV(K,I),K=1,NPG)
      READ (5,5002) CV(I),FC(I),IRR(I),TL(I),VG(I),VCB(I)
C
C      STEP 4      FOR THE INITIAL POLLUTANT SPECIES, BY PRODUCING
C                  SUBREGION, READ DESCRIPTION OF PRODUCTION LEVELS AND
C                  IRRIGATION PRACTICES. FOR ALL POLLUTANT SPECIES,
C                  ALSO READ POLLUTANT CONCENTRATIONS IN WATER.
C
30  JU = NVSR(I)
      DO 50 J = 1,JU
      IF (.NOT. OINIT) GO TO 40
      READ (5,5003) IR,RPV(I,IR)
      READ (5,5004) (FI(K,I,IR),K=1,2)
40  READ (5,5005) IR,CI(1,IR),BCI(1,IR),CI(2,IR),BCI(2,IR)
50  CONTINUE
60  CONTINUE
      RETURN
C
C      FORMAT STATEMENTS
C
5001 FORMAT (2A4,5I5)
5002 FORMAT (6(E12.5,1X))
5003 FORMAT (15,1X,E12.5)
5004 FORMAT (16F5.0)
5005 FORMAT (15,1X,5(E12.5,1X))
      END

      SUBROUTINE RDDERM
C
C      TITLE (READ DERMAL EXPOSURE INFORMATION)
C
C      SUBROUTINE RDDERM READS BASIC INFORMATION NEEDED TO CALCULATE
C      EXPOSURE THROUGH DERMAL CONTACT PATHWAYS.
C
C      COMMON      DESCRIPTION
C      TDA          DURATION OF DERMAL EXPOSURE TO ATMOSPHERE (1-DIM
C                  REAL ARRAY)
C      TDD          DURATION OF DERMAL EXPOSURE TO DOMESTIC WATER
C                  SUPPLIES (1-DIM REAL ARRAY)
C      TDR          DURATION OF DERMAL EXPOSURE TO RECREATIONAL WATERS
C                  (1-DIM REAL ARRAY)
C      EDA          FRACTION OF BODY SURFACE EXPOSED TO THE ATMOSPHERE
C      EDAD         FRACTION OF BODY SURFACE EXPOSED TO DOMESTIC WATER
C      EDAR         FRACTION OF BODY SURFACE EXPOSED TO RECREATIONAL
C                  WATERS
C      GS          COEFFICIENT OF DERMAL MASS TRANSFER PER UNIT AREA
C      CR          POLLUTANT CONCENTRATIONS IN RECREATIONAL WATERS
C                  (1-DIM REAL ARRAY)
C      BCR          BACKGROUND POLLUTANT CONCENTRATIONS IN RECREATIONAL
C                  WATERS (1-DIM REAL ARRAY)

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C      NRL          NUMBER OF RECREATIONAL SITES (INTEGER)
C      RUF          FRACTIONAL USE OF RECREATIONAL LOCATIONS (1-DIM REAL
C                   ARRAY)
C      NPG          NUMBER OF POPULATION GROUPS (INTEGER)
C      QINIT        LOGICAL INITIALIZATION INDICATOR--.TRUE. FOR THE
C                   INITIAL POLLUTANT SPECIES THEN .FALSE. THEREAFTER
C
C      CALLED BY    MAIN PROGRAM
C
C      REAL TDA(4),TDD(4),TDR(4),CR(5),RUF(5),BCR(5)
C      LOGICAL QINIT,QINHLE,QINGST,QDERML,QGRW,QSURFW
C      COMMON /DR/TDA,TDD,TDR,EDA,EDAD,EDAR,GS,CR,BCR,RUF,NRL
C      COMMON /N/NPG,NPR,PL
C      COMMON /Q/QINIT,QINHLE,QINGST,QDERML,QGRW,QSURFW
C
C      STEP 1      IF THIS IS THE INITIAL POLLUTANT SPECIES, READ
C                   BASIC EXPOSURE INFORMATION. OTHERWISE, BRANCH TO
C                   STEP 2.
C
C      IF (.NOT. QINIT) GO TO 10
C      READ (5,5001) (TDA(I),I=1,NPG)
C      READ (5,5001) (TDD(I),I=1,NPG)
C      READ (5,5001) (TDR(I),I=1,NPG)
C      READ (5,5001) EDA,EDAD,EDAR,GS
C
C      STEP 2      READ POLLUTANT CONCENTRATIONS IN RECREATIONAL AREAS.
C
C      10 READ (5,5002) NRL,(CR(I),I=1,NRL)
C      READ (5,5001) (BCR(I),I=1,NRL)
C
C      STEP 3      IF THIS IS THE INITIAL POLLUTANT SPECIES, READ THE
C                   RELATIVE USE FRACTIONS FOR RECREATIONAL AREAS
C
C      IF (QINIT) READ (5,5003) (RUF(I),I=1,NRL)
C      RETURN
C
C      FORMAT STATEMENTS
C
C      5001 FORMAT (6(E12.5,1X))
C      5002 FORMAT (15,1X,5(E12.5,1X))
C      5003 FORMAT (16F5.0)
C      END
C
C      SUBROUTINE WRCASE(NAO,NAP,NVS)
C
C      TITLE (WRITE CASE DESCRIPTION)
C
C      SUBROUTINE WRCASE PRINTS A GENERAL DESCRIPTION OF THE CASE
C
C      PARAMETER    DESCRIPTION
C      NAO          NUMBER OF SPECIES OF AQUATIC ORGANISMS (INTEGER)
C      NAP          NUMBER OF ANIMAL PRODUCTS (INTEGER)

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C     NVS             NUMBER OF VEGETABLE PRODUCTS (INTEGER)
C
C     COMMON          DESCRIPTION
C     NPG             NUMBER OF POPULATION GROUPS (INTEGER)
C     NPR             NUMBER OF POPULATION SUBREGIONS (INTEGER)
C     CASE            DESCRIPTION OF CASE (2-DIM REAL ARRAY)
C     AQUAT           NAME OF AQUATIC ORGANISMS (2-DIM REAL ARRAY)
C     ANIMAL          NAME OF ANIMAL PRODUCTS (2-DIM REAL ARRAY)
C     VEGET           NAME OF VEGETABLE PRODUCTS (2-DIM REAL ARRAY)
C     POPGRP          NAME OF POPULATION GROUPS (2-DIM REAL ARRAY)
C     P               POPULATION BY SUBREGION AND GROUP (2-DIM REAL ARRAY)
C     TPR             TOTAL POPULATION BY SUBREGION (1-DIM REAL ARRAY)
C     TP              TOTAL POPULATION BY GROUP (1-DIM REAL ARRAY)
C     TPS             TOTAL POPULATION
C     QINHLE          LOGICAL INHALATION EXPOSURE INDICATOR--.TRUE. IF
C                     INHALATION EXPOSURE IS TO BE CALCULATED
C     QINGST          LOGICAL INGESTION EXPOSURE INDICATOR
C     QDERML          LOGICAL DERMAL EXPOSURE INDICATOR
C     QGRW            LOGICAL GROUNDWATER USE INDICATOR
C     QSURFW          LOGICAL SURFACE WATER USE INDICATOR
C
C     VARIABLE        DESCRIPTION
C     TEMP            POPULATION SUMMATION VARIABLE
C
C     CALLED BY      MAIN PROGRAM
C
C     REAL CASE(4,18),AQUAT(2,5),ANIMAL(2,10),VEGET(2,10),POPGRP(2,4),
1     P(4,100),TPR(100)
C     LOGICAL QINIT,QINHLE,QINGST,QDERML,QGRW,QSURFW
C     COMMON /N/NPG,NPR,PL
C     COMMON /NAME/CASE,AQUAT,ANIMAL,VEGET
C     COMMON /POP/POPGRP,P,TPR,TPS,TP(4)
C     COMMON /Q/QINIT,QINHLE,QINGST,QDERML,QGRW,QSURFW
C
C     STEP 1          WRITE GENERAL DESCRIPTION OF CASE AND HEADING FOR
C                     EXPOSURE DESCRIPTION
C
C     DO 10 I = 1,4
10  WRITE (6,6001) (CASE(I,J),J=1,18)
C     WRITE (6,6002)
C
C     STEP 2          LIST INHALATION, IF APPROPRIATE. OTHERWISE, BRANCH
C                     TO STEP 3.
C
C     IF (QINHLE)     WRITE (6,6003)
C
C     STEP 3          DESCRIBE INGESTION EXPOSURE PATHWAYS AND TYPES OF
C                     FOOD CONSUMED. IF INGESTION IS NOT CONSIDERED,
C                     BRANCH TO STEP 4.
C
C     IF (.NOT. QINGST) GO TO 20
C     WRITE (6,6004)

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      IF (QSURFW) WRITE (6,6005)
      IF (QGRW) WRITE (6,6006)
      IF (NAO .NE. 0) WRITE (6,6007) ((AQUAT(J,I),J=1,2),I=1,NAO)
      IF (NAP .NE. 0) WRITE (6,6008) ((ANIMAL(J,I),J=1,2),I=1,NAP)
      IF (NVS .NE. 0) WRITE (6,6009) ((VEGET(J,I),J=1,2),I=1,NVS)
C
C   STEP 4      LIST DERMAL EXPOSURE PATHWAY, IF APPROPRIATE.
C
20 IF (QDERML) WRITE (6,6010)
C
C   STEP 5      WRITE HEADING FOR POPULATION DESCRIPTION AND SET
C               TEMPORARY SUMMATION VARIABLE TO ZERO.
C
      WRITE (6,6011) ((POPGRP(J,I),J=1,2),I=1,NPG)
      TEMP = 0.
C
C   STEP 6      IF THERE ARE MORE THAN 50 SUBREGIONS, BRANCH TO
C               STEP 7. OTHERWISE, SUM POPULATION OVER SUBREGIONS,
C               WRITE SUBREGION AND CUMULATIVE POPULATIONS, AND
C               BRANCH TO STEP 9.
C
      IF (NPR .GT. 50) GO TO 40
      DO 30 I = 1,NPR
      TEMP = TEMP+TPR(I)
30 WRITE (6,6012) I,(P(J,I),J=1,NPG),TPR(I),TEMP
      GO TO 70
C
C   STEP 7      IF MORE THAN 50 SUBREGIONS, SUM POPULATION OVER
C               SUBREGIONS IN GROUPS OF 50 AND WRITE SUBREGION AND
C               CUMULATIVE POPULATIONS.
C
40 DO 50 I = 1,50
      TEMP = TEMP+TPR(I)
50 WRITE (6,6012) I,(P(J,I),J=1,NPG),TPR(I),TEMP
      WRITE (6,6011) ((POPGRP(J,I),J=1,2),I=1,NPG)
      DO 60 I = 51,NPR
      TEMP = TEMP+TPR(I)
60 WRITE (6,6012) I,(P(J,I),J=1,NPG),TPR(I),TEMP
C
C   STEP 8      WRITE TOTAL POPULATION AND POPULATION BY GROUP.
C
70 WRITE (6,6013) (TP(J),J=1,NPG),TPS
      RETURN
C
C   FORMAT STATEMENTS
C
6001 FORMAT (26X,20A4)
6002 FORMAT (1H0,/,/,47X,38HEXPOSURE MODES AND PATHWAYS CONSIDERED,/)
6003 FORMAT (10X,36HINHALATION OF GASES AND PARTICULATES)
6004 FORMAT (10X,43HINGESTION OF PARTICULATES, WATER, AND FOOD ,
1      8HPRODUCTS)
6005 FORMAT (15X,13HSURFACE WATER)

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6006 FORMAT (15X,11HGROUNDWATER)
6007 FORMAT (15X,20HAQUATIC ORGANISMS - ,5(2A4,1X))
6008 FORMAT (15X,18HANIMAL PRODUCTS - ,10(2A4,1X))
6009 FORMAT (15X,21HVEGETABLE PRODUCTS - ,10(2A4,1X))
6010 FORMAT (10X,40HDERMAL CONTACT WITH WATER AND ATMOSPHERE)
6011 FORMAT (1H1,44X,42HPOPULATION DESCRIPTION FOR RISK ASSESSMENT,//,
1      5X,9HSUBREGION,11X,2A4,9X,2A4,9X,2A4,9X,2A4,17X,5HTOTAL,
2      12X,10HCUMULATIVE,/)
6012 FORMAT (8X,13,10X,4(1PE13.5,4X),2(7X,1PE13.5))
6013 FORMAT (1H0,3X,11HGROUP TOTAL,6X,4(1PE13.5,4X),7X,1PE13.5)
END

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SUBROUTINE WRPOLL(I)

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C
C  TITLE (WRITE POLLUTANT CONCENTRATIONS)
C
C  SUBROUTINE WRPOLL PRINTS POLLUTANT CONCENTRATIONS FOR THE CURRENT
C  POLLUTANT SPECIES.
C
C  PARAMETER      DESCRIPTION
C  I              INDEX SPECIFYING CURRENT POLLUTANT SPECIES (INTEGER)
C
C  COMMON         DESCRIPTION
C  C              POLLUTANT CONCENTRATIONS BY SUBREGION AND MEDIUM FOR
C  C              CURRENT POLLUTANT SPECIES (2-DIM REAL ARRAY)
C  BC             BACKGROUND POLLUTANT CONCENTRATIONS BY SUBREGION AND
C  BC             MEDIUM FOR CURRENT SPECIES (2-DIM REAL ARRAY)
C  NPR            NUMBER OF POPULATION SUBREGIONS (INTEGER)
C  POLLUT         NAME OF POLLUTANT SPECIES (2-DIM REAL ARRAY)
C
C  CALLED BY      MAIN PROGRAM
C
C  REAL POLLUT(2,5),C(5,100),BC(5,100)
C  COMMON /CON/C,BC
C  COMMON /N/NPG,NPR,PL
C  COMMON /POL/POLLUT
C  STEP 1         WRITE PAGE TITLE AND NAME OF POLLUTANT SPECIES.  IF
C  C              THERE ARE MORE THAN 50 SUBREGIONS, BRANCH TO STEP 3.
C
C  WRITE (6,6000) (POLLUT(J,I),J=1,2)
C  IF (NPR .GT. 50) GO TO 20
C
C  STEP 2         WRITE PLANT AND BACKGROUND POLLUTANT CONCENTRATIONS
C  C              BY SUBREGION, THEN RETURN.
C
C  DO 10 J = 1,NPR
10 WRITE (6,6001) J,(C(K,J),BC(K,J),K=1,5)
C  RETURN
C
C  STEP 3         IF MORE THAN 50 SUBREGIONS, WRITE PLANT AND BACK-
C  C              GROUND POLLUTANT CONCENTRATIONS BY SUBREGION IN
C  C              GROUPS OF 50.
C

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C
20 DO 30 J = 1,50
30 WRITE (6,6001) J,(C(K,J),BC(K,J),K=1,5)
   WRITE (6,6000) (POLLUT(J,I),J=1,2)
   DO 40 J = 51,NPR
40 WRITE (6,6001) J,(C(K,J),BC(K,J),K=1,5)
   RETURN

C
C   FORMAT STATEMENTS
C
6000 FORMAT (1H1,39X,43HENVIRONMENTAL CONCENTRATIONS FOR SPECIES - ,
1         2A4,/,31X,11HATMOSPHERIC,24X,12HSURFACE SOIL,12X,
2         8HSURFACE ,5HWATER,12X,11HGROUNDWATER,/,1X,10HPOPULATION,
3         12X,5HGASES,5X,7H(MG/M3),4X,12HPARTICULATES,12X,
4         7H(MG/KG),18X,6H(MG/L),18X,6H(MG/L),/,1X,9HSUBREGION,
5         1X,5(5X,4HCASE,5X,10HBACKGROUND),/)
6001 FORMAT (4X,I3,5X,10(1PE12.5))
      END

      SUBROUTINE CALINH(I)

C
C   TITLE (CALCULATE INHALATION EXPOSURE)
C
C   SUBROUTINE CALINH CALCULATES INHALATION EXPOSURE RATES BY
C   SUBREGION AND POPULATION GROUP FOR THE CURRENT POLLUTANT SPECIES.
C
C   PARAMETER      DESCRIPTION
C   I              INDEX SPECIFYING CURRENT POLLUTANT SPECIES (INTEGER)
C
C   COMMON         DESCRIPTION
C   C              POLLUTANT CONCENTRATIONS BY SUBREGION AND MEDIUM FOR
C                   CURRENT POLLUTANT SPECIES (2-DIM REAL ARRAY)
C   BC             BACKGROUND POLLUTANT CONCENTRATIONS BY SUBREGION AND
C                   MEDIUM FOR CURRENT SPECIES (2-DIM REAL ARRAY)
C   IH             INHALATION EXPOSURE RATE (2-DIM REAL ARRAY)
C   BIH            BACKGROUND INHALATION EXPOSURE RATE (2-DIM REAL
C                   ARRAY)
C   NPG            NUMBER OF POPULATION GROUPS (INTEGER)
C   NPR            NUMBER OF POPULATION SUBREGIONS (INTEGER)
C
C   CALLED BY      MAIN PROGRAM
C
      REAL C(5,100),BC(5,100),IH(5,4,100),BIH(5,4,100)
      COMMON /CON/C,BC
      COMMON /EXIH/IH,BIH
      COMMON /N/NPG,NPR,PL

C
C   STEP 1          CALCULATE PLANT AND BACKGROUND POLLUTANT INHALATION
C                   EXPOSURE RATES BY SUBREGION AND POPULATION GROUP
C                   FROM GASEOUS AND PARTICULATE CONCENTRATIONS.
C
      DO 10 J = 1,NPG

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DO 10 K = 1,NPR
BIH(I,J,K) = (BC(1,K)+BC(2,K))
IH(I,J,K) = (C(1,K)+C(2,K))
10 CONTINUE
RETURN
END

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SUBROUTINE CALING(I,NAO,NAP,NVS)

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C
C TITLE (CALCULATE INGESTION EXPOSURE)
C
C SUBROUTINE CALING CALCULATES BASIC INGESTION EXPOSURE RATES BY
C POPULATION GROUP AND SUBREGION FOR THE CURRENT POLLUTANT SPECIES.
C
C PARAMETER      DESCRIPTION
C I              INDEX SPECIFYING CURRENT POLLUTANT SPECIES (INTEGER)
C NAO            NUMBER OF SPECIES OF AQUATIC ORGANISMS (INTEGER)
C NAP            NUMBER OF ANIMAL PRODUCTS (INTEGER)
C NVS            NUMBER OF VEGETABLE PRODUCTS (INTEGER)
C
C COMMON
C C              POLLUTANT CONCENTRATIONS BY SUBREGION AND MEDIUM FOR
C                  CURRENT POLLUTANT SPECIES (2-DIM REAL ARRAY)
C BC             BACKGROUND POLLUTANT CONCENTRATIONS BY SUBREGION AND
C                  MEDIUM FOR CURRENT SPECIES (2-DIM REAL ARRAY)
C TF             FRACTION OF POLLUTANT SPECIES REMAINING AFTER
C                  DRINKING WATER TREATMENT (1-DIM REAL ARRAY)
C VF             FRACTION OF DOMESTIC WATER CONSUMED BY SUBREGION THAT
C                  COMES FROM SURFACE WATER SOURCES (1-DIM REAL ARRAY)
C IWT            DRINKING WATER TREATMENT PROCESSES USED WITHIN THE
C                  SUBREGION FOR SURFACE AND GROUNDWATER SOURCES (2-DIM
C                  INTEGER ARRAY)
C BIG            BACKGROUND INGESTION EXPOSURE FROM FOOD TYPES NOT
C                  CONSIDERED IN ANALYSIS (1-DIM REAL ARRAY)
C IGP            INGESTION EXPOSURE RATE FOR ATMOSPHERIC PARTICULATES
C                  (3-DIM REAL ARRAY)
C BIGP           BACKGROUND INGESTION EXPOSURE RATE FOR ATMOSPHERIC
C                  PARTICULATES (3-DIM REAL ARRAY)
C IGW            INGESTION EXPOSURE RATE FOR DRINKING WATER (3-DIM
C                  REAL ARRAY)
C BIGW           BACKGROUND INGESTION EXPOSURE RATE FOR DRINKING
C                  WATER (3-DIM REAL ARRAY)
C UI             INHALATION RATES BY POPULATION GROUP (1-DIM REAL
C                  ARRAY)
C TU            TOTAL FOOD CONSUMPTION RATES BY POPULATION GROUP
C                  (1-DIM REAL ARRAY)
C UW            DRINKING WATER CONSUMPTION RATES BY POPULATION GROUP
C                  (1-DIM REAL ARRAY)
C PIG           FRACTION OF INHALED PARTICULATES THAT IS INGESTED
C BMF           MEAN BACKGROUND POLLUTANT CONCENTRATION IN FOOD FROM
C                  OUTSIDE THE POWER PLANT REGION
C NPG           NUMBER OF POPULATION GROUPS (INTEGER)

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C   NPR          NUMBER OF POPULATION SUBREGIONS (INTEGER)
C   UA           CONSUMPTION OF ANIMAL PRODUCTS BY POPULATION GROUP
C                 (2-DIM REAL ARRAY)
C   UQ           CONSUMPTION OF AQUATIC ORGANISMS BY POPULATION GROUP
C                 (2-DIM REAL ARRAY)
C   UV           CONSUMPTION OF VEGETABLE PRODUCTS BY POPULATION
C                 GROUP (2-DIM REAL ARRAY)
C
C   EXTERNALS    DESCRIPTION
C   CALAQ        SUBROUTINE TO CALCULATE EXPOSURE FROM INGESTION OF
C                 AQUATIC ORGANISMS
C   CALVEG       SUBROUTINE TO CALCULATE EXPOSURE FROM INGESTION OF
C                 VEGETABLE PRODUCTS
C   CALAN        SUBROUTINE TO CALCULATE EXPOSURE FROM INGESTION OF
C                 ANIMAL PRODUCTS
C
C   VARIABLE     DESCRIPTION
C   TUT          TOTAL CONSUMPTION RATE FOR FOOD PRODUCTS CONSIDERED
C                 IN THE ANALYSIS (1-DIM REAL ARRAY)
C   IS           INDEX SPECIFYING CURRENT TREATMENT PROCESS FOR
C                 SURFACE WATER (INTEGER)
C   IG           INDEX SPECIFYING THE CURRENT TREATMENT PROCESS
C                 FOR GROUNDWATER (INTEGER)
C
C   CALLED BY    MAIN PROGRAM
C
C   REAL TU(4),TUT(4),UQ(4,5),UA(4,10),UV(4,10),BIG(4),UI(4),UW(4),
1     IGP(5,4,100),BIGP(5,4,100),C(5,100),BC(5,100),IGW(5,4,100),
2     BIGW(5,4,100),VF(100),TF(10)
C   INTEGER IWT(2,100)
C   COMMON /CON/C,BC
C   COMMON /DW/TF,VF,IWT,NTP
C   COMMON /EXIGB/BIG,IGP,BIGP,IGW,BIGW
C   COMMON /IGD/UI,W(4),TU,UW,PIG,BMF
C   COMMON /N/NPG,NPR,PL
C   COMMON /U/UA,UQ,UV
C
C   STEP 1       DETERMINE CONSUMPTION OF FOOD PRODUCTS NOT INCLUDED
C                 IN THE ANALYSIS.  CALCULATE POLLUTANT EXPOSURE BY
C                 POPULATION GROUP FROM THIS ADDITIONAL CONSUMPTION.
C
C   DO 40 J = 1,NPG
C     TUT(J) = 0.
C     DO 10 K = 1,NAO
10    TUT(J) = TUT(J)+UQ(J,K)
C     DO 20 K = 1,NAP
20    TUT(J) = TUT(J)+UA(J,K)
C     DO 30 K = 1,NVS
30    TUT(J) = TUT(J)+UV(J,K)
40    BIG(J) = (TU(J)-TUT(J))*BMF
C
C   STEP 2       CALCULATE PLANT AND BACKGROUND POLLUTANT EXPOSURE

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C          (BY GROUP AND SUBREGION) FOR INGESTION OF ATMOSPHERIC
C          PARTICULATES.
C
C          DO 50 J = 1,NPG
C          DO 50 K = 1,NPR
C          BIGP(I,J,K) = BC(2,K)*PIG*UI(J)
50 IGP(I,J,K) = C(2,K)*PIG*UI(J)
C
C          STEP 3          DETERMINE PLANT AND BACKGROUND POLLUTANT EXPOSURE
C                          FOR CONSUMPTION OF DRINKING WATER.  FOR EACH SUB-
C                          REGION, PARTITION TOTAL CONSUMPTION BETWEEN SURFACE
C                          AND GROUNDWATER SOURCES AND APPLY APPROPRIATE WATER
C                          TREATMENT PROCESS EFFICIENCIES.
C
C          DO 60 J = 1,NPG
C          DO 60 K = 1,NPR
C          IS = IWT(1,K)
C          IG = IWT(2,K)
C          BIGW(I,J,K) = BC(4,K)*UW(J)*VF(K)*TF(IS)+BC(5,K)
C                          *UW(J)*TF(IG)*(1.0-VF(K))
60 IGW(I,J,K) = C(4,K)*UW(J)*VF(K)*TF(IS)+C(5,K)*UW(J)*TF(IG)*
C          (1.0-VF(K))
C
C          STEP 4          IF APPROPRIATE, CALL CALAQ TO CALCULATE EXPOSURE
C                          FROM INGESTION OF AQUATIC ORGANISMS.  OTHERWISE,
C                          PROCEED TO STEP 5.
C
C          IF (NAO .NE. 0)  CALL CALAQ(I,NAO)
C
C          STEP 5          IF APPROPRIATE, CALL CALVEG TO CALCULATE EXPOSURE
C                          FROM INGESTION OF VEGETABLE PRODUCTS.  OTHERWISE,
C                          PROCEED TO STEP 6.
C
C          IF (NVS .NE. 0)  CALL CALVEG(I,NVS)
C
C          STEP 6          IF APPROPRIATE, CALL CALAN TO CALCULATE EXPOSURE
C                          FROM INGESTION OF ANIMAL PRODUCTS.  OTHERWISE,
C                          RETURN.
C
C          IF (NAP .NE. 0)  CALL CALAN(I,NAP,NVS)
C          RETURN
C          END
C
C          SUBROUTINE CALAQ(I,NAO)
C
C          TITLE (CALCULATE AQUATIC INGESTION EXPOSURE)
C
C          SUBROUTINE CALAQ DETERMINES BACKGROUND AND POLLUTANT EXPOSURE
C          RATES FROM CONSUMPTION OF AQUATIC ORGANISMS.
C
C          PARAMETER      DESCRIPTION
C          I               INDEX SPECIFYING CURRENT POLLUTANT SPECIES (INTEGER)

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C      NAO          NUMBER OF SPECIES OF AQUATIC ORGANISMS (INTEGER)
C
C      COMMON      DESCRIPTION
C      PQ          REGIONAL PRODUCTION OF AQUATIC ORGANISMS (1-DIM REAL
C                  ARRAY)
C      CQ          MEAN SURFACE WATER POLLUTANT CONCENTRATION WHERE
C                  AQUATIC ORGANISMS ARE FOUND (1-DIM REAL ARRAY)
C      BCQ         MEAN SURFACE WATER BACKGROUND POLLUTANT CONCENTRATION
C                  WHERE AQUATIC ORGANISMS ARE FOUND (1-DIM REAL ARRAY)
C      B           BIOCONCENTRATION FACTOR FOR AQUATIC ORGANISMS (1-DIM
C                  REAL ARRAY)
C      QCB         POLLUTANT CONCENTRATION IN SUPPLY OF ORGANISMS FROM
C                  OUTSIDE POWER PLANT REGION (1-DIM REAL ARRAY)
C      IGQ         TOTAL INGESTION EXPOSURE RATE FOR AQUATIC ORGANISMS
C                  (3-DIM REAL ARRAY)
C      BIGQ        TOTAL BACKGROUND INGESTION EXPOSURE RATE FOR AQUATIC
C                  ORGANISMS (3-DIM REAL ARRAY)
C      NPG         NUMBER OF POPULATION GROUPS (INTEGER)
C      NPR         NUMBER OF POPULATION SUBREGIONS (INTEGER)
C      TP          TOTAL POPULATION BY GROUP (1-DIM REAL ARRAY)
C      UQ          CONSUMPTION OF AQUATIC ORGANISMS BY POPULATION GROUP
C                  (2-DIM REAL ARRAY)
C
C      VARIABLE    DESCRIPTION
C      TUQ         REGIONAL CONSUMPTION OF AQUATIC ORGANISMS (1-DIM
C                  REAL ARRAY)
C      LQ          RATIO OF LOCAL CONSUMPTION TO LOCAL PRODUCTION OF
C                  AQUATIC ORGANISMS (1-DIM REAL ARRAY)
C      SIGQ        INGESTION EXPOSURE RATE BY TYPE OF AQUATIC ORGANISM
C                  (2-DIM REAL ARRAY)
C      BSIGQ       BACKGROUND INGESTION EXPOSURE RATE BY TYPE OF AQUATIC
C                  ORGANISM (2-DIM REAL ARRAY)
C
C      CALLED BY   CALING
C
C      REAL TUQ(5),TP(4),UQ(4,5),LQ(5),PQ(5),BSIGQ(4,5),SIGQ(4,5),
1      BCQ(5),B(5),QCB(5),CQ(5),IGQ(5,4,100),BIGQ(5,4,100)
C      COMMON /AQ/PQ,CQ,BCQ,B,QCB
C      COMMON /EXAQ/IGQ,BIGQ
C      COMMON /N/NPG,NPR,PL
C      COMMON /POP/POPGRP(2,4),P(4,100),TPR(100),TPS,TP
C      COMMON /U/UA(4,10),UQ,UV(4,10)
C
C      STEP 1      CALCULATE TOTAL REGIONAL CONSUMPTION BY TYPE OF
C                  AQUATIC ORGANISM.
C
C      DO 10 J = 1,NAO
C      TUQ(J) = 0.
C      DO 10 K = 1,NPG
10  TUQ(J) = TUQ(J)+TP(K)*UQ(K,J)
C
C      STEP 2      DETERMINE THE RATIO OF REGIONAL CONSUMPTION TO

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C          REGIONAL PRODUCTION FOR EACH ORGANISM.
C
      DO 20 J = 1,NAO
20  LQ(J) = TUQ(J)/PQ(J)
C
C      STEP 3      CALCULATE PLANT AND BACKGROUND POLLUTANT EXPOSURE
C                  RATES BY POPULATION GROUP FOR CONSUMPTION OF EACH
C                  ORGANISM.  IF REGIONAL PRODUCTION IS LESS THAN
C                  CONSUMPTION, INCLUDE THE CONTRIBUTION OF ORGANISMS
C                  SUPPLIED FROM OUTSIDE THE REGION.  SUM OVER AQUATIC
C                  ORGANISMS TO DETERMINE TOTAL EXPOSURE RATES.
C
      DO 50 K = 1,NPR
      DO 40 L = 1,NPG
      BIGQ(I,L,K) = 0.
      IGQ(I,L,K) = 0.
      DO 30 J = 1,NAO
      IF (LQ(J) .LE. 1.0)  BSIGQ(L,J) = BCQ(J)*B(J)*UQ(L,J)
      IF (LQ(J) .GT. 1.0)  BSIGQ(L,J) = (UQ(L,J)/LQ(J))*(BCQ(J)*B(J)
1      -QCB(J))+QCB(J)*UQ(L,J)
      IF (LQ(J) .LE. 1.0)  SIGQ(L,J) = CQ(J)*B(J)*UQ(L,J)
      IF (LQ(J) .GT. 1.0)  SIGQ(L,J) = (UQ(L,J)/LQ(J))*(CQ(J)*B(J)
1      -QCB(J))+QCB(J)*UQ(L,J)
      BIGQ(I,L,K) = BIGQ(I,L,K)+BSIGQ(L,J)
30  IGQ(I,L,K) = IGQ(I,L,K)+SIGQ(L,J)
40  CONTINUE
50  CONTINUE
      RETURN
      END

      SUBROUTINE CALVEG(I,NVS)
C
C      TITLE (CALCULATE VEGETABLE INGESTION EXPOSURE)
C
C      SUBROUTINE CALVEG CALCULATES BACKGROUND AND POLLUTANT EXPOSURE
C      RATES FROM CONSUMPTION OF VEGETABLE PRODUCTS.
C
C      PARAMETER      DESCRIPTION
C      I              INDEX SPECIFYING CURRENT POLLUTANT SPECIES (INTEGER)
C      NVS            NUMBER OF VEGETABLE PRODUCTS (INTEGER)
C
C      COMMON          DESCRIPTION
C      C              POLLUTANT CONCENTRATIONS BY SUBREGION AND MEDIUM FOR
C                      CURRENT POLLUTANT SPECIES (2-DIM REAL ARRAY)
C      BC              BACKGROUND POLLUTANT CONCENTRATIONS BY SUBREGION AND
C                      MEDIUM FOR CURRENT SPECIES (2-DIM REAL ARRAY)
C      AFD             ATMOSPHERIC PARTICULATE DEPOSITION RATES BY SUBREGION
C                      FOR CURRENT SPECIES (1-DIM REAL ARRAY)
C      BAFD            BACKGROUND PARTICULATE DEPOSITION RATES BY SUBREGION
C                      FOR CURRENT SPECIES (1-DIM REAL ARRAY)
C      IGV             TOTAL INGESTION EXPOSURE RATE FOR VEGETABLE PRODUCTS
C                      (3-DIM REAL ARRAY)

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C	BIGV	TOTAL BACKGROUND INGESTION EXPOSURE RATE FOR
C		VEGETABLE PRODUCTS (3-DIM REAL ARRAY)
C	NPG	NUMBER OF POPULATION GROUPS (INTEGER)
C	NPR	NUMBER OF POPULATION SUBREGIONS (INTEGER)
C	TP	TOTAL POPULATION BY GROUP (1-DIM REAL ARRAY)
C	QGRW	LOGICAL GROUNDWATER USE INDICATOR
C	QSURFW	LOGICAL SURFACE WATER USE INDICATOR
C	UV	CONSUMPTION OF VEGETABLE PRODUCTS BY POPULATION
C		GROUP (2-DIM REAL ARRAY)
C	VC	CONTRIBUTION TO POLLUTANT CONCENTRATION IN VEGETABLE
C		PRODUCTS FROM SOIL UPTAKE (2-DIM REAL ARRAY)
C	BVC	BACKGROUND CONTRIBUTION TO POLLUTANT CONCENTRATION IN
C		VEGETABLE PRODUCTS FROM SOIL UPTAKE (2-DIM REAL
C		ARRAY)
C	VSC	CONTRIBUTION TO POLLUTANT CONCENTRATION IN VEGETABLE
C		PRODUCTS FROM SURFACE DEPOSITION (2-DIM REAL ARRAY)
C	BVSC	BACKGROUND CONTRIBUTION TO POLLUTANT CONCENTRATION IN
C		VEGETABLE PRODUCTS FROM SURFACE DEPOSITION (2-DIM
C		REAL ARRAY)
C	CV	BIOCONCENTRATION FACTOR FOR VEGETABLE PRODUCTS
C		(1-DIM REAL ARRAY)
C	FC	CAPTURE FRACTION FOR VEGETABLE PRODUCTS (1-DIM REAL
C		ARRAY)
C	VG	VEGETATIVE SURFACE DENSITY (1-DIM REAL ARRAY)
C	VCB	BACKGROUND POLLUTANT CONCENTRATION IN PRODUCTS FROM
C		OUTSIDE THE POWER PLANT REGION (1-DIM REAL ARRAY)
C	RPV	SUBREGIONAL PRODUCTION OF VEGETABLE PRODUCTS (2-DIM
C		REAL ARRAY)
C	FI	FRACTION OF VEGETABLE PRODUCTION IRRIGATED (3-DIM
C		REAL ARRAY)
C	CI	POLLUTANT CONCENTRATION IN IRRIGATION WATER (2-DIM
C		REAL ARRAY)
C	BCI	BACKGROUND POLLUTANT CONCENTRATION IN IRRIGATION
C		WATER (2-DIM REAL ARRAY)
C	IRR	IRRIGATION RATE FOR VEGETABLE PRODUCTS (1-DIM REAL
C		ARRAY)
C	TL	TRANSLOCATION FACTOR FOR POLLUTANT MOVEMENT TO EDIBLE
C		PARTS OF PRODUCTS (1-DIM REAL ARRAY)
C	WL	WEATHERING LOSS RATE FROM VEGETATION SURFACES
C		
C	VARIABLE	DESCRIPTION
C	FD	POLLUTANT DEPOSITION FLUX FROM IRRIGATION WATER
C		(3-DIM REAL ARRAY)
C	BFD	BACKGROUND DEPOSITION FLUX FROM IRRIGATION WATER
C		(3-DIM REAL ARRAY)
C	TEMP	SUMMATION VARIABLE FOR POLLUTANT IN VEGETABLE
C		PRODUCTS
C	BTEMP	SUMMATION VARIABLE FOR BACKGROUND POLLUTANT IN
C		VEGETABLE PRODUCTS
C	PV	TOTAL REGIONAL PRODUCTION OF VEGETABLE PRODUCTS
C		(1-DIM REAL ARRAY)
C	MVC	MEAN POLLUTANT CONCENTRATION IN VEGETABLE PRODUCTS

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C          (1-DIM REAL ARRAY)
C      BMVC      MEAN BACKGROUND POLLUTANT CONCENTRATION IN VEGETABLE
C                  PRODUCTS (1-DIM REAL ARRAY)
C      TUV       REGIONAL CONSUMPTION OF VEGETABLE PRODUCTS (1-DIM
C                  REAL ARRAY)
C      LV        RATIO OF LOCAL CONSUMPTION TO LOCAL PRODUCTION OF
C                  VEGETABLE PRODUCTS (1-DIM REAL ARRAY)
C      SIGV      INGESTION EXPOSURE RATE BY VEGETABLE PRODUCT (2-DIM
C                  REAL ARRAY)
C      BSIGV     BACKGROUND INGESTION EXPOSURE RATE BY VEGETABLE
C                  PRODUCT (2-DIM REAL ARRAY)
C
C      CALLED BY  CALING
C
      REAL BFD(2,10,100),FD(2,10,100),BCI(2,100),IRR(10),FI(2,10,100),
1      CI(2,100),BVSC(10,100),BVC(10,100),VSC(10,100),VC(10,100),
2      FC(10),TL(10),VG(10),BAFD(100),VCB(10),CV(10),AFD(100),MFC,
3      C(5,100),PV(10),RPV(10,100),MVC(10),BMVC(10),TUV(10),LV(10),
4      BC(5,100),BIGV(5,4,100),IGV(5,4,100),BSIGV(4,10),SIGV(4,10)
      LOGICAL QINIT,QINHLE,QINGST,QDERML,QGRW,QSURFW
      COMMON /CON/C,BC
      COMMON /DEP/AFD,BAFD
      COMMON /EXVG/IGV,BIGV
      COMMON /N/NPG,NPR,PL
      COMMON /POP/POPGRP(2,4),P(4,100),TPR(100),TPS,TP(4)
      COMMON /Q/QINIT,QINHLE,QINGST,QDERML,QGRW,QSURFW
      COMMON /U/UA(4,10),UQ(4,5),UV(4,10)
      COMMON /VEGCN/VC,BVC,VSC,BVSC
      COMMON /VG/CV,FC,VG,VCB,RPV,FI,CI,BCI,MFC,IRR,TL,SD,TS,WL,NVSR
C
C      STEP 1      CALCULATE PLANT AND BACKGROUND POLLUTANT DEPOSITION
C                  FLUX FROM IRRIGATION.  FOR SURFACE AND GROUNDWATER,
C                  IF APPROPRIATE, USE THE IRRIGATION RATE, FRACTION OF
C                  CROP IRRIGATED, AND POLLUTANT CONCENTRATION IN THE
C                  IRRIGATION WATER OF EACH SUBREGION.
C
      DO 10 J = 1,NVS
      DO 10 K = 1,NPR
      DO 10 L = 1,2
      BFD(L,J,K) = 0.
      FD(L,J,K) = 0.
      IF ((L.EQ. 1) .AND. (.NOT. QSURFW)) GO TO 10
      IF ((L.EQ. 2) .AND. (.NOT. QGRW)) GO TO 10
      BFD(L,J,K) = BCI(L,K)*IRR(J)*FI(L,J,K)
      FD(L,J,K) = CI(L,K)*IRR(J)*FI(L,J,K)
10  CONTINUE
C
C      STEP 2      FIND PLANT AND BACKGROUND POLLUTANT CONCENTRATIONS
C                  IN EACH PRODUCT BY SUBREGION.  CALCULATE THESE
C                  CONCENTRATIONS FROM SURFACE DEPOSITION AND SOIL
C                  UPTAKE.
C

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DO 20 K = 1,NPR
DO 20 J = 1,NVS
BVSC(J,K) = (FC(J)*TL(J)/(VG(J)*WL))*(BAFD(K)+BFD(1,J,K)
1      +BFD(2,J,K))
BVC(J,K) = BC(3,K)*CV(J)
VSC(J,K) = (FC(J)*TL(J)/(VG(J)*WL))*(AFD(K)+FD(1,J,K)+FD(2,J,K))
VC(J,K) = C(3,K)*CV(J)
20 CONTINUE

C
C   STEP 3      CALCULATE MEAN PLANT AND BACKGROUND POLLUTANT CONGEN-
C               TRATIONS IN THE TOTAL REGIONAL PRODUCTION OF EACH
C               VEGETABLE PRODUCT USING A WEIGHTED SUM OVER THE
C               SUBREGIONS.
C
DO 40 J = 1,NVS
PV(J) = 0.
TEMP = 0.
BTEMP = 0.
DO 30 K = 1,NPR
PV(J) = PV(J)+RPV(J,K)
TEMP = TEMP+RPV(J,K)*(VC(J,K)+VSC(J,K))
BTEMP = BTEMP+RPV(J,K)*(BVC(J,K)+BVSC(J,K))
30 CONTINUE
MVC(J) = TEMP/PV(J)
BMVC(J) = BTEMP/PV(J)
40 CONTINUE

C
C   STEP 4      DETERMINE TOTAL REGIONAL CONSUMPTION OF EACH PRODUCT
C               AND THE RATIO OF REGIONAL CONSUMPTION TO REGIONAL
C               PRODUCTION.
C
DO 60 J = 1,NVS
TUV(J) = 0.
DO 50 K = 1,NPG
50 TUV(J) = TUV(J)+TP(K)*UV(K,J)
60 LV(J) = TUV(J)/PV(J)

C
C   STEP 5      CALCULATE PLANT AND BACKGROUND POLLUTANT EXPOSURE
C               RATES BY POPULATION GROUP FOR CONSUMPTION OF EACH
C               PRODUCT. IF REGIONAL PRODUCTION IS LESS THAN
C               CONSUMPTION, INCLUDE THE CONTRIBUTION OF VEGETABLE
C               PRODUCTS SUPPLIED FROM OUTSIDE THE REGION. SUM OVER
C               PRODUCTS TO DETERMINE TOTAL EXPOSURE RATES BY
C               POPULATION GROUP AND SUBREGION.
C
DO 90 K = 1,NPR
DO 80 L = 1,NPG
BIGV(I,L,K) = 0.
IGV(I,L,K) = 0.
DO 70 J = 1,NVS
IF (LV(J) .LE. 1.0) BSIGV(L,J) = UV(L,J)*BMVC(J)
IF (LV(J) .GT. 1.0) BSIGV(L,J) = (UV(L,J)/LV(J))*(BMVC(J)

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1          -VCB(J))+VCB(J)*UV(L,J)
  IF (LV(J) .LE. 1.0)    SIGV(L,J) = UV(L,J)*MVC(J)
  IF (LV(J) .GT. 1.0)    SIGV(L,J) = (UV(L,J)/LV(J))*(MVC(J)-
1          VCB(J))+VCB(J)*UV(L,J)
  BIGV(I,L,K) = BIGV(I,L,K)+BSIGV(L,J)
  IGV(I,L,K) = IGV(I,L,K)+SIGV(L,J)
70 CONTINUE
80 CONTINUE
90 CONTINUE
  RETURN
  END

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SUBROUTINE CALAN(I,NAP,NVS)

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C
C  TITLE (CALCULATE ANIMAL INGESTION EXPOSURE)
C
C  SUBROUTINE CALAN CALCULATES BACKGROUND AND POLLUTANT EXPOSURE
C  RATES FROM CONSUMPTION OF ANIMAL PRODUCTS.
C
C  PARAMETER      DESCRIPTION
C  I              INDEX SPECIFYING CURRENT POLLUTANT SPECIES (INTEGER)
C  NAP           NUMBER OF ANIMAL PRODUCTS (INTEGER)
C  NVS           NUMBER OF VEGETABLE PRODUCTS (INTEGER)
C
C  COMMON         DESCRIPTION
C  FEED           TYPES OF FEED FOR ANIMAL PRODUCTS (3-DIM REAL ARRAY)
C  MF            FRACTION OF DAILY POLLUTANT INTAKE REACHING THE
C              ANIMAL PRODUCT (1-DIM REAL ARRAY)
C  QW            WATER CONSUMPTION RATE FOR ANIMAL PRODUCTS (1-DIM
C              REAL ARRAY)
C  QF            FEED CONSUMPTION RATE FOR ANIMAL PRODUCTS (3-DIM
C              REAL ARRAY)
C  ACB           POLLUTANT CONCENTRATION IN ANIMAL PRODUCTS PRODUCED
C              OUTSIDE THE REGION (1-DIM REAL ARRAY)
C  RPA           SUBREGIONAL PRODUCTION OF ANIMAL PRODUCTS (2-DIM
C              REAL ARRAY)
C  CW           POLLUTANT CONCENTRATION IN ANIMAL DRINKING WATER
C              (2-DIM REAL ARRAY)
C  BCW          BACKGROUND POLLUTANT CONCENTRATION IN ANIMAL DRINKING
C              WATER (2-DIM REAL ARRAY)
C  CF           POLLUTANT CONCENTRATION IN ANIMAL FEED (3-DIM REAL
C              ARRAY)
C  BCF          BACKGROUND POLLUTANT CONCENTRATION IN ANIMAL FEED
C              (3-DIM REAL ARRAY)
C  NTF          NUMBER OF TYPES OF FEED CONSUMED BY ANIMALS (2-DIM
C              INTEGER ARRAY)
C  IGA          TOTAL INGESTION EXPOSURE RATE FOR ANIMAL PRODUCTS
C              (3-DIM REAL ARRAY)
C  BIGA         TOTAL BACKGROUND INGESTION EXPOSURE RATE FOR ANIMAL
C              PRODUCTS (3-DIM REAL ARRAY)
C  NPG          NUMBER OF POPULATION GROUPS (INTEGER)
C  NPR          NUMBER OF POPULATION SUBREGIONS (INTEGER)

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5      SIGA(4,10),BSIGA(4,10),MFC
      INTEGER NTF(10,100),NVS(10),NAPR(10)
      COMMON /AN/FEED,MF,QW,ACB,RPA,QF,CW,BCW,CF,BCF,NAPR,NTF
      COMMON /EXAN/IGA,BIGA
      COMMON /N/NPG,NPR,PL
      COMMON /NAME/CASE(4,18),AQUAT(2,5),ANIMAL(2,10),VEGET
      COMMON /POP/POPGRP(2,4),P(4,100),TPR(100),TPS,TP
      COMMON /U/UA,UQ(4,5),UV(4,10)
      COMMON /VEGCN/VC,BVC,VSC,BVSC
      COMMON /VG/CV(10),FC(10),VG(10),VCB(10),RPV(10,100),FI(2,10,100),
1      CI(2,100),BCI(2,100),MFC,IRR(10),TL(10),SD,TS,WL,NVS

C
C      STAGE 1      CALCULATE PLANT AND BACKGROUND POLLUTANT CONCEN-
C                  TRATIONS IN ANIMAL PRODUCTS.
C
C      STEP 1      FOR EACH PRODUCT, INITIALIZE PRODUCTION AND TEMPORARY
C                  CONCENTRATION VARIABLES.
C
      DO 50 J = 1,NAP
      PA(J) = 0.
      TEMPA = 0.
      BTEMPA = 0.

C
C      STEP 2      CYCLE THROUGH POPULATION SUBREGIONS.  DETERMINE TOTAL
C                  REGIONAL PRODUCTION AND THE NUMBER OF TYPES OF FEED.
C
      DO 40 K = 1,NPR
      PA(J) = PA(J)+RPA(J,K)
      KU = NTF(J,K)

C
C      STEP 3      FOR EACH TYPE OF FEED CALCULATE BACKGROUND AND PLANT
C                  POLLUTANT CONCENTRATIONS BY SUBREGION, USING THE
C                  VEGETABLE PRODUCT CONCENTRATIONS WHEN APPROPRIATE.
C
      DO 20 L = 1,KU
      DO 10 M = 1,NVS
      IF ((FEED(J,K,L) .NE. VEGET(1,M)) .OR. (RPV(M,K) .EQ. 0.))
1      GO TO 10
      CF(J,K,L) = VC(M,K)+VSC(M,K)
      BCF(J,K,L) = BVC(M,K)+BVSC(M,K)
      GO TO 20
10 CONTINUE
20 CONTINUE

C
C      STEP 4      SUM POLLUTANT INTAKE RATES OVER FEED TYPES.
C
      TEMPB = 0.
      BTEMPB = 0.
      DO 30 L = 1,KU
      BTEMPB = BTEMPB+QF(J,K,L)*BCF(J,K,L)
30 TEMPB = TEMPB+QF(J,K,L)*CF(J,K,L)
C

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C      STEP 5      DETERMINE POLLUTANT CONCENTRATIONS IN ANIMAL
C                  PRODUCTS FROM FEED AND WATER INTAKE RATES AND
C                  TRANSFER FRACTIONS.
C
C      BCM(J,K) = MF(J)*(BCW(J,K)*QW(J)+BTEMPB)
C      CM(J,K) = MF(J)*(CW(J,K)*QW(J)+TEMPB)
C
C      STEP 6      ADD POLLUTANT IN THIS ANIMAL PRODUCT FROM THIS
C                  SUBREGION TO TEMPORARY SUMMATION VARIABLES.
C
C      TEMPA = TEMPA+RPA(J,K)*CM(J,K)
C      BTEMPA = BTEMPA+RPA(J,K)*BCM(J,K)
40  CONTINUE
C
C      STEP 7      CALCULATE MEAN POLLUTANT CONCENTRATIONS FOR ALL
C                  REGIONAL PRODUCTION OF THIS ANIMAL PRODUCT.
C
C      MCM(J) = TEMPA/PA(J)
C      BMCM(J) = BTEMPA/PA(J)
50  CONTINUE
C
C      STAGE II    CALCULATE EXPOSURE RATES FOR INGESTION OF ANIMAL
C                  PRODUCTS.
C
C      STEP 1      DETERMINE TOTAL REGIONAL CONSUMPTION OF EACH PRODUCT
C                  AND THE RATIO OF REGIONAL CONSUMPTION TO REGIONAL
C                  PRODUCTION.
C
C      DO 70 J = 1,NAP
C      TUA(J) = 0.
C      DO 60 K = 1,NPG
60  TUA(J) = TUA(J)+TP(K)*UA(K,J)
70  LA(J) = TUA(J)/PA(J)
C
C      STEP 2      CALCULATE BACKGROUND AND PLANT POLLUTANT EXPOSURE
C                  RATES BY POPULATION GROUP FOR CONSUMPTION OF EACH
C                  PRODUCT. IF REGIONAL CONSUMPTION EXCEEDS PRODUCTION,
C                  INCLUDE THE CONTRIBUTION OF ANIMAL PRODUCTS SUPPLIED
C                  FROM OUTSIDE THE REGION. SUM OVER PRODUCTS TO
C                  DETERMINE TOTAL EXPOSURE RATES BY POPULATION GROUP
C                  AND SUBREGION.
C
C      DO 90 K = 1,NPR
C      DO 90 L = 1,NPG
C      IGA(I,L,K) = 0.
C      BIGA(I,L,K) = 0.
C      DO 80 J = 1,NAP
C      IF (LA(J) .LE. 1.0) BSIGA(L,J) = UA(L,J)*BMCM(J)
C      IF (LA(J) .GT. 1.0) BSIGA(L,J) = (UA(L,J)/LA(J))*(BMCM(J)
1  -ACB(J))+ACB(J)*UA(L,J)
C      IF (LA(J) .LE. 1.0) SIGA(L,J) = UA(L,J)*MCM(J)
C      IF (LA(J) .GT. 1.0) SIGA(L,J) = (UA(L,J)/LA(J))*(MCM(J)-

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1          ACB(J))+ACB(J)*UA(L,J)
  BIGA(I,L,K) = BIGA(I,L,K)+BSIGA(L,J)
  IGA(I,L,K) = IGA(I,L,K)+SIGA(L,J)
80 CONTINUE
90 CONTINUE
  RETURN
  END

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SUBROUTINE CALDER(I)

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C
C  TITLE (CALCULATE DERMAL EXPOSURE)
C
C  SUBROUTINE CALDER DETERMINES EXPOSURE RATES FROM DERMAL CONTACT
C  WITH THE POLLUTANT.
C
C  PARAMETER      DESCRIPTION
C  I              INDEX SPECIFYING CURRENT POLLUTANT SPECIES (INTEGER)
C
C  COMMON         DESCRIPTION
C  C              POLLUTANT CONCENTRATIONS BY SUBREGION AND MEDIUM FOR
C                  CURRENT POLLUTANT SPECIES (2-DIM REAL ARRAY)
C  BC             BACKGROUND POLLUTANT CONCENTRATIONS BY SUBREGION AND
C                  MEDIUM FOR CURRENT SPECIES (2-DIM REAL ARRAY)
C  TDA            DURATION OF DERMAL EXPOSURE TO ATMOSPHERE (1-DIM
C                  REAL ARRAY)
C  TDD            DURATION OF DERMAL EXPOSURE TO DOMESTIC WATER
C                  SUPPLIES (1-DIM REAL ARRAY)
C  TDR            DURATION OF DERMAL EXPOSURE TO RECREATIONAL WATERS
C                  (1-DIM REAL ARRAY)
C  EDA            FRACTION OF BODY SURFACE EXPOSED TO THE ATMOSPHERE
C  EDAD           FRACTION OF BODY SURFACE EXPOSED TO DOMESTIC WATER
C  EDAR           FRACTION OF BODY SURFACE EXPOSED TO RECREATIONAL
C                  WATERS
C  CR             POLLUTANT CONCENTRATIONS IN RECREATIONAL WATERS
C                  (1-DIM REAL ARRAY)
C  BCR            BACKGROUND POLLUTANT CONCENTRATIONS IN RECREATIONAL
C                  WATERS (1-DIM REAL ARRAY)
C  NRL            NUMBER OF RECREATIONAL SITES (INTEGER)
C  RUF            FRACTIONAL USE OF RECREATIONAL LOCATIONS (1-DIM REAL
C                  ARRAY)
C  TF             FRACTION OF POLLUTANT SPECIES REMAINING AFTER
C                  DRINKING WATER TREATMENT (1-DIM REAL ARRAY)
C  VF            FRACTION OF DOMESTIC WATER CONSUMED BY SUBREGION THAT
C                  COMES FROM SURFACE WATER SOURCES (1-DIM REAL ARRAY)
C  IWT            DRINKING WATER TREATMENT PROCESSES USED WITHIN THE
C                  SUBREGION FOR SURFACE AND GROUNDWATER SOURCES (2-DIM
C                  INTEGER ARRAY)
C  DA             DERMAL EXPOSURE RATE FROM ATMOSPHERIC POLLUTION
C                  (3-DIM REAL ARRAY)
C  BDA            DERMAL EXPOSURE RATE FROM BACKGROUND POLLUTION IN
C                  ATMOSPHERE (3-DIM REAL ARRAY)
C  DWD            DERMAL EXPOSURE RATE FROM DOMESTIC WATER SUPPLIES

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C      (3-DIM REAL ARRAY)
C      BDWD      DERMAL EXPOSURE RATE FROM BACKGROUND POLLUTION IN
C                  DOMESTIC WATER (3-DIM REAL ARRAY)
C      DWR       DERMAL EXPOSURE RATE FROM RECREATIONAL WATER USE
C                  (3-DIM REAL ARRAY)
C      BDWR      DERMAL EXPOSURE RATE FROM BACKGROUND POLLUTION IN
C                  RECREATIONAL WATER (3-DIM REAL ARRAY)
C      NPG       NUMBER OF POPULATION GROUPS (INTEGER)
C      NPR       NUMBER OF POPULATION SUBREGIONS (INTEGER)
C
C      VARIABLE   DESCRIPTION
C      IS         INDEX SPECIFYING CURRENT TREATMENT PROCESS FOR
C                  SURFACE WATER (INTEGER)
C      IG         INDEX SPECIFYING THE CURRENT TREATMENT PROCESS FOR
C                  GROUNDWATER (INTEGER)
C      BCRM       SUMMATION VARIABLE TO CALCULATE MEAN BACKGROUND
C                  POLLUTANT LEVEL IN RECREATIONAL WATER
C      CRM        SUMMATION VARIABLE TO CALCULATE MEAN POLLUTANT LEVEL
C                  IN RECREATIONAL WATER
C
C      CALLED BY   MAIN PROGRAM
C
C      REAL C(5,100),BC(5,100),TDA(4),TDD(4),TF(10),BDA(5,4,100),
1      DA(5,4,100),BDWD(5,4,100),DWD(5,4,100),VF(100),BCR(5),
2      CR(5),RUF(5),BDWR(5,4,100),DWR(5,4,100),TDR(4)
C      INTEGER IWT(2,100)
C      COMMON /CON/C,BC
C      COMMON /DR/TDA,TDD,TDR,EDA,EDAD,EDAR,GS,CR,BCR,RUF,NRL
C      COMMON /DW/TF,VF,IWT,NTP
C      COMMON /EXDR/DA,BDA,DWD,BDWD,DWR,BDWR
C      COMMON /N/NPG,NPR,PL
C
C      STEP 1      USE ATMOSPHERIC POLLUTANT CONCENTRATIONS AND
C                  EXPOSURE DATA TO CALCULATE EXPOSURE FROM DERMAL
C                  CONTACT WITH PARTICULATES AND GASES.
C
C      DO 10 J = 1,NPG
C      DO 10 K = 1,NPR
C      BDA(I,J,K) = (BC(1,K)+BC(2,K))*EDA*TDA(J)/8760.0
10  DA(I,J,K) = (C(1,K)+C(2,K))*EDA*TDA(J)/8760.0
C
C      STEP 2      DETERMINE DERMAL EXPOSURE LEVELS FROM CONTACT WITH
C                  DOMESTIC WATER SUPPLIES, PARTITIONING THE SUPPLIES
C                  BETWEEN SURFACE AND GROUNDWATER SOURCES AND
C                  INCORPORATING THE EFFECTS OF WATER TREATMENT.
C
C      DO 20 J = 1,NPG
C      DO 20 K = 1,NPR
C      IS = IWT(1,K)
C      IG = IWT(2,K)
C      BDWD(I,J,K) = TDD(J)*EDAD*(BC(4,K)*TF(IS)*VF(K)+BC(5,K)*TF(IG)*
1      (1.0-VF(K)))/8.760

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20 DWD(I,J,K) = TDD(J)*EDAD*(C(4,K)*TF(IS)*VF(K)+C(5,K)*TF(IG)*
1      (1.0-VF(K)))/8.760
C
C      STEP 3      INITIALIZE SUMMATION VARIABLES AND CALCULATE THE
C                  WEIGHTED MEAN POLLUTANT CONCENTRATIONS OVER ALL
C                  RECREATIONAL WATER USE LOCATIONS.
C
      BCRM = 0.
      CRM = 0.
      DO 30 J = 1,NRL
      BCRM = BCRM+BCR(J)*RUF(J)
30 CRM = CRM+CR(J)*RUF(J)
C
C      STEP 4      DETERMINE DERMAL EXPOSURE RATES USING MEAN POLLUTANT
C                  CONCENTRATIONS CALCULATED IN STEP 3 AND EXPOSURE
C                  INFORMATION.
C
      DO 40 J = 1,NPG
      DO 40 K = 1,NPR
      BDWR(I,J,K) = TDR(J)*BCRM*EDAR/8.760
40 DWR(I,J,K) = TDR(J)*CRM*EDAR/8.760
      RETURN
      END

      SUBROUTINE EXSUMC(I)
C
C      TITLE (EXPOSURE SUMMATION AND CONVERSION)
C
C      SUBROUTINE EXSUMC SUMS EXPOSURE RATES BY INTAKE MODES, CONVERTS
C      THESE SUMS TO PROPER FORM FOR HEALTH CALCULATIONS, AND DETERMINES
C      MEAN EXPOSURE RATES BY POPULATION SUBREGION.
C
      PARAMETER      DESCRIPTION
C      I              INDEX SPECIFYING CURRENT POLLUTANT SPECIES (INTEGER)
C
C      COMMON         DESCRIPTION
C      IGA            TOTAL INGESTION EXPOSURE RATE FOR ANIMAL PRODUCTS
C                    (3-DIM REAL ARRAY)
C      BIGA           TOTAL BACKGROUND INGESTION EXPOSURE RATE FOR ANIMAL
C                    PRODUCTS (3-DIM REAL ARRAY)
C      IGQ            TOTAL INGESTION EXPOSURE RATE FOR AQUATIC ORGANISMS
C                    (3-DIM REAL ARRAY)
C      BIGQ           TOTAL BACKGROUND INGESTION EXPOSURE RATE FOR AQUATIC
C                    ORGANISMS (3-DIM REAL ARRAY)
C      DA             DERMAL EXPOSURE RATE FROM ATMOSPHERIC POLLUTION
C                    (3-DIM REAL ARRAY)
C      BDA            DERMAL EXPOSURE RATE FROM BACKGROUND POLLUTION IN
C                    ATMOSPHERE (3-DIM REAL ARRAY)
C      DWD            DERMAL EXPOSURE RATE FROM DOMESTIC WATER SUPPLIES
C                    (3-DIM REAL ARRAY)
C      BDWD           DERMAL EXPOSURE RATE FROM BACKGROUND POLLUTION IN
C                    DOMESTIC WATER (3-DIM REAL ARRAY)

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C	DWR	DERMAL EXPOSURE RATE FROM RECREATIONAL WATER USE
C		(3-DIM REAL ARRAY)
C	BDWR	DERMAL EXPOSURE RATE FROM BACKGROUND POLLUTION IN
C		RECREATIONAL WATER (3-DIM REAL ARRAY)
C	BIG	BACKGROUND INGESTION EXPOSURE FROM FOOD TYPES NOT
C		CONSIDERED IN ANALYSIS (1-DIM REAL ARRAY)
C	IGP	INGESTION EXPOSURE RATE FOR ATMOSPHERIC PARTICULATES
C		(3-DIM REAL ARRAY)
C	BIGP	BACKGROUND INGESTION EXPOSURE RATE FOR ATMOSPHERIC
C		PARTICULATES (3-DIM REAL ARRAY)
C	IGW	INGESTION EXPOSURE RATE FOR DRINKING WATER (3-DIM
C		REAL ARRAY)
C	BIGW	BACKGROUND INGESTION EXPOSURE RATE FOR DRINKING
C		WATER (3-DIM REAL ARRAY)
C	IH	INHALATION EXPOSURE RATE (2-DIM REAL ARRAY)
C	BIH	BACKGROUND INHALATION EXPOSURE RATE (2-DIM REAL
C		ARRAY)
C	IGV	TOTAL INGESTION EXPOSURE RATE FOR VEGETABLE PRODUCTS
C		(3-DIM REAL ARRAY)
C	BIGV	TOTAL BACKGROUND INGESTION EXPOSURE RATE FOR
C		VEGETABLE PRODUCTS (3-DIM REAL ARRAY)
C	IHW	MEAN INHALATION EXPOSURE RATE BY SUBREGION (2-DIM
C		REAL ARRAY)
C	BIHW	MEAN BACKGROUND INHALATION EXPOSURE RATE BY SUBREGION
C		(2-DIM REAL ARRAY)
C	IGT	TOTAL INGESTION EXPOSURE RATE (3-DIM REAL ARRAY)
C	BIGT	TOTAL BACKGROUND INGESTION EXPOSURE RATE (3-DIM REAL
C		ARRAY)
C	IGTW	MEAN INGESTION EXPOSURE RATE BY SUBREGION (2-DIM
C		REAL ARRAY)
C	BIGTW	MEAN BACKGROUND INGESTION EXPOSURE RATE BY SUBREGION
C		(2-DIM REAL ARRAY)
C	DT	TOTAL DERMAL EXPOSURE RATE (3-DIM REAL ARRAY)
C	BDT	TOTAL BACKGROUND DERMAL EXPOSURE RATE (3-DIM REAL
C		ARRAY)
C	IHWC	CONVERTED MEAN INHALATION EXPOSURE RATE BY SUBREGION
C		(2-DIM REAL ARRAY)
C	BIHWC	CONVERTED MEAN BACKGROUND INHALATION EXPOSURE RATE
C		BY SUBREGION (2-DIM REAL ARRAY)
C	IGTWC	CONVERTED MEAN INGESTION EXPOSURE RATE BY SUBREGION
C		(2-DIM REAL ARRAY)
C	BIGTWC	CONVERTED MEAN BACKGROUND INGESTION EXPOSURE RATE BY
C		SUBREGION (2-DIM REAL ARRAY)
C	DTW	MEAN DERMAL EXPOSURE RATE BY SUBREGION (2-DIM REAL
C		ARRAY)
C	BDTW	MEAN BACKGROUND DERMAL EXPOSURE RATE BY SUBREGION
C		(2-DIM REAL ARRAY)
C	DTWC	CONVERTED MEAN DERMAL EXPOSURE RATE BY SUBREGION
C		(2-DIM REAL ARRAY)
C	BDTWC	CONVERTED MEAN BACKGROUND DERMAL EXPOSURE RATE BY
C		SUBREGION (2-DIM REAL ARRAY)
C	TU	TOTAL FOOD CONSUMPTION RATES BY POPULATION GROUP

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C          (1-DIM REAL ARRAY)
C      UW      DRINKING WATER CONSUMPTION RATES BY POPULATION GROUP
C          (1-DIM REAL ARRAY)
C      FS      SPECIFIC INHALATION RATE COEFFICIENT
C      HS      SPECIFIC INGESTION RATE COEFFICIENT
C      RSH     INHALATION EXPOSURE ABSORPTION FRACTION
C      RSG     INGESTION EXPOSURE ABSORPTION FRACTION
C      GS      COEFFICIENT OF DERMAL MASS TRANSFER PER UNIT AREA
C      NPG     NUMBER OF POPULATION GROUPS (INTEGER)
C      NPR     NUMBER OF POPULATION SUBREGIONS (INTEGER)
C      P       POPULATION BY SUBREGION AND GROUP (2-DIM REAL ARRAY)
C      TPR     TOTAL POPULATION BY SUBREGION (1-DIM REAL ARRAY)
C      QINHLE  LOGICAL INHALATION EXPOSURE INDICATOR--.TRUE. IF
C              INHALATION EXPOSURE IS TO BE CALCULATED
C      QINGST  LOGICAL INGESTION EXPOSURE INDICATOR
C      QDERML  LOGICAL DERMAL EXPOSURE INDICATOR
C
C      VARIABLE  DESCRIPTION
C      TEMP      SUMMATION VARIABLE TO CALCULATE MEAN EXPOSURE RATES
C      BTEMP     SUMMATION VARIABLE TO CALCULATE MEAN BACKGROUND
C              EXPOSURE RATES
C      TEMPC     SUMMATION VARIABLE TO CALCULATE CONVERTED MEAN
C              EXPOSURE RATES
C      BTEMPC    SUMMATION VARIABLE TO CALCULATE CONVERTED MEAN
C              BACKGROUND EXPOSURE RATES
C
C      CALLED BY  MAIN PROGRAM
C
C      REAL IH(5,4,100),BIH(5,4,100),P(4,100),IHW(5,100),BIHW(5,100),
1      IHC(5,100),BIHC(5,100),IGT(5,4,100),UW(4),DTWC(5,100),
2      BIGT(5,4,100),BIGV(5,4,100),BIGW(5,4,100),BIGQ(5,4,100),
3      BIGP(5,4,100),TPR(100),BIGA(5,4,100),BIG(4),IGTWC(5,100),
4      BIGTWC(5,100),BIGTW(5,100),TU(4),IGTW(5,100),BDTWC(5,100),
5      IGA(5,4,100),IGV(5,4,100),DT(5,4,100),BDT(5,4,100),
6      BDA(5,4,100),DA(5,4,100),BDWD(5,4,100),DWD(5,4,100),
7      BDWR(5,4,100),IGW(5,4,100),IGQ(5,4,100),BDTW(5,100),
8      DTW(5,100),DWR(5,4,100),IGP(5,4,100)
C      REAL TDA(4),TDD(4),TDR(4),CR(5),RUF(5),BCR(5)
C      LOGICAL QINIT,QINHLE,QINGST,QDERML,QGRW,QSURFW
C      COMMON /DR/TDA,TDD,TDR,EDA,EDAD,EDAR,GS,CR,BCR,RUF,NRL
C      COMMON /EXAN/IGA,BIGA
C      COMMON /EXAQ/IGQ,BIGQ
C      COMMON /EXDR/DA,BDA,DWD,BDWD,DWR,BDWR
C      COMMON /EXIGB/BIG,IGP,BIGP,IGW,BIGW
C      COMMON /EXIH/IH,BIH
C      COMMON /EXVG/IGV,BIGV
C      COMMON /EXW/IHW,BIHW,IGT,BIGT,IGTW,BIGTW,DT,BDT,DTW,BDTW
C      COMMON /EXWC/IHC,BIHC,IGTWC,BIGTWC,DTWC,BDTWC
C      COMMON /IGD/UI(4),W(4),TU,UW,PIG,BMF
C      COMMON /L/HLIFE,PLIFE,FS,HS,RSG,RSH
C      COMMON /N/NPG,NPR,PL
C      COMMON /POP/POPGRP(2,4),P,TPR,TPS,TP(4)

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COMMON /Q/QINIT,QINHLE,QINGST,QDERML,QGRW,QSURFW
C
C STEP 1      PROCESS EXPOSURE RATES BY POPULATION SUBREGION.  IF
C              INHALATION EXPOSURE HAS BEEN CONSIDERED, SET
C              SUMMATION VARIABLES TO ZERO.  OTHERWISE, BRANCH TO
C              STEP 4.
C
DO 60 K = 1,NPR
IF (.NOT. QINHLE) GO TO 20
TEMP = 0.
BTEMP = 0.
TEMPC = 0.
BTEMPC = 0.
C
C STEP 2      SUM EXPOSURE AND CONVERTED EXPOSURE VALUES OVER
C              POPULATION GROUPS USING SUBREGIONAL POPULATIONS AS
C              WEIGHTS.
C
DO 10 J = 1,NPG
TEMP = TEMP+IH(I,J,K)*P(J,K)
BTEMP = BTEMP+BIH(I,J,K)*P(J,K)
TEMPC = TEMPC+IH(I,J,K)*P(J,K)*FS*RSR
BTEMPC = BTEMPC+BIH(I,J,K)*P(J,K)*FS*RSR
10 CONTINUE
C
C STEP 3      CALCULATE WEIGHTED MEAN AND CONVERTED INHALATION
C              EXPOSURE RATES BY SUBREGION.
C
BIHW(I,K) = BTEMP/TPR(K)
IHW(I,K) = TEMP/TPR(K)
BIHWC(I,K) = BTEMPC/TPR(K)
IHC(I,K) = TEMPC/TPR(K)
C
C STEP 4      SET TEMPORARY INGESTION EXPOSURE SUMMATION VARIABLES
C              TO ZERO IF INGESTION HAS BEEN CONSIDERED.  OTHERWISE,
C              BRANCH TO STEP 7.
C
20 IF (.NOT. QINGST) GO TO 40
TEMP = 0.
BTEMP = 0.
TEMPC = 0.
BTEMPC = 0.
C
C STEP 5      SUM BACKGROUND AND PLANT POLLUTANT EXPOSURE RATES FOR
C              ALL TYPES OF INGESTION EXPOSURE.  SUM WEIGHTED AND
C              CONVERTED EXPOSURE RATES OVER POPULATION GROUPS USING
C              SUBREGIONAL POPULATIONS AS WEIGHTS.
C
DO 30 J = 1,NPG
BIGT(I,J,K) = BIGW(I,J,K)+BIGQ(I,J,K)+BIGP(I,J,K)
1  +BIGV(I,J,K)+BIGA(I,J,K)+BIG(J)
BTEMP = BTEMP+BIGT(I,J,K)*P(J,K)

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      BTEMPC = BTEMPC+(BIGT(I,J,K)*HS*RSG/(TU(J)+UW(J)))*P(J,K)
      IGT(I,J,K) = IGW(I,J,K)+IGQ(I,J,K)+IGP(I,J,K)+IGV(I,J,K)+
1      IGA(I,J,K)+BIG(J)
      TEMP = TEMP+IGT(I,J,K)*P(J,K)
      TEMPC = TEMPC+(IGT(I,J,K)*HS*RSG/(TU(J)+UW(J)))*P(J,K)
30 CONTINUE

C
C      STEP 6          CALCULATE WEIGHTED MEAN AND CONVERTED INGESTION
C                      EXPOSURE RATES BY SUBREGION.
C
      BIGTW(I,K) = BTEMP/TPR(K)
      IGTW(I,K) = TEMP/TPR(K)
      BIGTWC(I,K) = BTEMPC/TPR(K)
      IGTWC(I,K) = TEMPC/TPR(K)

C
C      STEP 7          IF DERMAL EXPOSURE HAS BEEN CONSIDERED, SET
C                      SUMMATION VARIABLES TO ZERO. OTHERWISE, BRANCH TO
C                      END OF LOOP FOR PROCESSING BY SUBREGION.
C
40 IF (.NOT. QDERML) GO TO 60
      TEMP = 0.
      BTEMP = 0.

C
C      STEP 8          CALCULATE TOTAL DERMAL EXPOSURE RATES BY SUMMING OVER
C                      THE SEPARATE PATHWAYS. SUM WEIGHTED EXPOSURE RATES
C                      OVER POPULATION GROUPS USING SUBREGIONAL POPULATIONS
C                      AS WEIGHTS.
C
      DO 50 J = 1,NPG
      BDT(I,J,K) = BDA(I,J,K)+BDWD(I,J,K)+BDWR(I,J,K)
      DT(I,J,K) = DA(I,J,K)+DWD(I,J,K)+DWR(I,J,K)
      TEMP = TEMP+DT(I,J,K)*P(J,K)
      BTEMP = BTEMP+BDT(I,J,K)*P(J,K)
50 CONTINUE

C
C      STEP 9          CALCULATE WEIGHTED MEAN DERMAL EXPOSURE RATES BY
C                      SUBREGION. CONTINUE PROCESSING DO LOOP OVER
C                      SUBREGIONS.
C
      BDTW(I,K) = BTEMP/TPR(K)
      DTW(I,K) = TEMP/TPR(K)
      BDTWC(I,K) = BDTW(I,K)*GS
      DTWC(I,K) = DTW(I,K)*GS
60 CONTINUE
      RETURN
      END

      SUBROUTINE WREXP(I,NAO,NAP,NVS)

C
C      TITLE (WRITE EXPOSURE RATES)
C
C      SUBROUTINE WREXP WRITES EXPOSURE RATES BY SUBREGION, PATHWAY, AND

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3	POPULATION GROUP.	
3		
3	PARAMETER	DESCRIPTION
3	I	INDEX SPECIFYING CURRENT POLLUTANT SPECIES (INTEGER)
3	NAO	NUMBER OF AQUATIC ORGANISMS (INTEGER)
3	NAP	NUMBER OF ANIMAL PRODUCTS (INTEGER)
3	NVS	NUMBER OF VEGETABLE PRODUCTS (INTEGER)
3		
3	COMMON	DESCRIPTION
3	IGA	TOTAL INGESTION EXPOSURE RATE FOR ANIMAL PRODUCTS (3-DIM REAL ARRAY)
3	BIGA	TOTAL BACKGROUND INGESTION EXPOSURE RATE FOR ANIMAL PRODUCTS (3-DIM REAL ARRAY)
3	IGQ	TOTAL INGESTION EXPOSURE RATE FOR AQUATIC ORGANISMS (3-DIM REAL ARRAY)
3	BIGQ	TOTAL BACKGROUND INGESTION EXPOSURE RATE FOR AQUATIC ORGANISMS (3-DIM REAL ARRAY)
3	DA	DERMAL EXPOSURE RATE FROM ATMOSPHERIC POLLUTION (3-DIM REAL ARRAY)
3	BDA	DERMAL EXPOSURE RATE FROM BACKGROUND POLLUTION IN ATMOSPHERE (3-DIM REAL ARRAY)
3	DWD	DERMAL EXPOSURE RATE FROM DOMESTIC WATER SUPPLIES (3-DIM REAL ARRAY)
3	BDWD	DERMAL EXPOSURE RATE FROM BACKGROUND POLLUTION IN DOMESTIC WATER (3-DIM REAL ARRAY)
3	DWR	DERMAL EXPOSURE RATE FROM RECREATIONAL WATER USE (3-DIM REAL ARRAY)
3	BDWR	DERMAL EXPOSURE RATE FROM BACKGROUND POLLUTION IN RECREATIONAL WATER (3-DIM REAL ARRAY)
3	BIG	BACKGROUND INGESTION EXPOSURE FROM FOOD TYPES NOT CONSIDERED IN ANALYSIS (1-DIM REAL ARRAY)
3	IGP	INGESTION EXPOSURE RATE FOR ATMOSPHERIC PARTICULATES (3-DIM REAL ARRAY)
3	BIGP	BACKGROUND INGESTION EXPOSURE RATE FOR ATMOSPHERIC PARTICULATES (3-DIM REAL ARRAY)
3	IGW	INGESTION EXPOSURE RATE FOR DRINKING WATER (3-DIM REAL ARRAY)
3	BIGW	BACKGROUND INGESTION EXPOSURE RATE FOR DRINKING WATER (3-DIM REAL ARRAY)
3	IH	INHALATION EXPOSURE RATE (2-DIM REAL ARRAY)
3	BIH	BACKGROUND INHALATION EXPOSURE RATE (2-DIM REAL ARRAY)
3	IGV	TOTAL INGESTION EXPOSURE RATE FOR VEGETABLE PRODUCTS (3-DIM REAL ARRAY)
3	BIGV	TOTAL BACKGROUND INGESTION EXPOSURE RATE FOR VEGETABLE PRODUCTS (3-DIM REAL ARRAY)
3	IHW	MEAN INHALATION EXPOSURE RATE BY SUBREGION (2-DIM REAL ARRAY)
3	BIHW	MEAN BACKGROUND INHALATION EXPOSURE RATE BY SUBREGION (2-DIM REAL ARRAY)
3	IGT	TOTAL INGESTION EXPOSURE RATE (3-DIM REAL ARRAY)
3	BIGT	TOTAL BACKGROUND INGESTION EXPOSURE RATE (3-DIM REAL

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C          ARRAY)
C      IGTW      MEAN INGESTION EXPOSURE RATE BY SUBREGION (2-DIM
C                REAL ARRAY)
C      BIGTW     MEAN BACKGROUND INGESTION EXPOSURE RATE BY SUBREGION
C                (2-DIM REAL ARRAY)
C      DT        TOTAL DERMAL EXPOSURE RATE (3-DIM REAL ARRAY)
C      BDT       TOTAL BACKGROUND DERMAL EXPOSURE RATE (3-DIM REAL
C                ARRAY)
C      DTW       MEAN DERMAL EXPOSURE RATE BY SUBREGION (2-DIM REAL
C                ARRAY)
C      BDTW      MEAN BACKGROUND DERMAL EXPOSURE RATE BY SUBREGION
C                (2-DIM REAL ARRAY)
C      NPG       NUMBER OF POPULATION GROUPS (INTEGER)
C      NPR       NUMBER OF POPULATION SUBREGIONS (INTEGER)
C      POPGRP    NAME OF POPULATION GROUPS (2-DIM REAL ARRAY)
C      P         POPULATION BY SUBREGION AND GROUP (2-DIM REAL ARRAY)
C      TPR       TOTAL POPULATION BY SUBREGION (1-DIM REAL ARRAY)
C      QINHLE    LOGICAL INHALATION EXPOSURE INDICATOR--.TRUE. IF
C                INHALATION EXPOSURE IS TO BE CALCULATED
C      QINGST    LOGICAL INGESTION EXPOSURE INDICATOR
C      QDERML    LOGICAL DERMAL EXPOSURE INDICATOR
C
C      CALLED BY  MAIN PROGRAM
C
C      REAL IH(5,4,100),IHW(5,100),BIH(5,4,100),BIHW(5,100),POPGRP(2,4),
1      IGQ(5,4,100),BIGQ(5,4,100),IGA(5,4,100),BIGA(5,4,100),
2      IGV(5,4,100),BIGV(5,4,100),IGW(5,4,100),BIGW(5,4,100),
3      IGP(5,4,100),BIGP(5,4,100),IGT(5,4,100),BIGT(5,4,100),
4      IGTW(5,100),BIGTW(5,100),DA(5,4,100),BDA(5,4,100),
5      DWR(5,4,100),BDWR(5,4,100),DWD(5,4,100),BDWD(5,4,100),
6      DT(5,4,100),BDT(5,4,100),DTW(5,100),BDTW(5,100),BIG(4)
      LOGICAL QINIT,QINHLE,QINGST,QDERML,QGRW,QSURFW
      COMMON /EXAN/IGA,BIGA
      COMMON /EXAQ/IGQ,BIGQ
      COMMON /EXDR/DA,BDA,DWD,BDWD,DWR,BDWR
      COMMON /EXIGB/BIG,IGP,BIGP,IGW,BIGW
      COMMON /EXIH/IH,BIH
      COMMON /EXVG/IGV,BIGV
      COMMON /EXW/IHW,BIHW,IGT,BIGT,IGTW,BIGTW,DT,BDT,DTW,BDTW
      COMMON /N/NPG,NPR,PL
      COMMON /POP/POPGRP,P(4,100),TPR(100),TPS,TP(4)
      COMMON /Q/QINIT,QINHLE,QINGST,QDERML,QGRW,QSURFW
C
C      STEP 1      WRITE GENERAL TITLE FOR EXPOSURE OUTPUT.
C
C      WRITE (6,6001)
C
C      STEP 2      IF INHALATION EXPOSURE IS NOT INCLUDED, BRANCH TO
C                  STEP 4. OTHERWISE, WRITE THE TITLE FOR INHALATION
C                  EXPOSURE OUTPUT. IF THERE ARE FEWER THAN 50
C                  SUBREGIONS, WRITE ALL INHALATION EXPOSURE RATES BY
C                  SUBREGION AND BRANCH TO STEP 4. IF THERE ARE MORE

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C          THAN 50 SUBREGIONS, BRANCH TO STEP 3.
C
C      IF (.NOT. QINHLE) GO TO 50
C      WRITE (6,6002)
C      IF (NPR .GT. 50) GO TO 20
C      DO 10 J = 1,NPR
10 WRITE (6,6003) J,IH(I,1,J),IHW(I,J),BIH(I,1,J),BIHW(I,J)
C      GO TO 50
C
C      STEP 3          WRITE ALL INHALATION EXPOSURE RATES BY SUBREGION IN
C                      GROUPS OF 50.
C
C      20 DO 30 J = 1,50
C      30 WRITE (6,6003) J,IH(I,1,J),IHW(I,J),BIH(I,1,J),BIHW(I,J)
C      WRITE (6,6004)
C      WRITE (6,6002)
C      DO 40 J = 51,NPR
C      40 WRITE (6,6003) J,IH(I,1,J),IHW(I,J),BIH(I,1,J),BIHW(I,J)
C
C      STEP 4          IF INGESTION EXPOSURE IS NOT INCLUDED, BRANCH TO
C                      STEP 17. OTHERWISE, BEGIN A NEW PAGE AND WRITE THE
C                      GENERAL TITLE FOR INGESTION EXPOSURE OUTPUT.
C
C      50 IF (.NOT. QINGST) GO TO 350
C      IF (QINHLE) WRITE (6,6004)
C      WRITE (6,6005)
C
C      STEP 5          IF NO AQUATIC ORGANISMS WERE CONSIDERED, BRANCH TO
C                      STEP 7. OTHERWISE, WRITE THE TITLE FOR AQUATIC
C                      ORGANISM INGESTION EXPOSURE RATES. IF FEWER THAN
C                      50 SUBREGIONS, WRITE EXPOSURE RATES BY SUBREGION
C                      AND PROCEED TO STEP 7. IF MORE THAN 50 SUBREGIONS,
C                      BRANCH TO STEP 6.
C
C      IF (NAO .EQ. 0) GO TO 100
C      WRITE (6,6006) ((POPGRP(K,J),K=1,2),J=1,4),
C      1 ((POPGRP(K,J),K=1,2),J=1,4)
C      IF (NPR .GT. 50) GO TO 70
C      DO 60 J = 1,NPR
C      60 WRITE (6,6007) J,(IGQ(I,K,J),K=1,4),(BIGQ(I,K,J),K=1,4)
C      GO TO 100
C
C      STEP 6          WRITE AQUATIC ORGANISM INGESTION EXPOSURE RATES BY
C                      SUBREGION IN GROUPS OF 50.
C
C      70 DO 80 J = 1,50
C      80 WRITE (6,6007) J,(IGQ(I,K,J),K=1,4),(BIGQ(I,K,J),K=1,4)
C      WRITE (6,6004)
C      WRITE (6,6006) ((POPGRP(K,J),K=1,2),J=1,4),
C      1 ((POPGRP(K,J),K=1,2),J=1,4)
C      DO 90 J = 51,NPR
C      90 WRITE (6,6007) J,(IGQ(I,K,J),K=1,4),(BIGQ(I,K,J),K=1,4)

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C
C     STEP 7           IF NO ANIMAL PRODUCTS ARE INCLUDED, BRANCH TO STEP
C                      9. OTHERWISE, WRITE THE TITLE FOR EXPOSURE FROM
C                      INGESTION OF ANIMAL PRODUCTS. IF FEWER THAN 50
C                      SUBREGIONS, WRITE ANIMAL PRODUCT INGESTION EXPOSURE
C                      RATES BY SUBREGION AND BRANCH TO STEP 9. IF MORE
C                      THAN 50 SUBREGIONS, PROCEED TO STEP 8.
C
100 IF (NAP .EQ. 0) GO TO 150
    IF (NAO .NE. 0) WRITE (6,6004)
    WRITE (6,6008) ((POPGRP(K,J),K=1,2),J=1,4),
1      ((POPGRP(K,J),K=1,2),J=1,4)
    IF (NPR .GT. 50) GO TO 120
    DO 110 J = 1,NPR
110 WRITE (6,6007) J,(IGA(I,K,J),K=1,4),(BIGA(I,K,J),K=1,4)
    GO TO 150
C
C     STEP 8           WRITE ANIMAL PRODUCT INGESTION EXPOSURE RATES BY
C                      SUBREGION IN GROUPS OF 50.
C
120 DO 130 J = 1,50
130 WRITE (6,6007) J,(IGA(I,K,J),K=1,4),(BIGA(I,K,J),K=1,4)
    WRITE (6,6004)
    WRITE (6,6008) ((POPGRP(K,J),K=1,2),J=1,4),
1      ((POPGRP(K,J),K=1,2),J=1,4)
    DO 140 J = 51,NPR
140 WRITE (6,6007) J,(IGA(I,K,J),K=1,4),(BIGA(I,K,J),K=1,4)
C
C     STEP 9           IF NO VEGETABLE PRODUCTS WERE CONSIDERED, BRANCH TO
C                      STEP 11. OTHERWISE, WRITE THE TITLE FOR EXPOSURE
C                      FROM INGESTION OF VEGETABLE PRODUCTS. IF FEWER THAN
C                      50 SUBREGIONS, WRITE VEGETABLE PRODUCT INGESTION
C                      EXPOSURE RATES BY SUBREGION AND BRANCH TO STEP 11.
C                      IF MORE THAN 50 SUBREGIONS, PROCEED TO STEP 10.
C
150 IF (NVS .EQ. 0) GO TO 200
    IF ((NAO .NE. 0) .OR. (NAP .NE. 0)) WRITE (6,6004)
    WRITE (6,6009) ((POPGRP(K,J),K=1,2),J=1,4),
1      ((POPGRP(K,J),K=1,2),J=1,4)
    IF (NPR .GT. 50) GO TO 170
    DO 160 J = 1,NPR
160 WRITE (6,6007) J,(IGV(I,K,J),K=1,4),(BIGV(I,K,J),K=1,4)
    GO TO 200
C
C     STEP 10          WRITE VEGETABLE PRODUCT INGESTION EXPOSURE RATES BY
C                      SUBREGION IN GROUPS OF 50.
C
170 DO 180 J = 1,50
180 WRITE (6,6007) J,(IGV(I,K,J),K=1,4),(BIGV(I,K,J),K=1,4)
    WRITE (6,6004)
    WRITE (6,6009) ((POPGRP(K,J),K=1,2),J=1,4),
1      ((POPGRP(K,J),K=1,2),J=1,4)

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DO 190 J = 51,NPR
190 WRITE (6,6007) J,(IGV(I,K,J),K=1,4),(BIGV(I,K,J),K=1,4)
C
C   STEP 11      WRITE THE TITLE FOR EXPOSURE FROM CONSUMPTION OF
C                 DRINKING WATER.  IF FEWER THAN 50 SUBREGIONS, WRITE
C                 DRINKING WATER CONSUMPTION EXPOSURE RATES BY
C                 SUBREGION AND PROCEED TO STEP 13.  IF MORE THAN 50
C                 SUBREGIONS, BRANCH TO STEP 12.
C
200 IF ((NAO .NE. 0) .OR. (NAP .NE. 0) .OR. (NVS .NE. 0))
1   WRITE (6,6004)
   WRITE (6,6010) ((POPGRP(K,J),K=1,2),J=1,4),
1   ((POPGRP(K,J),K=1,2),J=1,4)
   IF (NPR .GT. 50) GO TO 220
   DO 210 J = 1,NPR
210 WRITE (6,6007) J,(IGW(I,K,J),K=1,4),(BIGW(I,K,J),K=1,4)
   GO TO 250
C
C   STEP 12      WRITE DRINKING WATER CONSUMPTION EXPOSURE RATES BY
C                 SUBREGION IN GROUPS OF 50.
C
220 DO 230 J = 1,50
230 WRITE (6,6007) J,(IGW(I,K,J),K=1,4),(BIGW(I,K,J),K=1,4)
   WRITE (6,6004)
   WRITE (6,6010) ((POPGRP(K,J),K=1,2),J=1,4),
1   ((POPGRP(K,J),K=1,2),J=1,4)
   DO 240 J = 51,NPR
240 WRITE (6,6007) J,(IGW(I,K,J),K=1,4),(BIGW(I,K,J),K=1,4)
C
C   STEP 13      WRITE THE TITLE FOR EXPOSURE FROM INGESTION OF
C                 ATMOSPHERIC PARTICULATES.  IF FEWER THAN 50
C                 SUBREGIONS, WRITE ATMOSPHERIC PARTICULATE INGESTION
C                 EXPOSURE RATES BY SUBREGION AND PROCEED TO STEP 15.
C
250 WRITE (6,6011) ((POPGRP(K,J),K=1,2),J=1,4),
1   ((POPGRP(K,J),K=1,2),J=1,4)
   IF (NPR .GT. 50) GO TO 270
   DO 260 J = 1,NPR
260 WRITE (6,6007) J,(IGP(I,K,J),K=1,4),(BIGP(I,K,J),K=1,4)
   GO TO 300
C
C   STEP 14      WRITE ATMOSPHERIC PARTICULATE INGESTION EXPOSURE
C                 RATES BY SUBREGION IN GROUPS OF 50.
C
270 DO 280 J = 1,50
280 WRITE (6,6007) J,(IGP(I,K,J),K=1,4),(BIGP(I,K,J),K=1,4)
   WRITE (6,6011) ((POPGRP(K,J),K=1,2),J=1,4),
1   ((POPGRP(K,J),K=1,2),J=1,4)
   DO 290 J = 51,NPR
290 WRITE (6,6007) J,(IGP(I,K,J),K=1,4),(BIGP(I,K,J),K=1,4)
C
C   STEP 15      WRITE THE HEADING FOR THE SUMMARY OF INGESTION

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C          EXPOSURE RATES.  IF FEWER THAN 50 SUBREGIONS, WRITE
C          TOTAL INGESTION EXPOSURE RATES BY SUBREGION AND
C          BRANCH TO STEP 17.  IF MORE THAN 50 SUBREGIONS,
C          PROCEED TO STEP 16.
C
300 WRITE (6,6012) ((POPGRP(K,J),K=1,2),J=1,4),
1      ((POPGRP(K,J),K=1,2),J=1,4)
      IF (NPR .GT. 50) GO TO 320
      DO 310 J = 1,NPR
310 WRITE (6,6013) J,(IGT(I,K,J),K=1,4),IGTW(I,J),(BIGT(I,K,J),
1      K=1,4),BIGTW(I,J)
      GO TO 350
C
C      STEP 16      WRITE TOTAL INGESTION EXPOSURE RATES BY SUBREGION
C                   IN GROUPS OF 50.
C
320 DO 330 J = 1,50
330 WRITE (6,6013) J,(IGT(I,K,J),K=1,4),IGTW(I,J),(BIGT(I,K,J),
1      K=1,4),BIGTW(I,J)
      WRITE (6,6012) ((POPGRP(K,J),K=1,2),J=1,4),
1      ((POPGRP(K,J),K=1,2),J=1,4)
      DO 340 J = 51,NPR
340 WRITE (6,6013) J,(IGT(I,K,J),K=1,4),IGTW(I,J),(BIGT(I,K,J),
1      K=1,4),BIGTW(I,J)
C
C      STEP 17      IF DERMAL CONTACT EXPOSURE IS NOT CONSIDERED, RETURN.
C                   OTHERWISE, WRITE THE GENERAL TITLE FOR DERMAL CONTACT
C                   EXPOSURE RATES.
C
350 IF (.NOT. QDERML) RETURN
      IF (QINHLE .OR. QINGST) WRITE (6,6004)
      WRITE (6,6014)
C
C      STEP 18      WRITE THE TITLE FOR EXPOSURE THROUGH DERMAL CONTACT
C                   WITH ATMOSPHERIC GASES AND PARTICULATES.  IF FEWER
C                   THAN 50 SUBREGIONS, WRITE ATMOSPHERIC DERMAL CONTACT
C                   EXPOSURE RATES BY SUBREGION AND BRANCH TO STEP 20.
C                   IF MORE THAN 50 SUBREGIONS, PROCEED TO STEP 19.
C
      WRITE (6,6015) ((POPGRP(K,J),K=1,2),J=1,4),
1      ((POPGRP(K,J),K=1,2),J=1,4)
      IF (NPR .GT. 50) GO TO 370
      DO 360 J = 1,NPR
360 WRITE (6,6007) J,(DA(I,K,J),K=1,4),(BDA(I,K,J),K=1,4)
      GO TO 400
C
C      STEP 19      WRITE ATMOSPHERIC DERMAL CONTACT EXPOSURE RATES BY
C                   SUBREGION IN GROUPS OF 50.
C
370 DO 380 J = 1,50
380 WRITE (6,6007) J,(DA(I,K,J),K=1,4),(BDA(I,K,J),K=1,4)
      WRITE (6,6004)

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        WRITE (6,6015) ((POPGRP(K,J),K=1,2),J=1,4),
1          ((POPGRP(K,J),K=1,2),J=1,4)
        DO 390 J = 51,NPR
390 WRITE (6,6007) J,(DA(I,K,J),K=1,4),(BDA(I,K,J),K=1,4)
C
C      STEP 20      WRITE THE TITLE FOR EXPOSURE THROUGH DERMAL CONTACT
C                   WITH RECREATIONAL WATER. IF FEWER THAN 50
C                   SUBREGIONS, WRITE RECREATIONAL WATER DERMAL CONTACT
C                   EXPOSURE RATES BY SUBREGION AND BRANCH TO STEP 22.
C                   IF MORE THAN 50 SUBREGIONS, PROCEED TO STEP 21.
C
400 WRITE (6,6016) ((POPGRP(K,J),K=1,2),J=1,4),
1          ((POPGRP(K,J),K=1,2),J=1,4)
        IF (NPR .GT. 50) GO TO 420
        DO 410 J = 1,NPR
410 WRITE (6,6007) J,(DWR(I,K,J),K=1,4),(BDWR(I,K,J),K=1,4)
        GO TO 450
C
C      STEP 21      WRITE RECREATIONAL WATER DERMAL CONTACT EXPOSURE
C                   RATES BY SUBREGION IN GROUPS OF 50.
C
420 DO 430 J = 1,50
430 WRITE (6,6007) J,(DWR(I,K,J),K=1,4),(BDWR(I,K,J),K=1,4)
        WRITE (6,6016) ((POPGRP(K,J),K=1,2),J=1,4),
1          ((POPGRP(K,J),K=1,2),J=1,4)
        DO 440 J = 51,NPR
440 WRITE (6,6007) J,(DWR(I,K,J),K=1,4),(BDWR(I,K,J),K=1,4)
C
C      STEP 22      WRITE TITLE FOR EXPOSURE THROUGH DERMAL CONTACT WITH
C                   DOMESTIC WATER. IF FEWER THAN 50 SUBREGIONS, WRITE
C                   DOMESTIC WATER DERMAL EXPOSURE RATES BY SUBREGION
C                   AND PROCEED TO STEP 24. IF MORE THAN 50 SUBREGIONS,
C                   BRANCH TO STEP 23.
C
450 WRITE (6,6017) ((POPGRP(K,J),K=1,2),J=1,4),
1          ((POPGRP(K,J),K=1,2),J=1,4)
        IF (NPR .GT. 50) GO TO 470
        DO 460 J = 1,NPR
460 WRITE (6,6007) J,(DWD(I,K,J),K=1,4),(BDWD(I,K,J),K=1,4)
        GO TO 500
C
C      STEP 23      WRITE DOMESTIC WATER DERMAL EXPOSURE RATES BY
C                   SUBREGION IN GROUPS OF 50.
C
470 DO 480 J = 1,50
480 WRITE (6,6007) J,(DWD(I,K,J),K=1,4),(BDWD(I,K,J),K=1,4)
        WRITE (6,6017) ((POPGRP(K,J),K=1,2),J=1,4),
1          ((POPGRP(K,J),K=1,2),J=1,4)
        DO 490 J = 51,NPR
490 WRITE (6,6007) J,(DWD(I,K,J),K=1,4),(BDWD(I,K,J),K=1,4)
C
C      STEP 24      WRITE TITLE FOR SUMMARY OF DERMAL CONTACT EXPOSURE

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C          RATES.  IF FEWER THAN 50 SUBREGIONS, WRITE TOTAL
C          DERMAL CONTACT EXPOSURE RATES BY SUBREGION AND
C          RETURN.  IF MORE THAN 50 SUBREGIONS, BRANCH TO
C          STEP 25.
C
500 WRITE (6,6018) ((POPGRP(K,J),K=1,2),J=1,4),
1      ((POPGRP(K,J),K=1,2),J=1,4)
      IF (NPR .GT. 50) GO TO 520
      DO 510 J = 1,NPR
510 WRITE (6,6013) J,(DT(I,K,J),K=1,4),DTW(I,J),(BDT(I,K,J),K=1,4),
1      BDTW(I,J)
      RETURN
C
C          STEP 25      WRITE TOTAL DERMAL CONTACT EXPOSURE RATES BY
C          SUBREGION IN GROUPS OF 50.
C
520 DO 530 J = 1,50
530 WRITE (6,6013) J,(DT(I,K,J),K=1,4),DTW(I,J),(BDT(I,K,J),K=1,4),
1      BDTW(I,J)
      WRITE (6,6018) ((POPGRP(K,J),K=1,2),J=1,4),
1      ((POPGRP(K,J),K=1,2),J=1,4)
      DO 540 J = 51,NPR
540 WRITE (6,6013) J,(DT(I,K,J),K=1,4),DTW(I,J),(BDT(I,K,J),K=1,4),
1      BDTW(I,J)
      RETURN
C
C          FORMAT STATEMENTS
C
6001 FORMAT (1H1,54X,25HSUMMARY OF EXPOSURE RATES,/)
6002 FORMAT (1H0,39X,45HEXPOSURE THROUGH INHALATION OF GASES AND PART,
1      8HICULATES,/,69X,13HEXPOSURE RATE,/,72X,7H(MG/M3),/,
2      19X,9HSUBREGION,15X,5HPLANT,9X,10HMEAN PLANT,10X,
3      10HBACKGROUND,10X,5HMEAN ,10HBACKGROUND)
6003 FORMAT (22X,I3,13X,1PE13.5,4X,E13.5,7X,1PE13.5,9X,1PE13.5)
6004 FORMAT (1H1)
6005 FORMAT (1H0,48X,35HEXPOSURE THROUGH INGESTION PATHWAYS,/)
6006 FORMAT (1H0,/,43X,44HEXPOSURE THROUGH INGESTION OF AQUATIC ORGANI,
1      3HSMS,/,64X,13HEXPOSURE RATE,/,44X,5HPLANT,17X,
2      8H(MG/DAY),22X,10HBACKGROUND,/,6X,9HSUBREGION,5X,4(2X,
3      2A4,2X),12X,4(2X,2A4,2X))
6007 FORMAT (9X,I3,7X,4(1PE12.5),12X,4(1PE12.5))
6008 FORMAT (1H0,/,43X,40HEXPOSURE THROUGH INGESTION OF ANIMAL PRO,
1      5HDUCTS,/,64X,13HEXPOSURE RATE,/,44X,5HPLANT,17X,
2      8H(MG/DAY),22X,10HBACKGROUND,/,6X,9HSUBREGION,5X,4(2X,
3      2A4,2X),12X,4(2X,2A4,2X))
6009 FORMAT (1H0,/,42X,43HEXPOSURE THROUGH INGESTION OF VEGETABLE PRO,
1      5HDUCTS,/,64X,13HEXPOSURE RATE,/,44X,5HPLANT,17X,
2      8H(MG/DAY),22X,10HBACKGROUND,/,6X,9HSUBREGION,5X,4(2X,
3      2A4,2X),12X,4(2X,2A4,2X))
6010 FORMAT (1H0,/,44X,44HEXPOSURE THROUGH INGESTION OF DRINKING WATER,
1      /,64X,13HEXPOSURE RATE,/,44X,5HPLANT,17X,8H(MG/DAY),
2      22X,10HBACKGROUND,/,6X,9HSUBREGION,5X,4(2X,2A4,2X),12X,

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      3      4(2X,2A4,2X))
6011 FORMAT (1H1,/,45X,42HEXPOSURE THROUGH INGESTION OF PARTICULATES,
      1      //,64X,13HEXPOSURE RATE,/,44X,5HPLANT,17X,8H(MG/DAY),
      2      22X,10HBACKGROUND,/,6X,9HSUBREGION,5X,4(2X,2A4,2X),
      3      12X,4(2X,2A4,2X))
6012 FORMAT (1H1,/,44X,43HSUMMARY CALCULATIONS FOR INGESTION EXPOSURE,
      1      //,69X,13HEXPOSURE RATE,/,44X,5HPLANT,22X,8H(MG/DAY),17X,
      2      10HBACKGROUND,/,1X,9HSUBREGION,3X,4(2X,2A4,2X),3X,4HMEAN,
      3      5X,4(2X,2A4,2X),3X,4HMEAN)
6013 FORMAT (4X,I3,5X,10(1PE12.5))
6014 FORMAT (1H0,46X,40HEXPOSURE THROUGH DERMAL CONTACT PATHWAYS,/)
6015 FORMAT (1H0,/,30X,43HEXPOSURE THROUGH DERMAL CONTACT WITH ATMOSP,
      1      28HHERIC PARTICULATES AND GASES,/,64X,13HEXPOSURE RATE,/,
      2      44X,5HPLANT,18X,7H(MG/M3),22X,10HBACKGROUND,/,6X,
      3      9HSUBREGION,5X,4(2X,2A4,2X),12X,4(2X,2A4,2X))
6016 FORMAT (1H1,/,38X,43HEXPOSURE THROUGH DERMAL CONTACT WITH RECREA,
      1      12HTIONAL WATER,/,64X,13HEXPOSURE RATE,/,44X,5HPLANT,18X,
      2      7H(MG/M3),22X,10HBACKGROUND,/,6X,9HSUBREGION,5X,4(2X,2A4,
      3      2X),12X,4(2X,2A4,2X))
6017 FORMAT (1H1,/,40X,43HEXPOSURE THROUGH DERMAL CONTACT WITH DOMEST,
      1      8HIC WATER,/,64X,13HEXPOSURE RATE,/,44X,5HPLANT,18X,
      2      7H(MG/M3),22X,10HBACKGROUND,/,6X,9HSUBREGION,5X,4(2X,2A4,
      3      2X),12X,4(2X,2A4,2X))
6018 FORMAT (1H1,46X,40HSUMMARY OF DERMAL CONTACT EXPOSURE RATES,/,
      1      69X,13HEXPOSURE RATE,/,44X,5HPLANT,13X,7H(MG/M3),17X,
      2      10HBACKGROUND,/,1X,9HSUBREGION,3X,4(2X,2A4,2X),3X,4HMEAN,
      3      5X,4(2X,2A4,2X),3X,4HMEAN)
      END

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SUBROUTINE HEALTH(NHE)

TITLE (HEALTH EFFECTS CALCULATION)

SUBROUTINE HEALTH CALCULATES THE HEALTH EFFECTS ASSOCIATED WITH
EXPOSURE TO ALL POLLUTANT SPECIES.

PARAMETER	DESCRIPTION
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NHE	NUMBER OF CHRONIC HEALTH EFFECTS (INTEGER)
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COMMON	DESCRIPTION
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TMH	TOTAL INHALATION DOSE FROM COMBINED POLLUTANT SPECIES (1-DIM REAL ARRAY)
-----	---

BTMH	TOTAL INHALATION DOSE FROM BACKGROUND LEVELS OF COMBINED POLLUTANT SPECIES (1-DIM REAL ARRAY)
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TMG	TOTAL INGESTION DOSE FROM COMBINED POLLUTANT SPECIES (1-DIM REAL ARRAY)
-----	--

BTMG	TOTAL INGESTION DOSE FROM BACKGROUND LEVELS OF COMBINED POLLUTANT SPECIES (1-DIM REAL ARRAY)
------	---

TMD	TOTAL DERMAL CONTACT DOSE FROM COMBINED POLLUTANT SPECIES (1-DIM REAL ARRAY)
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BTMD	TOTAL DERMAL CONTACT DOSE FROM BACKGROUND LEVELS OF COMBINED POLLUTANT SPECIES (1-DIM REAL ARRAY)
------	--

C	AT	PARAMETERS FOR CURRENT VERSION OF THE MULTISTAGE
C		DOSE-RESPONSE MODEL (1-DIM REAL ARRAY)
C	IHWC	CONVERTED MEAN INHALATION EXPOSURE RATE BY SUBREGION
C		(2-DIM REAL ARRAY)
C	BIHWC	CONVERTED MEAN BACKGROUND INHALATION EXPOSURE RATE
C		BY SUBREGION (2-DIM REAL ARRAY)
C	IGTWC	CONVERTED MEAN INGESTION EXPOSURE RATE BY SUBREGION
C		(2-DIM REAL ARRAY)
C	BIGTWC	CONVERTED MEAN BACKGROUND INGESTION EXPOSURE RATE BY
C		SUBREGION (2-DIM REAL ARRAY)
C	DTWC	CONVERTED MEAN DERMAL EXPOSURE RATE BY SUBREGION
C		(2-DIM REAL ARRAY)
C	BDTWC	CONVERTED MEAN BACKGROUND DERMAL EXPOSURE RATE BY
C		SUBREGION (2-DIM REAL ARRAY)
C	EFFECT	NAME OF HEALTH EFFECTS (2-DIM REAL ARRAY)
C	KMODE	INTAKE MODE FOR HEALTH EFFECTS (2-DIM REAL ARRAY)
C	NSC	NUMBER OF POLLUTANT SPECIES CAUSING HEALTH EFFECTS
C		(1-DIM INTEGER ARRAY)
C	PSC	POLLUTANT SPECIES PRODUCING HEALTH EFFECTS (2-DIM
C		INTEGER ARRAY)
C	R	HEALTH RISK FOR POWER PLANT POLLUTANT LEVELS (3-DIM
C		REAL ARRAY)
C	BR	HEALTH RISK FOR BACKGROUND POLLUTANT LEVELS (2-DIM
C		REAL ARRAY)
C	BRW	HEALTH RISK FOR BACKGROUND POLLUTANT LEVELS USING
C		WEIBULL MODEL FOR PLANT LIFETIME EXPOSURE (1-DIM
C		REAL ARRAY)
C	RISK	NET HEALTH RISK FROM POWER PLANT (3-DIM REAL ARRAY)
C		
C	EXTERNALS	DESCRIPTION
C	COMCS	SUBROUTINE TO COMBINE EXPOSURE RATES FOR POLLUTANT
C		SPECIES
C	ONEHIT	SUBROUTINE TO CALCULATE RISK USING A ONE-HIT DOSE-
C		RESPONSE MODEL
C	MULTIH	SUBROUTINE TO CALCULATE RISK USING A MULTIHIT DOSE-
C		RESPONSE MODEL
C	MULTAD	SUBROUTINE TO CALCULATE RISK USING AN ARMITAGE/DOLL
C		MULTISTAGE DOSE-RESPONSE MODEL
C	MULTC	SUBROUTINE TO CALCULATE RISK USING A CRUMP DOSE-
C		RESPONSE MODEL
C	PROBIT	SUBROUTINE TO CALCULATE RISK USING A PROBIT DOSE-
C		RESPONSE MODEL
C	WEIB	SUBROUTINE TO CALCULATE RISK USING A WEIBULL DOSE-
C		RESPONSE MODEL
C		
C	VARIABLE	DESCRIPTION
C	IN	NUMBER OF POLLUTANT SPECIES RESPONSIBLE FOR CURRENT
C		HEALTH EFFECT (INTEGER)
C		RATHER THAN PLANT LIFE (INTEGER)
C	IPSC	LIST OF POLLUTANT SPECIES RESPONSIBLE FOR CURRENT
C		HEALTH EFFECT (1-DIM INTEGER ARRAY)
C	HEM	NUMBER REPRESENTING THE INTAKE MODE FOR EACH HEALTH


```

C          EFFECT (1-DIM INTEGER ARRAY)
C      IX      NUMBER OF DOSE-RESPONSE MODELS USED WITH CURRENT
C              HEALTH EFFECT (INTEGER)
C      DR      NAME OF DOSE-RESPONSE MODELS USED WITH CURRENT
C              HEALTH EFFECT (2-DIM REAL ARRAY)
C      DRM     REFERENCE LIST OF DOSE-RESPONSE MODEL NAMES (2-DIM
C              REAL ARRAY)
C      IND     INDICATOR FOR CURRENT DOSE-RESPONSE MODEL (INTEGER)
C      AO      PARAMETERS FOR ONE-HIT DOSE-RESPONSE MODELS
C              (1-DIM REAL ARRAY)
C      AM      PARAMETERS FOR MULTIHIT DOSE-RESPONSE MODELS
C              (1-DIM REAL ARRAY)
C      ISTG    NUMBER OF STAGES IN CURRENT MULTISTAGE DOSE-RESPONSE
C              MODEL (INTEGER)
C      IL      NUMBER OF PARAMETERS TO BE READ FOR CURRENT MULTI-
C              STAGE DOSE-RESPONSE MODEL (INTEGER)
C      AAD     PARAMETERS FOR ARMITAGE/DOLL MULTISTAGE DOSE-RESPONSE
C              MODELS (2-DIM REAL ARRAY)
C      JSTG    NUMBER OF STAGES IN CURRENT CRUMP DOSE-RESPONSE MODEL
C              (INTEGER)
C      JL      NUMBER OF PARAMETERS TO BE READ FOR CURRENT CRUMP
C              DOSE-RESPONSE MODEL (INTEGER)
C      AAC     PARAMETERS FOR CRUMP MULTISTAGE DOSE-RESPONSE MODELS
C              (2-DIM REAL ARRAY)
C      AP      PARAMETERS FOR PROBIT DOSE-RESPONSE MODELS (2-DIM
C              REAL ARRAY)
C      AW      PARAMETERS FOR WEIBULL DOSE-RESPONSE MODELS (2-DIM
C              REAL ARRAY)
C
C      CALLED BY  MAIN PROGRAM
C
      REAL TMH(100),BTMH(100),TMG(100),BTMG(100),TMD(100),BTMD(100),
1      KMODE(2,10),EFFECT(2,10),JMODE(2,3),IHWC(5,100),BIHWC(5,100),
2      IGTWC(5,100),BIGTWC(5,100),DTWC(5,100),BDTWC(5,100),DRM(2,6),
3      DR(2,10,5),AO(10),R(10,6,2),BR(10,6),RISK(10,6,2),AM(10,2),
4      AAD(10,10),AT(10),AAC(10,10),AP(10,2),AW(10,3),BRW(10)
      INTEGER NDR(10),NSC(10),PSC(10,5),HEM(10),IPSC(5)
      COMMON /B/TMH,BTMH,TMG,BTMG,TMD,BTMD
      COMMON /D/AT
      COMMON /EXWC/IHWC,BIHWC,IGTWC,BIGTWC,DTWC,BDTWC
      COMMON /HLEF/EFFECT,KMODE,NSC,PSC
      COMMON /RISK/R,BR,BRW,RISK
      DATA DRM(1,1),DRM(2,1),DRM(1,2),DRM(2,2),DRM(1,3),DRM(2,3),
1      DRM(1,4),DRM(2,4),DRM(1,5),DRM(2,5),DRM(1,6),DRM(2,6)/4HONEH,
2      4HIT ,4HMULT,4HINIT,4HMULT,4HISTA,4HCRUM,4HP ,4HPROB,
3      4HIT ,4HWEIB,4HULL /
      DATA JMODE(1,1),JMODE(2,1),JMODE(1,2),JMODE(2,2),JMODE(1,3),
1      JMODE(2,3)/4HINHA,4HLE ,4HINGE,4HST ,4HDERM,4HAL /
C
C      STEP 1      INITIALIZE RISK VARIABLES.
C
      DO 10 IJ = 1,10

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BRW(IJ) = 0.
DO 10 J = 1,6
BR(IJ,J) = 0.
DO 10 K = 1,2
R(IJ,J,K) = 0.
10 RISK(IJ,J,K) = 0.
C
C STEP 2 READ THE NUMBER OF HEALTH EFFECTS TO BE CONSIDERED.
C BEGIN A LOOP THROUGH THESE HEALTH EFFECTS.
C
READ (5,5001) NHE
DO 220 I = 1,NHE
C
C STEP 3 INITIALIZE VARIABLES SPECIFYING TOTAL EXPOSURE DOSE
C BY INTAKE MODE AND SUBREGION.
C
DO 20 IJ = 1,100
TMH(IJ) = 0.
BTMH(IJ) = 0.
TMG(IJ) = 0.
BTMG(IJ) = 0.
TMD(IJ) = 0.
20 BTMD(IJ) = 0.
C
C STEP 4 READ DESCRIPTION OF INTAKE MODE, EFFECT, RELEVANT
C POLLUTANT SPECIES, AND NUMBER OF DOSE-RESPONSE MODELS
C FOR THE CURRENT HEALTH EFFECT. SET CURRENT INDICATOR
C VALUES FOR MODE AND NUMBER OF CHEMICAL SPECIES.
C
READ (5,5002) (KMODE(K,I),K=1,2),(EFFECT(J,I),J=1,2),NDR(I),NSC(I)
DO 30 K = 1,3
IF (KMODE(1,I) .EQ. JMODE(1,K)) HEM(I) = K
30 CONTINUE
IN = NSC(I)
C
C STEP 5 READ THE LIST OF RELEVANT CHEMICAL SPECIES AND PLACE
C THE LIST IN A TEMPORARY ARRAY.
C
READ (5,5001) (PSC(I,J),J=1,IN)
DO 40 J = 1,IN
40 IPSC(J) = PSC(I,J)
C
C STEP 6 USING THE CURRENT VALUE OF THE INTAKE MODE INDICATOR
C AND LIST OF CHEMICAL SPECIES, COMBINE THE RELEVANT
C EXPOSURE RATES FOR INHALATION, INGESTION, OR DERMAL
C CONTACT.
C
IF (HEM(I) .GT. 1) GO TO 50
CALL COMCS(IHWC,IPSC,IN,TMH)
CALL COMCS(BIHWC,IPSC,IN,BTMH)
GO TO 70
50 IF (HEM(I) .GT. 2) GO TO 60

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      CALL COMCS(IGTWC,IPSC,IN,TMG)
      CALL COMCS(BIGTWC,IPSC,IN,BTMG)
      GO TO 70
60  CALL COMCS(DTWC,IPSC,IN,TMD)
      CALL COMCS(BDTWC,IPSC,IN,BTMD)
C
C
C
C
      STEP 7      SET THE INDEX FOR THE CURRENT NUMBER OF DOSE-RESPONSE
                   MODELS AND BEGIN DO LOOP TO CALCULATE RATE USING EACH
                   APPROPRIATE MODEL.
70  IX = NDR(I)
      DO 210 J = 1,IX
C
C
C
C
      STEP 8      READ THE DOSE-RESPONSE MODEL NAME AND SET INDICATOR
                   VALUE.  BRANCH ACCORDING TO THE INDICATOR.
                   BRANCH      INDICATOR      MODEL
                   STEP 9      1      ONE-HIT
                   STEP 10     2      MULTI HIT
                   STEP 11     3      MULTISTAGE
                   STEP 13     4      CRUMP
                   STEP 15     5      PROBIT
                   STEP 16     6      WEIBULL
C
C
C
C
      READ (5,5002) (DR(K,I,J),K=1,2)
      DO 80 K = 1,6
      IF ((DR(1,I,J).EQ.DRM(1,K)).AND.(DR(2,I,J).EQ.DRM(2,K)))  IND=K
80  CONTINUE
      GO TO (90,100,110,150,190,200),IND
C
C
C
C
      STEP 9      FOR THE ONE-HIT MODEL, READ THE PARAMETER VALUE,
                   CALL THE SUBROUTINE TO CALCULATE PLANT AND BACKGROUND
                   RISKS, AND DETERMINE THE NET RISK, USING MEAN LIFE-
                   TIME DOSE AND FULL LIFETIME EXPOSURE.  BRANCH TO
                   THE END OF THE DO LOOP FOR DOSE-RESPONSE MODELS.
90  READ (5,5003) AO(I)
      CALL ONEHIT(AO(I),HEM(I),R(I,1,1),R(I,1,2),BR(I,1))
      RISK(I,1,1) = R(I,1,1)-BR(I,1)
      RISK(I,1,2) = R(I,1,2)-BR(I,1)
      GO TO 210
C
C
C
C
      STEP 10     FOR THE MULTI HIT MODEL, READ TWO PARAMETER VALUES,
                   CALL THE SUBROUTINE TO CALCULATE PLANT AND BACK-
                   GROUND RISKS, AND DETERMINE THE NET RISK, USING MEAN
                   LIFETIME DOSE AND FULL LIFETIME EXPOSURE.  BRANCH
                   TO THE END OF THE DO LOOP FOR DOSE-RESPONSE MODELS.
100 READ (5,5003) (AM(I,K),K=1,2)
      CALL MULTI H(AM(I,1),AM(I,2),HEM(I),R(I,2,1),R(I,2,2),BR(I,2))
      RISK(I,2,1) = R(I,2,1)-BR(I,2)
      RISK(I,2,2) = R(I,2,2)-BR(I,2)
      GO TO 210

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C
C STEP 11      FOR THE MULTISTAGE MODEL, READ THE NUMBER OF STAGES
C              AND ADD ONE TO FIND THE NUMBER OF PARAMETERS.  READ
C              THE VALUE OF THESE PARAMETERS IN GROUPS OF 6 AND
C              PLACE THEM IN A TEMPORARY ARRAY.
C
110 READ (5,5001) ISTG
    IL = ISTG+1
    READ (5,5003) (AAD(I,K),K=1,IL)
    DO 140 K = 1,IL
140 AT(K) = AAD(I,K)
C
C STEP 12      CALL THE SUBROUTINE TO DETERMINE PLANT AND BACKGROUND
C              RISKS AND CALCULATE THE NET RISK, USING BOTH PLANT
C              LIFETIME AND HUMAN LIFETIME DOSE.  BRANCH TO THE END
C              OF THE LOOP FOR DOSE-RESPONSE MODELS.
C
    CALL MULTAD(ISTG,HEM(I),R(I,3,1),R(I,3,2),BR(I,3))
    RISK(I,3,1) = R(I,3,1)-BR(I,3)
    RISK(I,3,2) = R(I,3,2)-BR(I,3)
    GO TO 210
C
C STEP 13      FOR THE CRUMP MODEL, READ THE NUMBER OF STAGES AND
C              ADD ONE TO FIND THE NUMBER OF PARAMETERS.  READ THE
C              VALUES OF THESE PARAMETERS IN GROUPS OF 6 AND PLACE
C              THEM IN A TEMPORARY ARRAY.
C
150 READ (5,5001) JSTG
    JL = JSTG+1
    READ (5,5003) (AAC(I,K),K=1,JL)
    DO 180 K = 1,JL
180 AT(K) = AAC(I,K)
C
C STEP 14      CALL THE SUBROUTINE TO CALCULATE PLANT AND BACKGROUND
C              RISK VALUES AND DETERMINE NET RISK, USING BOTH PLANT
C              LIFETIME AND HUMAN LIFETIME EXPOSURE.  BRANCH TO THE
C              END OF THE DO LOOP FOR DOSE-RESPONSE MODELS.
C
    CALL MULTC(AT,JSTG,HEM(I),R(I,4,1),R(I,4,2),BR(I,4))
    RISK(I,4,1) = R(I,4,1)-BR(I,4)
    RISK(I,4,2) = R(I,4,2)-BR(I,4)
    GO TO 210
C
C STEP 15      FOR THE PROBIT MODEL, READ THE PARAMETER VALUES,
C              CALL THE SUBROUTINE TO DETERMINE PLANT AND BACKGROUND
C              RISKS AND CALCULATE NET RISK, USING BOTH PLANT
C              LIFETIME AND HUMAN LIFETIME EXPOSURE.  BRANCH TO THE
C              END OF THE LOOP FOR DOSE-RESPONSE MODELS.
C
190 READ (5,5003) (AP(I,K),K=1,2)
    CALL PROBIT(AP(I,1),AP(I,2),HEM(I),R(I,5,1),R(I,5,2),BR(I,5))
    RISK(I,5,1) = R(I,5,1)-BR(I,5)

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RISK(I,5,2) = R(I,5,2)-BR(I,5)
GO TO 210
C
C STEP 16 FOR THE WEIBULL MODEL, READ THE PARAMETER VALUES,
C CALL THE SUBROUTINE TO DETERMINE PLANT AND BACKGROUND
C RISK VALUES, AND CALCULATE NET RISK, USING BOTH
C PLANT LIFETIME AND HUMAN LIFETIME EXPOSURE. CONTINUE
C THE LOOPS ON DOSE-RESPONSE MODELS AND HEALTH EFFECTS.
C
200 READ (5,5003) (AW(I,K),K=1,3)
CALL WEIB(AW(I,1),AW(I,2),AW(I,3),HEM(I),R(I,6,1),R(I,6,2),
1 BRW(I),BR(I,6))
RISK(I,6,1) = R(I,6,1)-BRW(I)
RISK(I,6,2) = R(I,6,2)-BR(I,6)
210 CONTINUE
220 CONTINUE
RETURN
C
C FORMAT STATEMENTS
C
5001 FORMAT (16I5)
5002 FORMAT (2(2A4,2X),2I5)
5003 FORMAT (6(E12.5,1X))
END

SUBROUTINE WRHLTH(NHE)
C
C TITLE (WRITE HEALTH EFFECTS RESULTS)
C
C SUBROUTINE WRHLTH PRINTS THE RESULTS OF THE HEALTH EFFECTS
C CALCULATIONS.
C
C PARAMETER DESCRIPTION
C NHE NUMBER OF HEALTH EFFECTS (INTEGER)
C
C COMMON DESCRIPTION
C EFFECT NAME OF HEALTH EFFECTS (2-DIM REAL ARRAY)
C KMODE INTAKE MODE FOR HEALTH EFFECTS (2-DIM REAL ARRAY)
C NSC NUMBER OF POLLUTANT SPECIES CAUSING HEALTH EFFECTS
C (1-DIM INTEGER ARRAY)
C PSC POLLUTANT SPECIES PRODUCING HEALTH EFFECTS (2-DIM
C INTEGER ARRAY)
C POLLUT NAME OF POLLUTANT SPECIES (2-DIM REAL ARRAY)
C R HEALTH RISK FOR POWER PLANT POLLUTANT LEVELS (3-DIM
C REAL ARRAY)
C BR HEALTH RISK FOR BACKGROUND POLLUTANT LEVELS (2-DIM
C REAL ARRAY)
C BRW HEALTH RISK FOR BACKGROUND POLLUTANT LEVELS USING
C WEIBULL MODEL FOR PLANT LIFETIME EXPOSURE (1-DIM
C REAL ARRAY)
C RISK NET HEALTH RISK FROM POWER PLANT (3-DIM REAL ARRAY)
C

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C      VARIABLE      DESCRIPTION
C      IN            NUMBER OF POLLUTANT SPECIES RESPONSIBLE FOR CURRENT
C                   HEALTH EFFECT (INTEGER)
C      ISP           INDEX OF CURRENT POLLUTANT SPECIES (INTEGER)
C      CSPCS         NAME OF CURRENT POLLUTANT SPECIES (2-DIM REAL ARRAY)
C
C      CALLED BY     MAIN PROGRAM
C
C      REAL POLLUT(2,5),EFFECT(2,10),KMODE(2,10),CSPCS(2,5),BR(10,6),
1      R(10,6,2),RISK(10,6,2),BRW(10)
C      INTEGER NSC(10),PSC(10,5)
C      COMMON /HLEF/EFFECT,KMODE,NSC,PSC
C      COMMON /POL/POLLUT
C      COMMON /RISK/R,BR,BRW,RISK
C
C      STEP 1        WRITE GENERAL HEADING TO HEALTH EFFECTS OUTPUT.
C
C      WRITE (6,6001)
C      WRITE (6,6002)
C
C      STEP 2        FOR EACH HEALTH EFFECT, IDENTIFY THE POLLUTANT
C                   SPECIES AND PLACE THEIR NAMES IN A TEMPORARY ARRAY.
C                   PRINT A DESCRIPTION OF EACH HEALTH EFFECT.
C
C      DO 20 I = 1,NHE
C      IN = NSC(I)
C      DO 10 J = 1,IN
C      ISP = PSC(I,J)
C      DO 10 K = 1,2
10 CSPCS(K,J) = POLLUT(K,ISP)
20 WRITE (6,6003) (EFFECT(K,I),K=1,2),(KMODE(K,I),K=1,2),
1      ((CSPCS(K,J),K=1,2),J=1,IN)
C
C      STEP 3        WRITE THE HEADING FOR BACKGROUND RISK RESULTS.  FOR
C                   EACH HEALTH EFFECT, WRITE THE BACKGROUND RISK
C                   RESULTS BY DOSE-RESPONSE MODEL.
C
C      DO 60 IJ = 1,2
C      IF (IJ .EQ. 1) WRITE (6,6004)
C      IF (IJ .EQ. 2) WRITE (6,6010)
C      WRITE (6,6005)
C      DO 30 I = 1,NHE
C      IF (IJ .EQ. 1) WRITE (6,6006) (EFFECT(K,I),K=1,2),(BR(I,J),
1      J=1,5),BRW(I)
30 IF (IJ .EQ. 2) WRITE (6,6006) (EFFECT(K,I),K=1,2),(BR(I,J),J=1,6)
C
C      STEP 4        WRITE THE HEADING FOR TOTAL PLANT RISK RESULTS.  FOR
C                   PLANT AND HUMAN LIFETIME EXPOSURES, BY HEALTH EFFECT,
C                   WRITE THE PLANT RISK RESULTS BY DOSE-RESPONSE MODEL.
C
C      IF (IJ .EQ. 1) WRITE (6,6007)
C      IF (IJ .EQ. 2) WRITE (6,6009)

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C          ARRAY)
C
C  COMMON      DESCRIPTION
C  NPR         NUMBER OF POPULATION SUBREGIONS (INTEGER)
C  TPR         TOTAL POPULATION BY SUBREGION (1-DIM REAL ARRAY)
C
C  VARIABLE    DESCRIPTION
C  DTPR        TOTAL POPULATION BY SUBREGION (1-DIM DOUBLE PRECISION
C              ARRAY)
C
C  CALLED BY   ONEHIT - MULTIH - MULTAD - MULTC - PROBIT - WEIB
C
C              DOUBLE PRECISION X(100),DTPR(100)
C              COMMON /N/NPG,NPR,PL
C              COMMON /POP/POPGRP(2,4),P(4,100),TPR(100),TPS,TP(4)
C
C  STEP 1      CONVERT POPULATION DATA TO DOUBLE PRECISION.
C
C              DO 5 I = 1,100
C 5 DTPR(I) = TPR(I)
C
C  STEP 2      INITIALIZE RISK SUMMATION VARIABLE.  CALCULATE
C              RISK BY SUBREGION AND ADD TO TOTAL.
C
C              RC = 0.0D00
C              DO 10 I = 1,NPR
C              RC = RC+X(I)*DTPR(I)
C 10 CONTINUE
C              RETURN
C              END
C
C              DOUBLE PRECISION FUNCTION RGAMMA(A,X)
C
C              TITLE (GAMMA FUNCTION CALCULATION)
C
C              FUNCTION RGAMMA CALCULATES MULTIHIT DOSE-RESPONSE PROBABILITIES
C              USING A RECURRENCE FORMULA, POLYNOMIAL APPROXIMATION, AND SERIES
C              SUMMATION FOR THE INCOMPLETE GAMMA FUNCTION
C
C              PARAMETER      DESCRIPTION
C              A              NUMBER OF HITS IN THE MULTIHIT MODEL (DOUBLE
C                              PRECISION)
C              X              POLLUTANT DOSE (DOUBLE PRECISION)
C
C              VARIABLE      DESCRIPTION
C              SM             SERIES SUMMATION VARIABLE (DOUBLE PRECISION)
C              RI             DOUBLE PRECISION EQUIVALENT OF LOOP COUNTER (DOUBLE
C                              PRECISION)
C              D              SUMMATION DENOMINATOR (DOUBLE PRECISION)
C              TM             CURRENT SUMMATION TERM (DOUBLE PRECISION)
C              TS             RATIO OF CURRENT TERM TO CURRENT SUM IN SUMMATION
C                              LOOP (DOUBLE PRECISION)

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C      TP      CURRENT TERM VALUE IN RECURRENCE FORMULA (DOUBLE
C      PRECISION)
C      P      CURRENT PRODUCT VALUE IN RECURRENCE FORMULA (DOUBLE
C      PRECISION)
C      SMA     CURRENT SUM IN POLYNOMIAL APPROXIMATION (DOUBLE
C      PRECISION)
C      B      POLYNOMIAL APPROXIMATION COEFFICIENTS (1-DIM DOUBLE
C      PRECISION ARRAY)
C      FN     VALUE OF GAMMA FUNCTION FOR A (DOUBLE PRECISION)
C
C      EXTERNALS  DESCRIPTION
C      DABS     FORTRAN DOUBLE PRECISION ABSOLUTE VALUE ROUTINE
C      DEXP     FORTRAN DOUBLE PRECISION EXPONENTIAL ROUTINE
C
C      CALLED BY  MULTIH
C
C      DOUBLE PRECISION B(8),A,X,SM,RI,D,TM,TS,TP,P,SMA,FN
C      DATA B(1),B(2),B(3),B(4),B(5),B(6),B(7),B(8)/-0.577191652D00,
1      0.988205891D00,-0.897056937D00,0.918206857D00,-0.756704078D00,
2      0.482199394D00,-0.193527818D00,0.035868343D00/
C
C      STEP 1      INITIALIZE THE SUMMATION VARIABLE AND BEGIN SUMMATION
C                  LOOP FOR THE INCOMPLETE GAMMA FUNCTION.
C
C      SM = 0.0D00
C      DO 20 I = 1,100
C
C      STEP 2      USE LOOP INDEX VALUE TO DETERMINE THE VALUE OF THE
C                  CURRENT TERM DENOMINATOR.  CALCULATE THE CURRENT
C                  TERM VALUE.
C
C      RI = I-1
C      IF (I .EQ. 1) D = A
C      IF (I .GT. 1) D = D*(A+RI)
C      TM = (X**(A+RI))/D
C
C      STEP 3      AFTER THE INITIAL PASS THROUGH THE LOOP, TEST THE
C                  RELATIVE SIZE OF THE CURRENT TERM.  IF IT EXCEEDS
C                  THE LIMIT, ADD IT TO THE SUMMATION VARIABLE AND
C                  CONTINUE THE LOOP AT STEP 2.  IF IT DOES NOT EXCEED
C                  THE LIMIT, BRANCH TO STEP 4.
C
C      IF (I .EQ. 1) GO TO 10
C      TS = DABS(TM/SM)
C      IF (TS .LT. 1.0D-08) GO TO 25
10  SM = SM+TM
20  CONTINUE
C
C      STEP 4      SET INITIAL VALUES FOR THE RECURRENCE FORMULA TERM
C                  AND PRODUCT.  IF THE NUMBER OF HITS DOES NOT EXCEED
C                  ONE, BRANCH TO STEP 6.  OTHERWISE CONTINUE TO STEP 5.
C

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25 TP = A
   P = 1.0D00
   IF (A .LE. 1.0D00) GO TO 40
C
C   STEP 5      REDUCE THE CURRENT TERM BY ONE AND MULTIPLY BY THE
C               CURRENT PRODUCT. REPEAT THIS STEP UNTIL THE CURRENT
C               TERM VALUE DOES NOT EXCEED ONE. PROCEED TO STEP 6.
C
30 TP = TP-1.0D00
   P = TP*P
   IF (TP .GT. 1.0D00) GO TO 30
C
C   STEP 6      INITIALIZE THE SUMMATION VARIABLE. CALCULATE THE
C               COMPLETE GAMMA FUNCTION FOR THE CURRENT TERM VALUE
C               USING THE POLYNOMIAL APPROXIMATION.
C
40 SMA = 1.0D00
   DO 50 I = 1,8
     SMA = SMA+B(I)*(TP**I)
50 CONTINUE
C
C   STEP 7      COMBINE THE RESULTS OF PREVIOUS STEPS TO DETERMINE
C               THE GAMMA FUNCTION VALUE FOR A.
C
   FN = SMA*P/TP
C
C   STEP 8      COMBINE THE PREVIOUS RESULTS TO DETERMINE THE
C               MULTIHIT PROBABILITY VALUE.
C
   RGAMMA = SM/(FN*DEXP(X))
   RETURN
   END

DOUBLE PRECISION FUNCTION PROB(J,D,T)
C
C   TITLE (MULTISTAGE PROBABILITY)
C
C   DOUBLE PRECISION FUNCTION PROB CALCULATES THE MULTISTAGE DOSE-
C   RESPONSE MODEL PROBABILITY.
C
C   PARAMETER   DESCRIPTION
C   J           NUMBER OF STAGES IN MULTISTAGE MODEL (INTEGER)
C   D           POLLUTANT DOSE (DOUBLE PRECISION)
C   T           RELATIVE LENGTH OF EXPOSURE PERIOD
C
C   COMMON      DESCRIPTION
C   AL          MULTISTAGE MODEL PARAMETERS (1-DIM REAL ARRAY)
C
C   EXTERNALS   DESCRIPTION
C   FACT        FUNCTION TO CALCULATE FACTORIAL VALUES
C
C   VARIABLE    DESCRIPTION

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C      PT          VALUE OF CURRENT PRODUCT IN CALCULATION LOOP (DOUBLE
C                  PRECISION)
C      DAL         DOUBLE PRECISION VERSION OF MULTISTAGE MODEL
C                  PARAMETERS (1-DIM DOUBLE PRECISION ARRAY)
C      DT          DOUBLE PRECISION VERSION OF EXPOSURE PERIOD
C      JL          UPPER LIMIT FOR CALCULATION LOOP (INTEGER)
C
C      CALLED BY    MULTIH
C
C      DOUBLE PRECISION DAL(10),PT,DT,D
C      COMMON /D/AL(10)
C
C      STEP 1       TEST THE NUMBER OF HITS.  IF ZERO, SET THE
C                  PROBABILITY EQUAL TO ONE AND RETURN.  OTHERWISE,
C                  CONTINUE TO STEP 2.
C
C      IF (J .GT. 0)  GO TO 5
C      PROB = 1.0D00
C      RETURN
C
C      STEP 2       SET THE INITIAL VALUE OF THE CURRENT PRODUCT TERM.
C                  CONVERT PARAMETER VALUES AND EXPOSURE TIME TO DOUBLE
C                  PRECISION EQUIVALENTS.
C
C      5 PT = AL(1)
C      DO 8 I = 1,10
C      8 DAL(I) = AL(I)
C      DT = T
C
C      STEP 3       SET THE UPPER LIMIT OF THE CALCULATION LOOP TO ONE
C                  GREATER THAN THE NUMBER OF STAGES.  PASS THROUGH
C                  THE LOOP CALCULATING THE CURRENT PRODUCT FROM THE
C                  DOSE AND PARAMETER VALUES.
C
C      JL = J+1
C      DO 10 I = 2,JL
C      PT = PT*(1.0D00+DAL(I)*D)
C 10 CONTINUE
C
C      STEP 4       DETERMINE THE PROBABILITY FROM THE FINAL PRODUCT
C                  VALUE, THE EXPOSURE TIME, AND NUMBER OF STAGES.
C
C      PROB = (PT*DT**J)/FACT(J)
C      RETURN
C      END
C
C      DOUBLE PRECISION FUNCTION CUMND(X)
C
C      TITLE (CUMULATIVE NORMAL DISTRIBUTION)
C
C      DOUBLE PRECISION FUNCTION CUMND CALCULATES THE VALUE OF THE
C      CUMULATIVE NORMAL DISTRIBUTION FUNCTION FOR ITS ARGUMENT.

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C
C   PARAMETER   DESCRIPTION
C   X           ARGUMENT FOR CUMULATIVE NORMAL DISTRIBUTION (DOUBLE
C               PRECISION)
C
C   EXTERNALS   DESCRIPTION
C   DABS        FORTRAN DOUBLE PRECISION ABSOLUTE VALUE FUNCTION
C   FACT        FUNCTION TO CALCULATE FACTORIAL VALUES
C
C   VARIABLE    DESCRIPTION
C   SM          CURRENT SUMMATION VARIABLE IN SERIES APPROXIMATION
C               (DOUBLE PRECISION)
C   TS          RATIO OF CURRENT TERM TO CURRENT SUM IN SERIES
C               APPROXIMATION (DOUBLE PRECISION)
C   IND         INDICATOR VARIABLE FOR NEGATIVE ARGUMENT (INTEGER)
C   TP          CURRENT SUMMATION VARIABLE FOR POLYNOMIAL
C               APPROXIMATION (DOUBLE PRECISION)
C   N           DECREMENTED LOOP INDEX VARIABLE USED AS SERIES
C               APPROXIMATION EXPONENT (INTEGER)
C   TM          CURRENT TERM IN SERIES APPROXIMATION (DOUBLE
C               PRECISION)
C   C           COEFFICIENTS FOR POLYNOMIAL APPROXIMATION (1-DIM
C               DOUBLE PRECISION ARRAY)
C   P           ABSOLUTE VALUE OF FINAL CUMULATIVE PROBABILITY
C               (DOUBLE PRECISION)
C
C   CALLED BY   PROBIT
C
C   DOUBLE PRECISION SM, TM, TS, P, C(6), TP, X
C   DATA C(1), C(2), C(3), C(4), C(5), C(6) / 4.98673470D-02, 2.11410061D-02,
1   3.2776263D-03, 3.80036D-05, 4.88906D-05, 5.3830D-06 /
C
C   STEP 1      INITIALIZE VARIABLES FOR BOTH CALCULATION PROCEDURES.
C
C   SM = 0.0D00
C   TS = 0.0D00
C   IND = 0
C   TP = 0.0D00
C
C   STEP 2      DETERMINE THE SIGN OF THE ARGUMENT AND SET IT EQUAL
C               TO ITS ABSOLUTE VALUE. IF THE VALUE OF THE ARGUMENT
C               EXCEEDS 3.5, BRANCH TO STEP 6 TO USE THE POLYNOMIAL
C               APPROXIMATION METHOD. OTHERWISE, BEGIN THE LOOP FOR
C               THE SERIES APPROXIMATION.
C
C   IF (X .GE. 0.0D00) GO TO 5
C   IND = 1
C   X = -X
C   5 IF (X .GT. 3.5D00) GO TO 30
C   DO 20 I = 1, 100
C
C   STEP 3      SET THE EXPONENT VARIABLE TO ONE LESS THAN THE INDEX

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C          AND CALCULATE THE VALUE OF THE CURRENT TERM.
C
C      N = I-1
C      TM = (((-1.0D00)**N)*(X**(2*N+1)))/(FACT(N)*(2.0D00**N)*
1      (2.0D00*N+1))
C
C      STEP 4      AFTER THE INITIAL PASS THROUGH THE LOOP, TEST THE
C                  RELATIVE SIZE OF THE CURRENT TERM.  IF IT EXCEEDS
C                  THE LIMIT, ADD IT TO THE SUMMATION VARIABLE AND
C                  CONTINUE THE LOOP AT STEP 3.  IF IT DOES NOT EXCEED
C                  THE LIMIT, BRANCH TO STEP 5.
C
C      IF (I .EQ. 1)  GO TO 10
C      TS = DABS(TM/SM)
C      IF (TS .LT. 1.0D-10)  GO TO 25
10  SM = SM+TM
20  CONTINUE
C
C      STEP 5      CALCULATE THE VALUE OF THE DISTRIBUTION FUNCTION FROM
C                  THE SUMMATION AND BRANCH TO STEP 7.
C
25  P = 0.5D00+0.39894228040D00*SM
    GO TO 50
C
C      STEP 6      CALCULATE THE VALUE OF THE DISTRIBUTION USING THE
C                  ALTERNATIVE POLYNOMIAL APPROXIMATION FOR LARGE VALUES
C                  OF THE ARGUMENT.
C
30  DO 40 I = 1,6
40  TP = TP+C(I)*X**I
    P = 1.0D00-0.5D00*(1.0D00+TP)**(-16)
C
C      STEP 7      IF THE ORIGINAL ARGUMENT WAS NEGATIVE, SUBTRACT THE
C                  RESULT FROM ONE TO DETERMINE THE FINAL VALUE TO BE
C                  RETURNED.
C
50  IF (IND .EQ. 1)  P = 1.0D00-P
    CUMND = P
    RETURN
    END
C
C      DOUBLE PRECISION FUNCTION FACT(I)
C
C      TITLE (FACTORIAL)
C
C      DOUBLE PRECISION FUNCTION FACT CALCULATES THE FACTORIAL VALUE OF
C      ITS ARGUMENT.
C
C      PARAMETER      DESCRIPTION
C      I              ARGUMENT OF FACTORIAL FUNCTION
C
C      VARIABLE      DESCRIPTION

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C      F          CURRENT VALUE OF FACTORIAL (DOUBLE PRECISION)
C
C      CALLED BY   PROB - CUMND - MULTAD
C
C      DOUBLE PRECISION F,RJ
C
C      STEP 1      IF THE ARGUMENT IS ZERO, SET FACTORIAL TO ONE AND
C                  RETURN. OTHERWISE, PROCEED TO STEP 2.
C
C      IF (I .GT. 0) GO TO 5
C      FACT = 1.0D00
C      RETURN
C
C      STEP 2      SET INITIAL VALUE OF FACTORIAL TO ONE AND PASS
C                  THROUGH MULTIPLICATION LOOP. MULTIPLY BY THE LOOP
C                  VARIABLE AFTER CONVERSION TO DOUBLE PRECISION.
C
C      5 F = 1.0D00
C      DO 10 J = 1,I
C      RJ = J
C      F = F*RJ
C      10 CONTINUE
C
C      STEP 3      SET THE FACTORIAL VALUE EQUAL TO THE CURRENT PRODUCT.
C
C      FACT = F
C      RETURN
C      END
C
C      SUBROUTINE COMCS(EM,SC,NSC,CEM)
C
C      TITLE (COMBINE CHEMICAL SPECIES)
C
C      SUBROUTINE COMCS ADDS THE EXPOSURE RATES FOR SELECTED POLLUTANT
C      SPECIES.
C
C      PARAMETER    DESCRIPTION
C      EM           MEAN CONVERTED EXPOSURE RATES BY SUBREGION (2-DIM
C                  REAL ARRAY)
C      SC           LIST OF POLLUTANT SPECIES TO BE COMBINED (1-DIM
C                  INTEGER ARRAY)
C      NSC          NUMBER OF POLLUTANT SPECIES TO BE COMBINED (INTEGER)
C      CEM          EXPOSURE RATES FOR COMBINED POLLUTANT SPECIES (1-DIM
C                  REAL ARRAY)
C
C      COMMON       DESCRIPTION
C      NPR          NUMBER OF POPULATION SUBREGIONS
C
C      VARIABLE     DESCRIPTION
C      IJ           INDEX OF CURRENT POLLUTANT SPECIES
C
C      CALLED BY    HEALTH

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C
COMMON /N/NPG,NPR,PL
REAL EM(5,100),CEM(100)
INTEGER SC(5)

C
C STEP 1      INITIALIZE COMBINED EXPOSURE RATES BY SUBREGION.
C
DO 10 J = 1,NPR
CEM(J) = 0.

C
C STEP 2      IDENTIFY EACH POLLUTANT SPECIES AND ADD ITS EXPOSURE
C              RATE TO THE TOTAL FOR EACH SUBREGION.
C
DO 5 K = 1,NSC
IJ = SC(K)
5 CEM(J) = CEM(J)+EM(IJ,J)
10 CONTINUE
RETURN
END

SUBROUTINE DOSEC(IM,IND,D,BD)

C
C TITLE (DOSE CALCULATION)
C
C SUBROUTINE DOSEC CALCULATES AVERAGE LIFETIME POLLUTANT DOSES FROM
C EXPOSURE RATES.
C
C PARAMETER      DESCRIPTION
C IM             INDEX SPECIFYING EXPOSURE INTAKE MODE (INTEGER)
C IND            INDICATOR TO SPECIFY NO AVERAGING OF PLANT AND
C                BACKGROUND EXPOSURE RATES (INTEGER)
C D              CALCULATED DOSE RATES BY SUBREGION (1-DIM DOUBLE
C                PRECISION ARRAY)
C BD             CALCULATED BACKGROUND DOSE RATES BY SUBREGION (1-DIM
C                DOUBLE PRECISION ARRAY)
C
C COMMON         DESCRIPTION
C TMH            TOTAL INHALATION DOSE FROM COMBINED POLLUTANT
C                SPECIES (1-DIM REAL ARRAY)
C BTMH           TOTAL INHALATION DOSE FROM BACKGROUND LEVELS OF
C                COMBINED POLLUTANT SPECIES (1-DIM REAL ARRAY)
C TMG            TOTAL INGESTION DOSE FROM COMBINED POLLUTANT SPECIES
C                (1-DIM REAL ARRAY)
C BTMG           TOTAL INGESTION DOSE FROM BACKGROUND LEVELS OF
C                COMBINED POLLUTANT SPECIES (1-DIM REAL ARRAY)
C TMD            TOTAL DERMAL CONTACT DOSE FROM COMBINED POLLUTANT
C                SPECIES (1-DIM REAL ARRAY)
C BTMD           TOTAL DERMAL CONTACT DOSE FROM BACKGROUND LEVELS OF
C                COMBINED POLLUTANT SPECIES (1-DIM REAL ARRAY)
C NPR            NUMBER OF POPULATION SUBREGIONS (INTEGER)
C
C CALLED BY      ONEHIT - MULTIH - MULTAD - MULTC - PROBIT - WEIB

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C
COMMON /B/TMH(100),BTMH(100),TMG(100),BTMG(100),TMD(100),BTMD(100)
COMMON /N/NPG,NPR,PL
DOUBLE PRECISION D(100),BD(100)

C
C STEP 1      DETERMINE INTAKE MODE.  IF NOT INHALATION, BRANCH TO
C              STEP 2.  IF INHALATION, DETERMINE METHOD OF PLANT
C              LEVEL DOSE AVERAGING, CALCULATE PLANT LEVEL AND
C              BACKGROUND DOSES BY SUBREGION, AND RETURN.
C
      IF (IM .GT. 1) GO TO 20
      DO 10 J = 1,NPR
      IF (IND .EQ. 1) D(J) = TMH(J)
      IF (IND .EQ. 0) D(J) = TMH(J)*PL+BTMH(J)*(1.0-PL)
      BD(J) = BTMH(J)
10 CONTINUE
      RETURN

C
C STEP 2      DETERMINE INTAKE MODE.  IF NOT INGESTION, BRANCH TO
C              STEP 3.  IF INGESTION, DETERMINE METHOD OF PLANT
C              LEVEL DOSE AVERAGING, CALCULATE PLANT LEVEL AND
C              BACKGROUND DOSES BY SUBREGION, AND RETURN.
C
20 IF (IM .GT. 2) GO TO 40
      DO 30 J = 1,NPR
      IF (IND .EQ. 1) D(J) = TMG(J)
      IF (IND .EQ. 0) D(J) = TMG(J)*PL+BTMG(J)*(1.0-PL)
      BD(J) = BTMG(J)
30 CONTINUE
      RETURN

C
C STEP 3      DETERMINE METHOD OF PLANT LEVEL DOSE AVERAGING.
C              CALCULATE PLANT LEVEL AND BACKGROUND DOSES BY
C              SUBREGION AND RETURN.
C
40 DO 50 J = 1,NPR
      IF (IND .EQ. 1) D(J) = TMD(J)
      IF (IND .EQ. 0) D(J) = TMD(J)*PL+BTMD(J)*(1.0-PL)
      BD(J) = BTMD(J)
50 CONTINUE
      RETURN
      END

      SUBROUTINE ONEHIT(A,IM,PRSK,HRSK,BRSK)

C
C TITLE (ONE-HIT MODEL RISK)
C
C SUBROUTINE ONEHIT CALCULATES PLANT LEVEL AND BACKGROUND RISKS
C USING THE ONE-HIT DOSE-RESPONSE MODEL.
C
C PARAMETER DESCRIPTION
C A ONE-HIT MODEL PARAMETER

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C      IM          INDEX SPECIFYING EXPOSURE INTAKE MODE (INTEGER)
C      PRSK        PLANT POLLUTANT LEVEL RISK FOR PLANT LIFETIME
C                  EXPOSURE
C      HRSK        PLANT POLLUTANT LEVEL RISK FOR HUMAN LIFETIME
C                  EXPOSURE
C      BRSK        BACKGROUND POLLUTANT LEVEL RISK
C
C      COMMON      DESCRIPTION
C      NPR         NUMBER OF POPULATION SUBREGIONS
C
C      EXTERNALS   DESCRIPTION
C      DOSEC       SUBROUTINE TO CALCULATE EXPOSURE DOSES
C      DEXP        FORTRAN DOUBLE PRECISION EXPONENTIAL FUNCTION
C      RC          FUNCTION TO CALCULATE RISK
C
C      VARIABLE    DESCRIPTION
C      PDOSE       PLANT LEVEL EXPOSURE DOSE BY SUBREGION FOR PLANT
C                  LIFETIME EXPOSURE (1-DIM DOUBLE PRECISION ARRAY)
C      HDOSE       PLANT LEVEL EXPOSURE DOSE BY SUBREGION FOR HUMAN
C                  LIFETIME EXPOSURE (1-DIM DOUBLE PRECISION ARRAY)
C      BDOSE       BACKGROUND LEVEL EXPOSURE DOSE BY SUBREGION (1-DIM
C                  DOUBLE PRECISION ARRAY)
C      PPR         HEALTH EFFECT PROBABILITY FOR PLANT LEVEL DOSE BY
C                  SUBREGION FOR PLANT LIFETIME EXPOSURE (1-DIM DOUBLE
C                  PRECISION ARRAY)
C      HPR         HEALTH EFFECT PROBABILITY FOR PLANT LEVEL DOSE BY
C                  SUBREGION FOR HUMAN LIFETIME EXPOSURE (1-DIM DOUBLE
C                  PRECISION ARRAY)
C      BPR         HEALTH EFFECT PROBABILITY FOR BACKGROUND DOSE BY
C                  SUBREGION (1-DIM DOUBLE PRECISION ARRAY)
C
C      CALLED BY   HEALTH
C
C      DOUBLE PRECISION DA,PPR(100),BPR(100),PDOSE(100),HDOSE(100),
1      BDOSE(100),HPR(100)
C      COMMON /N/NPG,NPR,PL
C
C      STEP 1      CALCULATE PLANT LEVEL AND BACKGROUND DOSES.
C
C      CALL DOSEC(IM,0,PDOSE,BDOSE)
C      CALL DOSEC(IM,1,HDOSE,BDOSE)
C
C      STEP 2      CONVERT MODEL PARAMETER TO DOUBLE PRECISION.
C                  CALCULATE PROBABILITIES BY SUBREGION FOR PLANT LEVEL
C                  AND BACKGROUND DOSES.
C
C      DA = A
C      DO 20 J = 1,NPR
C      PPR(J) = 1.0D00-DEXP(-DA*PDOSE(J))
C      HPR(J) = 1.0D00-DEXP(-DA*HDOSE(J))
C      BPR(J) = 1.0D00-DEXP(-DA*BDOSE(J))
20 CONTINUE

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C
C STEP 3      USE PROBABILITIES TO CALCULATE PLANT LEVEL AND
C              BACKGROUND RISKS.
C
C PRSK = RC(PPR)
C HRSK = RC(HPR)
C BRSK = RC(BPR)
C RETURN
C END

```

SUBROUTINE MULTIH(X,A,IM,PRSK,HRSK,BSRK)

```

C
C TITLE (MULTIHIT MODEL RISK)
C
C SUBROUTINE MULTIH CALCULATES PLANT LEVEL AND BACKGROUND RISKS
C USING THE MULTIHIT DOSE-RESPONSE MODEL.
C
C PARAMETER      DESCRIPTION
C X              DOSE PARAMETER FOR MULTIHIT MODEL
C A              NUMBER OF HITS IN MULTIHIT MODEL
C IM             INDEX SPECIFYING EXPOSURE INTAKE MODE (INTEGER)
C PRSK           PLANT POLLUTANT LEVEL RISK FOR PLANT LIFETIME
C                EXPOSURE
C HRSK           PLANT POLLUTANT LEVEL RISK FOR HUMAN LIFETIME
C                EXPOSURE
C BRSK           BACKGROUND POLLUTANT LEVEL RISK
C
C COMMON         DESCRIPTION
C NPR            NUMBER OF POPULATION SUBREGIONS (INTEGER)
C
C EXTERNALS      DESCRIPTION
C DOSEC          SUBROUTINE TO CALCULATE EXPOSURE DOSES
C RGAMMA         FUNCTION TO CALCULATE MULTIHIT PROBABILITIES
C RC            FUNCTION TO CALCULATE RISK
C
C VARIABLE       DESCRIPTION
C PDOSE          PLANT LEVEL EXPOSURE DOSE BY SUBREGION FOR PLANT
C                LIFETIME EXPOSURE (1-DIM DOUBLE PRECISION ARRAY)
C HDOSE          PLANT LEVEL EXPOSURE DOSE BY SUBREGION FOR HUMAN
C                LIFETIME EXPOSURE (1-DIM DOUBLE PRECISION ARRAY)
C BDOSE          BACKGROUND LEVEL EXPOSURE DOSE BY SUBREGION (1-DIM
C                DOUBLE PRECISION ARRAY)
C DX             DOUBLE PRECISION CONVERSION OF MULTIHIT MODEL DOSE
C                PARAMETER (DOUBLE PRECISION)
C DA             DOUBLE PRECISION CONVERSION OF NUMBER OF HITS IN
C                MULTIHIT MODEL (DOUBLE PRECISION)
C XP             CONVERTED PLANT LEVEL DOSE FOR PLANT LIFETIME
C                EXPOSURE (DOUBLE PRECISION)
C XH             CONVERTED PLANT LEVEL DOSE FOR HUMAN LIFETIME
C                EXPOSURE (DOUBLE PRECISION)
C XB             CONVERTED BACKGROUND LEVEL DOSE (DOUBLE PRECISION)
C PPR           HEALTH EFFECT PROBABILITY FOR PLANT LEVEL DOSE BY

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C          SUBREGION FOR PLANT LIFETIME EXPOSURE (1-DIM
C          DOUBLE PRECISION ARRAY)
C      HPR      HEALTH EFFECT PROBABILITY FOR PLANT LEVEL DOSE BY
C              SUBREGION FOR HUMAN LIFETIME EXPOSURE (1-DIM
C              DOUBLE PRECISION ARRAY)
C      BPR      HEALTH EFFECT PROBABILITY FOR BACKGROUND DOSE BY
C              SUBREGION (1-DIM DOUBLE PRECISION ARRAY)
C
C      COMMON /N/NPG,NPR,PL
C      DOUBLE PRECISION PPR(100),HPR(100),BPR(100),PDOSE(100),BDOSE(100),
1      HDOSE(100),DX,XP,XH,XB,DA
C
C      STEP 1      CALCULATE PLANT LEVEL AND BACKGROUND DOSES.
C
C      CALL DOSEC(IM,0,PDOSE,BDOSE)
C      CALL DOSEC(IM,1,HDOSE,BDOSE)
C
C      STEP 2      CONVERT MODEL PARAMETERS TO DOUBLE PRECISION.
C
C      DX = X
C      DA = A
C
C      STEP 3      CALCULATE CONVERTED PLANT LEVEL AND BACKGROUND
C                  POLLUTANT DOSES AND RISK PROBABILITIES BY SUBREGION.
C
C      DO 20 J = 1,NPR
C      XP = DX*PDOSE(J)
C      XH = DX*HDOSE(J)
C      XB = DX*BDOSE(J)
C      PPR(J) = RGAMMA(DA,XP)
C      HPR(J) = RGAMMA(DA,XH)
C      BPR(J) = RGAMMA(DA,XB)
20  CONTINUE
C
C      STEP 4      USE RISK PROBABILITIES TO CALCULATE PLANT LEVEL AND
C                  BACKGROUND RISKS.
C
C      PRSK = RC(PPR)
C      HRSK = RC(HPR)
C      BRSK = RC(BPR)
C      RETURN
C      END
C
C      SUBROUTINE MULTAD(NST,IM,PRSK,HRSK,BRSK)
C
C      TITLE (MULTISTAGE ARMITAGE/DOLL MODEL RISK)
C
C      SUBROUTINE MULTAD CALCULATES THE PLANT LEVEL AND BACKGROUND RISKS
C      USING THE ARMITAGE/DOLL VERSION OF THE MULTISTAGE DOSE-RESPONSE
C      MODEL.
C
C      PARAMETER      DESCRIPTION

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C   NST          NUMBER OF STAGES IN THE MULTISTAGE MODEL (INTEGER)
C   IM           INDEX SPECIFYING EXPOSURE INTAKE MODE (INTEGER)
C   PRSK        PLANT POLLUTANT LEVEL RISK FOR PLANT LIFETIME
C               EXPOSURE
C   HRSK        PLANT POLLUTANT LEVEL RISK FOR HUMAN LIFETIME
C               EXPOSURE
C   BRSK        BACKGROUND POLLUTANT LEVEL RISK
C
C   COMMON      DESCRIPTION
C   NPR         NUMBER OF POPULATION SUBREGIONS (INTEGER)
C   PL          RATIO OF POWER PLANT OPERATING LIFE TO HUMAN LIFE
C
C   EXTERNALS   DESCRIPTION
C   DOSEC       SUBROUTINE TO CALCULATE EXPOSURE DOSES
C   PROB        FUNCTION TO CALCULATE MULTISTAGE PROBABILITIES
C   FACT        FUNCTION TO CALCULATE FACTORIALS
C   RC          FUNCTION TO CALCULATE RISK
C
C   VARIABLE    DESCRIPTION
C   DOSE        PLANT LEVEL EXPOSURE DOSE BY SUBREGION (1-DIM DOUBLE
C               PRECISION ARRAY)
C   BDOSE       BACKGROUND LEVEL EXPOSURE DOSE BY SUBREGION (1-DIM
C               DOUBLE PRECISION ARRAY)
C   PTMP        CURRENT SUM IN PLANT LEVEL PROBABILITY CALCULATION
C               LOOP FOR PLANT LIFETIME EXPOSURE (DOUBLE PRECISION)
C   LN          UPPER LIMIT IN PLANT LEVEL PROBABILITY CALCULATION
C               LOOP (INTEGER)
C   I           DECREMENTED LOOP INDEX IN PROBABILITY CALCULATION
C               LOOP (INTEGER)
C   IK          MODEL STAGE INDICATOR IN PROBABILITY CALCULATION
C               (INTEGER)
C   TN          FRACTION OF HUMAN LIFETIME WITHOUT PLANT LEVEL
C               POLLUTANT EXPOSURE
C   PPR         HEALTH EFFECT PROBABILITY FOR PLANT LEVEL DOSE BY
C               SUBREGION FOR PLANT LIFETIME EXPOSURE (1-DIM DOUBLE
C               PRECISION ARRAY)
C   HPR         HEALTH EFFECT PROBABILITY FOR PLANT LEVEL DOSE BY
C               SUBREGION FOR HUMAN LIFETIME EXPOSURE (1-DIM DOUBLE
C               PRECISION ARRAY)
C   BPR         HEALTH EFFECT PROBABILITY FOR BACKGROUND DOSE BY
C               SUBREGION (1-DIM DOUBLE PRECISION ARRAY)
C
C   CALLED BY   HEALTH
C
C   COMMON /N/NPG,NPR,PL
C   DOUBLE PRECISION PTMP,PPR(100),HPR(100),BPR(100),DOSE(100),
1      BDOSE(100)
C
C   STEP 1      CALCULATE PLANT LEVEL AND BACKGROUND EXPOSURE DOSES.
C
C   CALL DOSEC(IM,1,DOSE,BDOSE)
C

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C      STEP 2      USING THE EQUATION FOR MULTISTAGE PROBABILITIES UNDER
C                  VARIABLE EXPOSURE RATES, BEGIN THE LOOP TO CALCULATE
C                  THE PLANT LEVEL RISK PROBABILITY BY SUBREGION.
C
C      DO 20 J = 1,NPR
C
C      STEP 3      INITIALIZE THE LOOP SUMMATION VARIABLE AND DETERMINE
C                  THE UPPER LIMIT FOR THE PROBABILITY CALCULATION LOOP.
C
C      PTMP = 0.0D00
C      LN = NST+1
C
C      STEP 4      PASS THROUGH THE LOOP TO CALCULATE THE PLANT LEVEL
C                  RISK PROBABILITY.
C
C      DO 10 N = 1,LN
C      I = N-1
C      IK = NST-I
C      TN = 1.0-PL
C      PTMP = PTMP+(PROB(IK,DOSE(J),PL)*PROB(NST,BDOSE(J),TN)*FACT(NST))
C      1 / (PROB(IK,BDOSE(J),TN)*FACT(I)*FACT(IK))
C 10 CONTINUE
C
C      STEP 5      DETERMINE PLANT LEVEL AND BACKGROUND RISK
C                  PROBABILITIES FOR THE CURRENT SUBREGION.
C
C      PPR(J) = PTMP
C      HPR(J) = PROB(NST,DOSE(J),1.0)
C      BPR(J) = PROB(NST,BDOSE(J),1.0)
C 20 CONTINUE
C
C      STEP 6      CALCULATE PLANT LEVEL AND BACKGROUND RISKS FROM
C                  THE RISK PROBABILITIES.
C
C      PRSK = RC(PPR)
C      HRSK = RC(HPR)
C      BRSK = RC(BPR)
C      RETURN
C      END
C
C      SUBROUTINE MULTC(A,NST,IM,PRSK,HRSK,BSRK)
C
C      TITLE (MULTISTAGE CRUMP MODEL RISK)
C
C      SUBROUTINE MULTC CALCULATES THE PLANT LEVEL AND BACKGROUND RISK
C      USING THE CRUMP VERSION OF THE MULTISTAGE DOSE-RESPONSE MODEL.
C
C      PARAMETER      DESCRIPTION
C      A              DOSE PARAMETERS FOR THE CRUMP MODEL (1-DIM REAL
C                      ARRAY)
C      NST            NUMBER OF STAGES IN THE CRUMP MODEL (INTEGER)
C      IM             INDEX SPECIFYING EXPOSURE INTAKE MODE (INTEGER)

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C      PRSK      PLANT POLLUTANT LEVEL RISK FOR PLANT LIFETIME
C      EXPOSURE
C      HRSK      PLANT POLLUTANT LEVEL RISK FOR HUMAN LIFETIME
C      EXPOSURE
C      BRSK      BACKGROUND POLLUTANT LEVEL RISK
C
C      COMMON    DESCRIPTION
C      NPR      NUMBER OF POPULATION SUBREGIONS (INTEGER)
C
C      EXTERNALS  DESCRIPTION
C      DOSEC     SUBROUTINE TO CALCULATE EXPOSURE DOSES
C      DEXP      FORTRAN DOUBLE PRECISION EXPONENTIAL FUNCTION
C      RC        FUNCTION TO CALCULATE RISK
C
C      VARIABLE  DESCRIPTION
C      PDOSE     PLANT LEVEL EXPOSURE DOSE BY SUBREGION FOR PLANT
C      LIFETIME EXPOSURE (1-DIM DOUBLE PRECISION ARRAY)
C      HDOSE     PLANT LEVEL EXPOSURE DOSE BY SUBREGION FOR HUMAN
C      LIFETIME EXPOSURE (1-DIM DOUBLE PRECISION ARRAY)
C      BDOSE     BACKGROUND LEVEL EXPOSURE DOSE BY SUBREGION (1-DIM
C      DOUBLE PRECISION ARRAY)
C      DA        DOUBLE PRECISION CONVERSION OF CRUMP MODEL PARAMETERS
C      (1-DIM DOUBLE PRECISION ARRAY)
C      BTM       CURRENT BACKGROUND PROBABILITY SUMMATION VARIABLE IN
C      CALCULATION LOOP (DOUBLE PRECISION)
C      PTM       CURRENT PLANT LEVEL PROBABILITY SUMMATION VARIABLE IN
C      PLANT LIFETIME CALCULATION LOOP (DOUBLE PRECISION)
C      HTM       CURRENT PLANT LEVEL PROBABILITY SUMMATION VARIABLE IN
C      HUMAN LIFETIME CALCULATION LOOP (DOUBLE PRECISION)
C      LN        UPPER LIMIT IN PROBABILITY CALCULATION LOOP (INTEGER)
C      I         DECREMENTED LOOP INDEX IN PROBABILITY CALCULATION
C      LOOP (INTEGER)
C      PPR       HEALTH EFFECT PROBABILITY FOR PLANT LEVEL DOSE BY
C      SUBREGION FOR PLANT LIFETIME EXPOSURE (1-DIM DOUBLE
C      PRECISION ARRAY)
C      HPR       HEALTH EFFECT PROBABILITY FOR PLANT LEVEL DOSE BY
C      SUBREGION FOR HUMAN LIFETIME EXPOSURE (1-DIM DOUBLE
C      PRECISION ARRAY)
C      BPR       HEALTH EFFECT PROBABILITY FOR BACKGROUND DOSE BY
C      SUBREGION (1-DIM DOUBLE PRECISION ARRAY)
C
C      CALLED BY  HEALTH
C
C      COMMON /N/NPG,NPR,PL
C      REAL A(10)
C      DOUBLE PRECISION DA(10),PPR(100),BPR(100),PDOSE(100),BDOSE(100),
1      HPR(100),HDOSE(100),HTM,PTM,BTM
C
C      STEP 1      CALCULATE PLANT LEVEL AND BACKGROUND EXPOSURE DOSES.
C
C      CALL DOSEC(IM,0,PDOSE,BDOSE)
C      CALL DOSEC(IM,1,HDOSE,BDOSE)

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C
C STEP 2      CONVERT DOSE-RESPONSE MODEL PARAMETERS TO DOUBLE
C             PRECISION.
C
C   DO 5 I = 1,10
5 DA(I) = A(I)
C
C STEP 3      BEGIN LOOP TO CALCULATE RISK PROBABILITIES BY
C             SUBREGION.  SET SUMMATION VARIABLES TO ZERO AND
C             DETERMINE UPPER LIMIT FOR PROBABILITY CALCULATION
C             LOOP.
C
C   DO 20 J = 1,NPR
C   BTM = 0.0D00
C   PTM = 0.0D00
C   HTM = 0.0D00
C   LN = NST+1
C
C STEP 4      LOOP THROUGH STAGES TO CALCULATE PLANT LEVEL AND
C             BACKGROUND PROBABILITY SUMMATIONS FOR THIS SUBREGION.
C
C   DO 10 N = 1,LN
C   I = N-1
C   PTM = PTM+DA(N)*(PDOSE(J)**I)
C   HTM = HTM+DA(N)*(HDOSE(J)**I)
C   BTM = BTM+DA(N)*(BDOSE(J)**I)
10 CONTINUE
C
C STEP 5      CALCULATE PLANT LEVEL AND BACKGROUND RISK
C             PROBABILITIES FROM SUMMATION VARIABLES.
C
C   PPR(J) = 1.0D00-DEXP(-PTM)
C   HPR(J) = 1.0D00-DEXP(-HTM)
C   BPR(J) = 1.0D00-DEXP(-BTM)
20 CONTINUE
C
C STEP 6      USE RISK PROBABILITIES TO DETERMINE PLANT LEVEL AND
C             BACKGROUND RISKS.
C
C   PRSK = RC(PPR)
C   HRSK = RC(HPR)
C   BRSK = RC(BPR)
C   RETURN
C   END
C
C   SUBROUTINE PROBIT(A,B,IM,PRSK,HRSK,BRSK)
C
C   TITLE (PROBIT MODEL RISK)
C
C   SUBROUTINE PROBIT CALCULATES THE PLANT LEVEL AND BACKGROUND RISK
C   USING THE PROBIT DOSE-RESPONSE MODEL.
C

```

C	PARAMETER	DESCRIPTION
C	A	SLOPE PARAMETER FOR DOSE CALCULATION IN PROBIT MODEL
C	B	INTERCEPT PARAMETER FOR DOSE CALCULATION IN PROBIT
C		MODEL
C	IM	INDEX SPECIFYING EXPOSURE INTAKE MODE (INTEGER)
C	PRSK	PLANT POLLUTANT LEVEL RISK FOR PLANT LIFETIME
C		EXPOSURE
C	HRSK	PLANT POLLUTANT LEVEL RISK FOR HUMAN LIFETIME
C		EXPOSURE
C	BRSK	BACKGROUND POLLUTANT LEVEL RISK
C		
C	COMMON	DESCRIPTION
C	NPR	NUMBER OF POPULATION SUBREGIONS (INTEGER)
C		
C	EXTERNALS	DESCRIPTION
C	DOSEC	SUBROUTINE TO CALCULATE EXPOSURE DOSES
C	DLOG10	FORTTRAN DOUBLE PRECISION BASE-10 LOG FUNCTION
C	CUMND	FUNCTION TO CALCULATE CUMULATIVE NORMAL DISTRIBUTION
C	RC	FUNCTION TO CALCULATE RISK
C		
C	VARIABLE	DESCRIPTION
C	PDOSE	PLANT LEVEL EXPOSURE DOSE BY SUBREGION FOR PLANT
C		LIFETIME EXPOSURE (1-DIM DOUBLE PRECISION ARRAY)
C	HDOSE	PLANT LEVEL EXPOSURE DOSE BY SUBREGION FOR HUMAN
C		LIFETIME EXPOSURE (1-DIM DOUBLE PRECISION ARRAY)
C	BDOSE	BACKGROUND LEVEL EXPOSURE DOSE BY SUBREGION (1-DIM
C		DOUBLE PRECISION ARRAY)
C	DA	DOUBLE PRECISION CONVERSION OF PROBIT DOSE INTERCEPT
C		PARAMETER (DOUBLE PRECISION)
C	DB	DOUBLE PRECISION CONVERSION OF PROBIT DOSE SLOPE
C		PARAMETER (DOUBLE PRECISION)
C	PTD	TOTAL CONVERTED PLANT LEVEL DOSE FOR PLANT LIFETIME
C		EXPOSURE (DOUBLE PRECISION)
C	HTD	TOTAL CONVERTED PLANT LEVEL DOSE FOR HUMAN LIFETIME
C		EXPOSURE (DOUBLE PRECISION)
C	BTD	TOTAL CONVERTED BACKGROUND LEVEL DOSE (DOUBLE
C		PRECISION)
C	PPR	HEALTH EFFECT PROBABILITY FOR PLANT LEVEL DOSE BY
C		SUBREGION FOR PLANT LIFETIME EXPOSURE (1-DIM DOUBLE
C		PRECISION ARRAY)
C	HPR	HEALTH EFFECT PROBABILITY FOR PLANT LEVEL DOSE BY
C		SUBREGION FOR HUMAN LIFETIME EXPOSURE (1-DIM DOUBLE
C		PRECISION ARRAY)
C	BPR	HEALTH EFFECT PROBABILITY FOR BACKGROUND DOSE BY
C		SUBREGION (1-DIM DOUBLE PRECISION ARRAY)
C		
C	CALLED BY	HEALTH
C		
		DOUBLE PRECISION PPR(100),BPR(100),PDOSE(100),BDOSE(100),PTD,BTD,
1		HPR(100),HDOSE(100),HTD,DA,DB
		COMMON /N/NPG,NPR,PL
C		


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C STEP 1 CALCULATE PLANT LEVEL AND BACKGROUND EXPOSURE DOSES.
C
CALL DOSEC(IM,0,PDOSE,BDOSE)
CALL DOSEC(IM,1,HDOSE,BDOSE)
C
C STEP 2 CONVERT MODEL PARAMETERS TO DOUBLE PRECISION. BEGIN
C LOOP TO CALCULATE RISK PROBABILITIES BY SUBREGION.
C
DA = A
DB = B
DO 20 J = 1,NPR
C
C STEP 3 CONVERT DOSE RATES TO PROBIT LOG FORM AND CALCULATE
C PLANT LEVEL AND BACKGROUND RISK PROBABILITIES.
C
PTD = DA+DB*DLOG10(PDOSE(J))
HTD = DA+DB*DLOG10(HDOSE(J))
BTD = DA+DB*DLOG10(BDOSE(J))
PPR(J) = CUMND(PTD)
HPR(J) = CUMND(HTD)
BPR(J) = CUMND(BTD)
20 CONTINUE
C
C STEP 4 USE RISK PROBABILITIES TO DETERMINE PLANT LEVEL AND
C BACKGROUND RISKS.
C
PRSK = RC(PPR)
HRSK = RC(HPR)
BRSK = RC(BPR)
RETURN
END

SUBROUTINE WEIB(B,M,V,IM,PRSK,HRSK,BPRSK,BHRSK)
C
C TITLE (WEIBULL MODEL RISK)
C
C SUBROUTINE WEIB CALCULATES THE PLANT LEVEL AND BACKGROUND RISK
C USING THE WEIBULL DOSE-RESPONSE MODEL.
C
C PARAMETER DESCRIPTION
C B MULTIPLICATIVE CONSTANT IN WEIBULL MODEL
C M DOSE EXPONENT IN WEIBULL MODEL
C V TIME EXPONENT IN WEIBULL MODEL
C IM INDEX SPECIFYING EXPOSURE INTAKE MODE (INTEGER)
C PRSK PLANT POLLUTANT LEVEL RISK FOR PLANT LIFETIME
C EXPOSURE
C HRSK PLANT POLLUTANT LEVEL RISK FOR HUMAN LIFETIME
C EXPOSURE
C BPRSK BACKGROUND POLLUTANT LEVEL RISK FOR PLANT LIFETIME
C EXPOSURE
C BHRSK BACKGROUND POLLUTANT LEVEL RISK FOR HUMAN LIFETIME
C EXPOSURE

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C
C      COMMON      DESCRIPTION
C      NPR         NUMBER OF POPULATION SUBREGIONS (INTEGER)
C      HLF         LENGTH OF HUMAN LIFE
C      PLF         SERVICE LIFE OF POWER PLANT
C
C      EXTERNAL    DESCRIPTION
C      DOSEC       SUBROUTINE TO CALCULATE EXPOSURE DOSES
C      DEXP        FORTRAN DOUBLE PRECISION EXPONENTIAL FUNCTION
C      RC          FUNCTION TO CALCULATE RISK
C
C      VARIABLE    DESCRIPTION
C      DOSE        PLANT LEVEL EXPOSURE DOSE BY SUBREGION (1-DIM DOUBLE
C                  PRECISION ARRAY)
C      BDOSE       BACKGROUND LEVEL EXPOSURE DOSE BY SUBREGION (1-DIM
C                  DOUBLE PRECISION ARRAY)
C      DB          DOUBLE PRECISION CONVERSION OF WEIBULL MULTIPLICATIVE
C                  CONSTANT PARAMETER (DOUBLE PRECISION)
C      DM          DOUBLE PRECISION CONVERSION OF WEIBULL DOSE EXPONENT
C                  (DOUBLE PRECISION)
C      DV          DOUBLE PRECISION CONVERSION OF WEIBULL TIME EXPONENT
C                  (DOUBLE PRECISION)
C      DPLF        DOUBLE PRECISION CONVERSION OF PLANT LIFE (DOUBLE
C                  PRECISION)
C      DHLF        DOUBLE PRECISION CONVERSION OF HUMAN LIFE (DOUBLE
C                  PRECISION)
C      PPR         HEALTH EFFECT PROBABILITY FOR PLANT LEVEL DOSE BY
C                  SUBREGION FOR PLANT LIFETIME EXPOSURE (1-DIM DOUBLE
C                  PRECISION ARRAY)
C      HPR         HEALTH EFFECT PROBABILITY FOR PLANT LEVEL DOSE BY
C                  SUBREGION FOR HUMAN LIFETIME EXPOSURE (1-DIM DOUBLE
C                  PRECISION ARRAY)
C      BPPR        HEALTH EFFECT PROBABILITY FOR BACKGROUND DOSE BY
C                  SUBREGION FOR PLANT LIFETIME EXPOSURE (1-DIM DOUBLE
C                  PRECISION ARRAY)
C      BHPR        HEALTH EFFECT PROBABILITY FOR BACKGROUND DOSE BY
C                  SUBREGION FOR HUMAN LIFETIME EXPOSURE (1-DIM DOUBLE
C                  PRECISION ARRAY)
C
C      CALLED BY   HEALTH
C
C      DOUBLE PRECISION PPR(100),BPPR(100),DOSE(100),BDOSE(100),DB,DM,DV,
1      DPLF,DHLF,HPR(100),BHPR(100)
C      REAL M
C      COMMON /L/HLF,PLF,FS,HS,RSG,RSH
C      COMMON /N/NPG,NPR,PL
C
C      STEP 1      CALCULATE PLANT LEVEL AND BACKGROUND EXPOSURE DOSES.
C
C      CALL DOSEC(IM,1,DOSE,BDOSE)
C
C      STEP 2      CONVERT VARIABLES TO DOUBLE PRECISION.  BEGIN LOOP TO

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C          CALCULATE RISK PROBABILITIES BY SUBREGION.
C
DB = B
DM = M
DV = V
DPLF = PLF
DHLF = HLF
DO 20 J = 1,NPR
C
C      STEP 3          CALCULATE PLANT LEVEL AND BACKGROUND RISK
C                      PROBABILITIES FOR EACH SUBREGION.
C
PPR(J) = 1.0D00-DEXP(-DB*(DOSE(J)**DM)*(DPLF**DV))
HPR(J) = 1.0D00-DEXP(-DB*(DOSE(J)**DM)*(DHLF**DV))
BPPR(J) = 1.0D00-DEXP(-DB*(BDOSE(J)**DM)*(DPLF**DV))
BHPR(J) = 1.0D00-DEXP(-DB*(BDOSE(J)**DM)*(DHLF**DV))
20 CONTINUE
C
C      STEP 4          USE RISK PROBABILITIES TO DETERMINE PLANT LEVEL AND
C                      BACKGROUND RISKS.
C
PRSK = RC(PPR)
HRSK = RC(HPR)
BPRSK = RC(BPPR)
BHRSK = RC(BHPR)
RETURN
END

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