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ESTIMATING THE CHRONIC HEALTH RISK FROM COAL-FIRED POWER PLANT TOXIC EMISSIONS

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Summary

A general methodology for performing risk assessment is briefly discussed. This approach provides a framework within which the analyst can use specific environmental transport, exposure, and dose—response models that are appropriate to a particular problem. The framework has been implemented in a computer program that incorporates simplified environmental transport models with the models for calculating exposure rates and chronic health effects. The general framework and computer program are applied to illustrative case studies of coal-fired power plant emissions of arsenic and selenium. The problem of uncertainty in the results is discussed and several potential analysis approaches evaluated. A selected approach to uncertainty analysis is applied to the case studies. General conclusions about the risk assessment process and specific conclusions about the case studies are presented.

Introduction

Legislative and administrative actions in recent years have reflected a growing national concern over the effects of toxic pollutant emissions from industrial sources. The 1977 amendments to the Federal Water Pollution Control Act 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 risks 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. Because of the increasing use of coal in power generation, toxic emissions from power plants have become the subject of growing concern within both 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.

This paper is primarly concerned with chronic human health risk. Health risk assessment has been most thoroughly developed for release of radionuclides by nuclear power plants. The Nuclear Regulatory Commission (NRC) has published guides to be used in estimating the dispersion of effluents and the subsequent annual radiation doses to the exposed human population [1—3]. Several authors have compiled lists of available computer codes for this type of analysis, including Strenge et al. [4] and Hoffman et al. [5]. Until recently, however, this extensive literature relating to both accidental and routine releases of radionuclides had not been applied to the release of other toxic pollutants. Moghissi et al. [6], Walsh et al. [7], and Rupp et al. [8] represent early attempts to develop general approaches for calculating human exposure and chronic health risk from toxic emissions.

Using this basic foundation, our research has (1) developed a general methodology for assessing the risks associated with the control of toxic substances emitted by coal-fired power plants, and (2) demonstrated the use of this methodology by applying it to two case studies. Although the risk methodology emphasizes the contamination of surface water bodies, it incorporates toxic emissions to all environmental media and represents an integrated risk assessment framework for emission of toxic pollutants from area sources. Emitted pollutants are traced through the environment to determine the exposure rates and subsequent chronic health effects in the exposed population. The general framework and its application to a case study have been described in detail in Bolten et al. [9].

General methodology

The general risk assessment framework consists 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, one must use models, measured data, or a combination of both at each stage of the analysis. The framework is designed to permit use of alternative models at the different stages of the analysis, depending on problem requirements and the specific constraints of the situation. The overall process can be described using Figs. 1 and 2, which present a schematic outline of the risk assessment process*.

*In these figures, rectangles indicate processes or 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 pollutant through the environment and the risk assessment process.

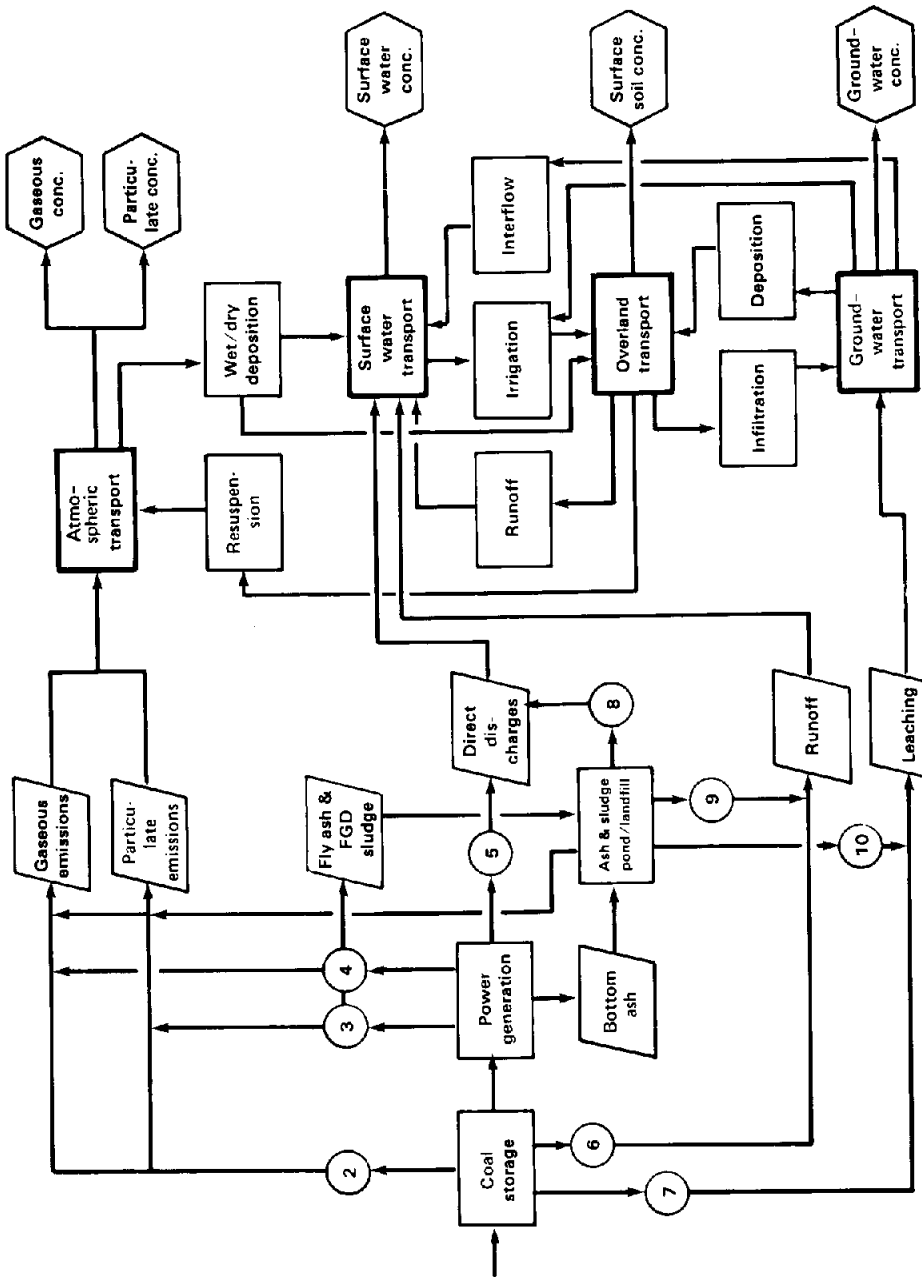

Fig. 1. Risk assessment framework. Part 1.

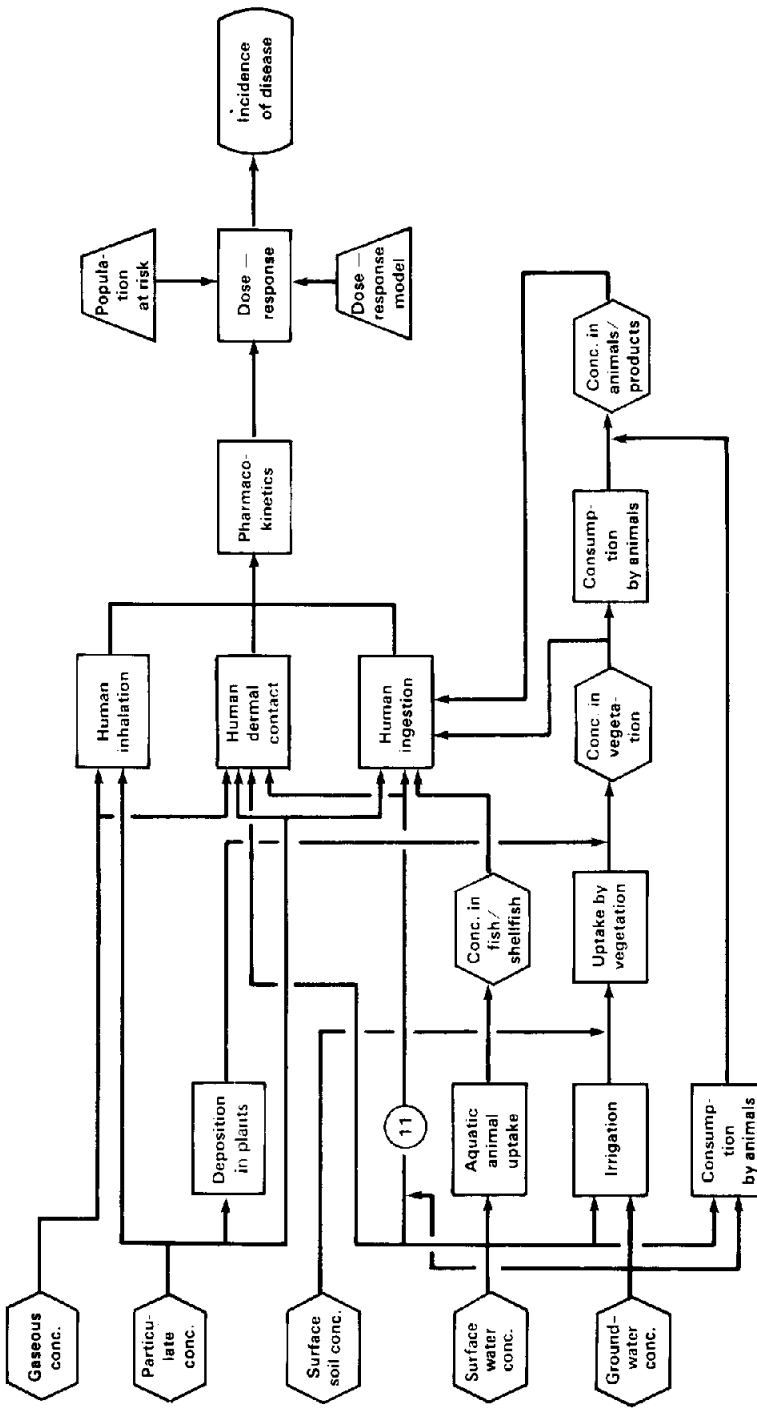

Fig. 2. Risk assessment framework. Part 2.

Figure 1 traces the toxic pollutant from the input coal (arrow entering at the left of the figure) through the source generation and environmental transport stages. The pollutant passes through the coal storage pile and various processes within the power plant to be discharged into the environment. These discharges include emissions into the air, surface water, soil, and groundwater. Discharges into the different media are dispersed, transformed, and transferred between media. The figure indicates the general processes and interactions between media that must be considered in determining the distribution of pollutant concentrations in the region surrounding the source.

Figure 2 begins with these environmental concentrations and shows the stages of exposure calculation, population description, and dose–response modeling. Pollutant concentrations in the media are passed through the exposure network to generate exposure rates by intake mode for the region. These results are combined with information about the regional population distribution and with dose–response models for the pollutant. From this, the overall risk associated with the original emission levels can be determined.

Source generation

Coal-fired power plants can emit pollutants into the air, surface water, soil, and groundwater. Because a comprehensive risk assessment should include all of these emissions, one must be able to measure or calculate the appropriate discharge rates for all pollutant species of interest. Atmospheric emissions may include both stack gases and fugitive emissions from coal storage piles and ash disposal operations. Depending on plant water management systems, many sources in a power plant can contribute to surface water pollution. These are shown schematically in Fig. 3. 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.

Environmental transport

After release from the 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, one 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 uses a fully coupled multimedia model containing submodels for each of the media (air, soil, groundwater, and surface water). These submodels are fully integrated and each can interact with all other

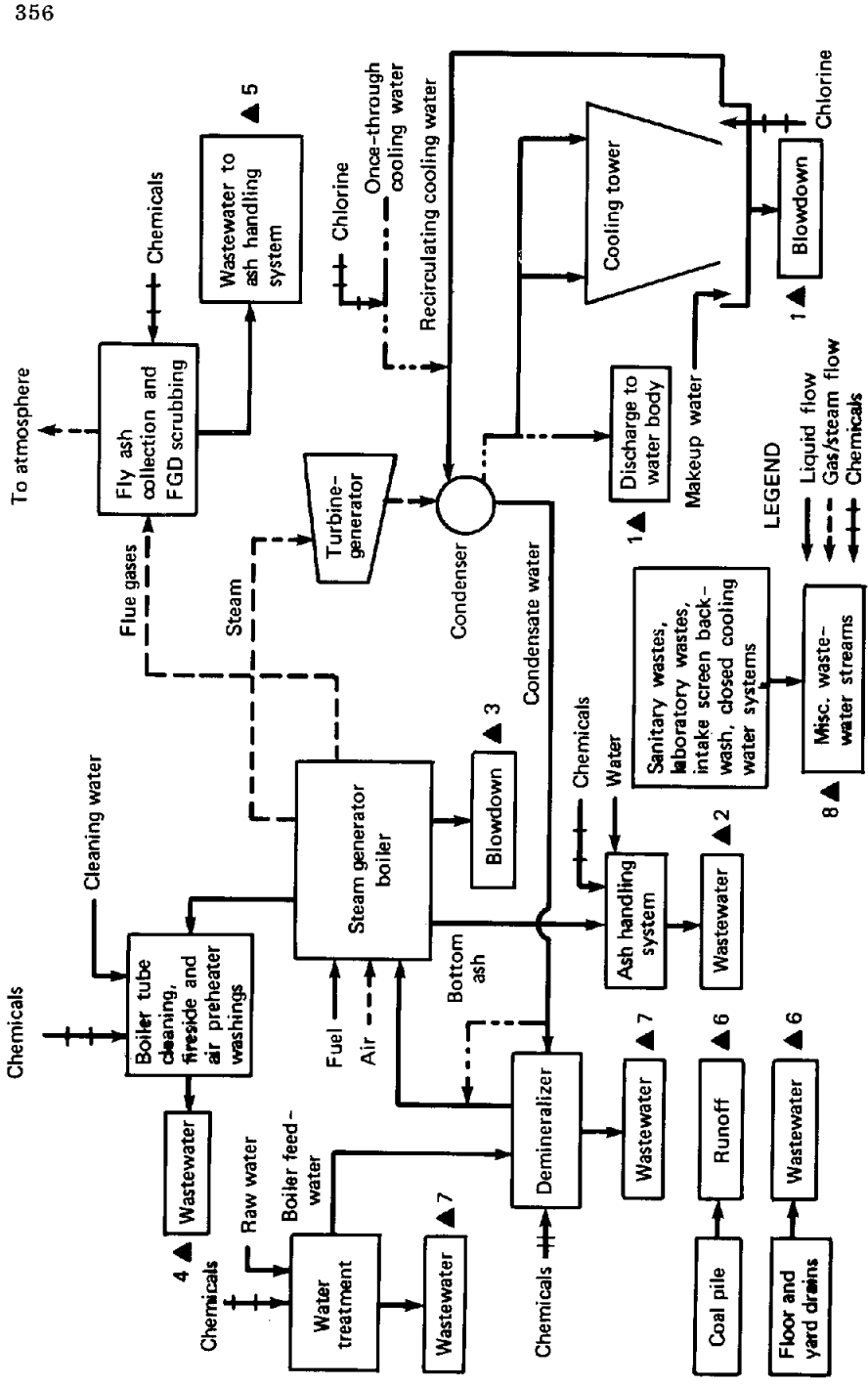

Fig. 3. Processes, flows, and emissions for a coal-fired power plant (Source: J.D. Colley et al., Assessment of Technology for Control of Toxic Effluents from the Electric Utility Industry, U.S. Environmental Protection Agency, EPA–600/7–78–090, Research Triangle Park, June 1978.

submodels. Unfortunately, this type of model can quickly become extremely large and difficult to use unless the varous 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 utilizes a composite multimedia model. In this model one selects appropriate models for each medium and couples their inputs and outputs externally, using either small computer programs or hand calculations. This allows the use of more realistic models, if they are needed, while retaining the option of employing the simplified models if they are suitable. 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. The ability to substitute alternative models also provides an opportunity to compare intermediate outputs between models and to assess the relative sensitivity of results to model choice.

For detailed analysis, we have chosen to use the composite multimedia approach. We must therefore address the problem of adequately coupling the models to account for intermedia transfers. This coupling is achieved 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 based on a general analysis of the problem and potential transfer pathways.

Extremely complex phenomena are involved in pollutant transport, transformation, and transfer processes in the air, water, soil, and groundwater. Calculating pollutant concentrations and transfer rates will normally be difficult. The environmental transport models in each medium needed to make these calculations are complex, and require extensive time for data collection and reduction as well as analysis of results. This type of analysis (which we will call Level II) is necessary, however, to provide the detailed results required in most risk assessments.

Before proceeding with a Level II environmental transport analysis using the composite multimedia approach, the analyst should perform a first-order or Level I analysis. This Level I analysis supplements the more detailed Level II assessment process and serves several purposes. Through simplified calculations of concentrations and exposure rates, the analyst can gain an appreciation of the relative importance of different environmental pathways and may be able to bound the problem. Not only will this lead to better understanding of the overall problem, but it may allow limited resources to be allocated more productively to the various aspects of the problem. If upper bounds determined using sensitivity analysis in Level I models indicate that certain pathways or emissions will not be important, the analyst can concentrate on the other areas where the Level I results are either ambiguous or definitely indicate a problem.

The Level I analysis may also be useful by indicating which intermedia

transfers are significant. This knowledge can improve the selection of environmental transport models and provide insight into the problem that can reduce the need for subsequent iterations between complex Level II models in the detailed analysis.

In general, the Level I approach uses simple models and aggregated data to make simplified calculations. The assumptions and simplifications inherent in these first approximations can generate significant errors if not used correctly. The analyst must always be aware of the limitations of the models and data and must realize that these simplified calculations are best used in extensive sensitivity analysis.

Exposure pathways

This phase of the analysis calculates total exposure by intake route for the population surrounding the emission source. The calculation involves summing the exposure from each separate pathway contributing to the three intake modes: inhalation, ingestion, and dermal contact. In addition to the direct pathways, the summation may consider pollutant transport through various stages of the food web. Because we are dealing with chronic health effects using steady-state assumptions, exposure rates will be long-term averages summed over the life of the power plant or the individual, whichever is most appropriate. Over this period, exposure rates may vary because of (1) environmental conditions, (2) 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 time.

These rates must reflect both the contribution from the power plant emissions as well as those from natural and other pollutant sources. Some of these background contributions will be incorporated into the pollutant concentrations derived from the environmental transport models. Others, such as pollutant in foods and beverages originating outside the region, must specifically be added in the exposure stage of the analysis. Although data limitations may make it difficult to estimate these contributions, one can assume that they will remain relatively constant between alternative scenarios (variations in assumed emission levels).

Population models

The risk assessment methodology should determine the net risk to regional population from chronic health conditions associated with a toxic pollutant. Population specification for the analysis should reflect, to the extent possible, variations in response to the pollutant associated with population subgroups. It should also incorporate variations in exposure rate due to location, activity, mobility, age, sex, and other characteristics. In most cases, data will not be available to incorporate these distinctions and the analysis will have to be based on a number of simplifying assumptions. As in our case studies, analysts frequently assume a stationary, con-

stant population, partitioned into several subgroups, with a given spatial distribution in the area.

Health effects

Toxic pollutants may be taken up by the population through inhalation of gases and particulates, ingestion of contaminated air, water, or locally grown foods, or dermal contact to agent-containing water or air. To assess whether these exposures constitute a significant hazard to the population, the dose of each pollutant over time must be evaluated for each geographically and biologically distinct portion of the population. These doses are used to estimate the increased risk over background of a particular toxic response (or set of responses). To pass from the dose to estimated risk, one must use 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. Because we are concerned with chronic (long-term) risk rather than acute events, we are particularly interested in dose—response models of chemical carcinogenesis*.

The commonly used dose—response models have been described extensively in the literature. The derivation of time-dependent forms for these models is described in Bolten et al. [9]. None of the common dose—response models can be said to have a more sound molecular biologic/biochemical basis than the others. Alternative models should be used whenever possible to provide various estimates of the health effects, particularly because some models typically generate either high or low response rates for given doses. In many cases, the available data may not be sufficient in quantity or quality to estimate the necessary parameters for some of the more complex models. To use a wide variety of models, one must have age-response data (dose, exposure time, and response) for three or more data sets with at least two doses, two exposure periods, and the control group included. For the toxic pollutants, this quality of information is virtually never available.

Computer program

The basic approach to calculating chronic health risk has been incorporated into a computer program called RATE (Risk Assessment of Toxic Emissions). This program, described in Bolten et al. [9, 15], contains (1) simplified environmental transport models for the air, overland, and surface water media, (2) an exposure model for the primary pathways associated with inhalation, ingestion, and dermal contact, and (3) six alternative dose—response models for calculating the health risk to the described population. This model can therefore be used in a Level I analysis of toxic

*These models may equally well be used to estimate the prevalence of other chronic diseases such as liver or kidney necrosis, teratogenicity, or fetotoxicity.

risk and as the exposure and health risk component of a Level II analysis. In the latter case, other environmental transport models (or measured values) would be used to supply the necessary pollutant concentrations in the region.

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 or meeting regulations if its results have an uncertainty of several orders of magnitude. Even if the overall uncertainty in an assessment cannot be reduced, before completing the work an analyst should try to obtain some measure of the amount of uncertainty and its primary causes.

The basic source of uncertainty in an analysis is lack of knowledge about the problem and the environment. This lack of knowledge can be separated into four areas. 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 errors in the alternative models and submodels that could be used in an analysis. These errors 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 difference sources of uncertainty in different ways. Treatment of the first source (the stochastic character of nature) is explicit in the risk estimation process and requires use of probabilistic models to describe certain occurrences and characteristics. 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 a problem.

By using alternative models at various stages of the analysis, the risk analyst can investigate the extent of model uncertainty. These errors cannot always be approximated, however. The normal method of judging model accuracy is by experimentation and model verification. Some models, particularly for environmental transport, have been to some extent verified by empirical evidence. Dose—response extrapolation models, however, have generally not been verified, and we have no prior knowledge about their adequacy. Thus, model selection must be a subjective undertaking

connected with an unknowable degree of uncertainty. In this case, one can only investigate the sensitivity of results to model choice, as we have done in one of the case studies discussed below.

Finally, uncertainty can arise from errors in measurements used to estimate the values of input data and parameters required by the various models. Because such measurements 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 propagates 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.

A number of theoretical approaches have been applied to the problem of quantifying uncertainty associated with input and parameter values. Cox and Baybutt [11] address five alternative methods; we will consider their proposals and address three more possible approaches. In general, the potential methods for dealing with parameter uncertainty include (1) analytic techniques, (2) Monte Carlo simulation, (3) response surface analysis, (4) differential sensitivity analysis, (5) confidence intervals, (6) extreme values, (7) linear propagation of errors, and (8) simple sensitivity analysis. Of these choices, five could reasonably be used in risk assessment. These are (1) response surface analysis, (2) differential sensitivity analysis, (3) extreme values, (4) linear propagation of errors, and (5) simple sensitivity analysis. The relative advantages and disadvantages of these five methods are summarized in Bolten et al. [9], using some material from Cox and Baybutt [11].

Based on the survey of approaches, the simple sensitivity analysis method was chosen for use in the case studies. In this approach, one makes three-point estimates of each parameter (a best estimate, a low, and a high value) rather than a distribution of values. During the analysis, one examines how changing the values of each parameter (among the three estimates) affects the results. For the parameters having greater influence on the net risk, point changes in two or three parameters can be made. In this way, sensitivity analysis can be considered as a somewhat informal class of uncertainty analysis.

Clearly, it would be much too difficult, in most cases, to perform sensitivity analysis for each parameter and pair of parameters. To reduce the number of parameters, one should study the models and data to determine the parameters that meet two conditions: (1) they might significantly affect the results and (2) their values are not well established and have large uncertainty. For these parameters, one makes the three-point estimates and performs the single parameter sensitivity runs. To aid in determining whether there are synergistic effects, one can perform the sensitivity analysis using several pairs of parameters. Results of this type are reported in the discussion of the case studies.

Case studies

To demonstrate how the overall risk analysis framework would be used, we have applied it to two simplified, illustrative case studies. These studies

TABLE 1

Description of 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
Ash transport and disposal system	Wet ash handling with combined ash basin and landfill disposal

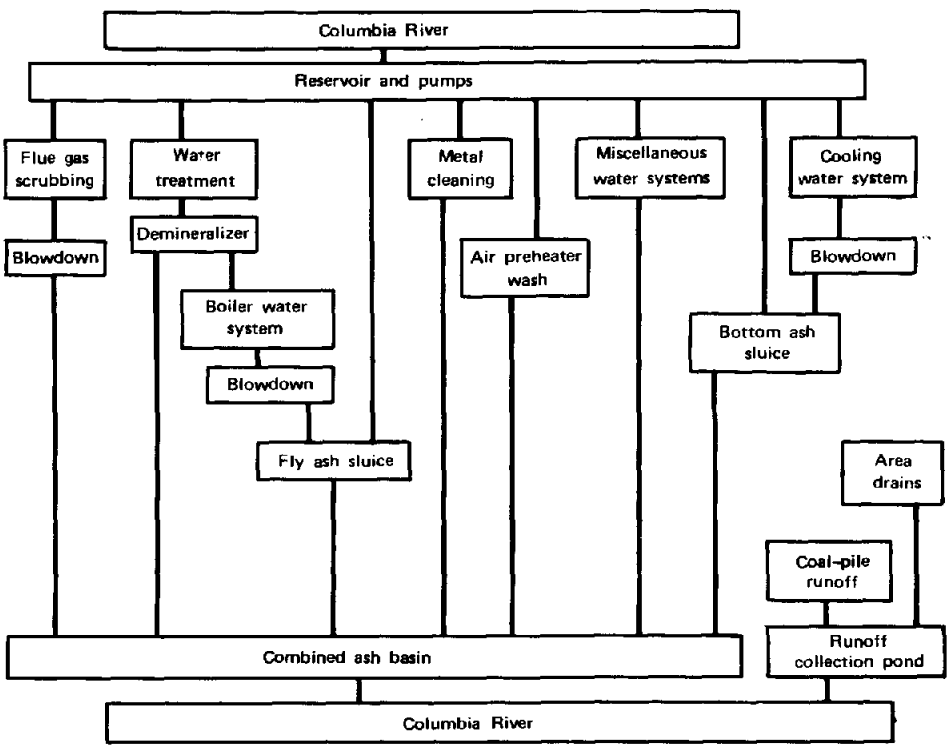

Fig. 4. Schematic diagram of power plant water systems.

involve a hypothetical coal-fired power plant of 500 MWe generating capacity sited near rivers that are used as the source of makeup water and as the receiving water for plant discharges. The power plant is assumed to be a base-load installation, operating more or less continuously. Specific characteristics of the plant are shown in Table 1. A schematic diagram of the plant water systems is shown in Fig. 4. Aside from the basic power plant description, the two case studies differ in all other aspects.

Arsenic case study

In the first study, described in Bolten et al. [9], the plant was located on the Columbia River in the Hanford Nuclear Reservation near Richland, Washington. The analysis was performed for arsenic emissions from the plant. Arsenic was selected because (1) arsenic emissions may constitute a potential health hazard, (2) emissions data are more plentiful than for most other toxic pollutants, (3) the chronic health effects of arsenic are well documented with sufficient information to estimate dose–response functions, and (4) arsenic can appear in significant concentrations in more than one power plant discharge stream.

This study focused on population exposure through ingestion of drinking water from surface sources. The exposed population was determined to be approximately 100,000, situated between 40 and 50 km downstream in the cities of Richland, Pasco, and Kennewick. This population was divided into four groups: infants, children, teenagers, and adults. These groups were assumed to consume, 0.55, 0.71, 0.71, and 1.01 l/day of drinking water from the Columbia River.

The environmental transport analysis was performed at different levels. Atmospheric transport was not included for two reasons. First, the study looked at ingestion exposure rather than inhalation. Second, because of meteorological and hydrologic conditions, runoff of deposited arsenic into the Columbia River would be negligibly small. A simplified Level I analysis for groundwater contamination determined that arsenic levels reaching the Columbia River or other drinking water sources through the soil or groundwater would also be negligibly small. Consequently, we concentrated our analysis on the risk associated with direct plant discharges of arsenic into the river.

For this work, we used a Level II time-dependent, surface water transport model, SERATRA, described in Onishi et al. [12], to simulate arsenic behavior in the water and river sediments. The background arsenic concentration in the river was assumed to be 1.6 μg/l, partitioned between the dissolved state and suspended sand, silt, and clay. 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, providing not only equilibrium conditions but also insight into the physical mechanisms creating the equilibrium conditions. The study area was 169

km long, starting 30 km above the plant site and ending 139 km below the plant at McNary Dam. This area was partitioned into 16 segments for the model. The dissolved and total arsenic concentrations in the water for the background and power plant situations are shown in Table 2.

TABLE 2

Background and computed arsenic concentrations (μg/l)

Segment	Power plant present		Background level	
	Dissolved	Total	Dissolved	Total
1	1.42	1.60	1.42	1.60
2	1.42	1.60	1.42	1.60
3	1.62	1.80	1.42	1.60
4	1.61	1.80	1.42	1.60
5	1.61	1.80	1.42	1.60
6	1.61	1.80	1.42	1.60
7	1.61	1.80	1.42	1.60
8	1.60	1.80	1.42	1.60
9	1.60	1.80	1.42	1.60
10	1.60	1.80	1.42	1.60
11	1.58	1.75	1.42	1.60
12	1.58	1.75	1.42	1.60
13	1.58	1.75	1.42	1.60
14	1.57	1.74	1.42	1.60
15	1.55	1.72	1.42	1.60
16	1.53	1.70	1.42	1.60

The analysis use the RATE program to calculate exposure rates from direct ingestion of drinking water, assuming conventional coagulation and filtration water treatment processes. The health risk to the population was calculated with RATE using four alternative dose—response models: (1) one-hit, (2) multihit, (3) probit, and (4) Weibull. Parameters for these models were estimated from data found in Tseng et al. [13, 14], which discuss epidemiological studies based on exposure of a Taiwanese population to arsenic in the water supply.

Using nominal values of all parameters and pessimistic assumptions about arsenic emissions and river flows, we calculated the net risk of skin cancer with the four dose—response models. This risk represents the expected total lifetime cases of skin cancer in the regional population caused by exposure to environmental arsenic levels minus the number of cases associated with background arsenic levels in the environment. Background arsenic levels include not only arsenic in the drinking water but also arsenic in food supplies.

Two different values were used for the exposure time in the dose—response models in an attempt to bracket the actual expected number of

cancer cases. For the lower limit, the exposure time was set to 35 years, the lifetime of the power plant. This assumes that no cases caused by plant emissions will be found in the population after this time, and clearly underestimates expected response. Exposed individuals will still carry a propensity toward the 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. For the upper limit, the exposure time was set to 72 years, the expected lifetime of an individual. 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 of this analysis are shown in Table 3. As one would expect from the nature of the dose—response models, the one-hit, multihit, and probit risks for different exposure periods scale directly with the difference in time. The Weibull model shows a much larger increase in risk because this model has a nonlinear time dependence. A more detailed discussion of this analysis and the models can be found in Bolten et al. [9].

TABLE 3

Net risk for arsenic case study (Lifetime cases)

Alternative	Dose—response model			
	One-hit	Multihit	Probit	Weibull
Plant lifetime	0.123	0.0177	0.00164	0.0202
Human lifetime	0.252	0.0364	0.00337	0.3310

As described earlier, we performed a limited sensitivity analysis for all stages of the risk assessment. We noted earlier that one major source of uncertainty in risk assessment lies in the limitations of the models used. One can deal with this problem to a limited extent. Individual models should be calibrated and verified, whenever possible, to minimize this uncertainty. Moreover, our framework permits the user to compare the results obtained from alternative models using the same input data. This information, when combined with the results of uncertainty analysis applied to model parameters and inputs, should provide insight into the application and limitations of particular models and the relative contributions of these models to overall uncertainty in the calculations.

To investigate parameter uncertainty, we performed a simple sensitivity analysis for all stages of this risk assessment. For those parameters which met the two criteria, we established both optimistic and pessimistic values. "Optimistic" values should lead to reduced estimates of risk, and "pessimistic" values should cause higher levels of risk. The nominal, optimistic, and pessimistic values for all selected parameters are shown in Table 4.

Because the arsenic emissions from the plant were worst-case values,

TABLE 4

Values for parameters in sensitivity analysis

Parameter[a]	Optimistic	Nominal	Pessimistic
K_d (m^3/kg)		13.0	1.30
BMF (mg/kg)	0.0001	0.01	0.1
PL (year)	25	35	45
TF	0.05	0.25	1.00
HS	0.0577	0.1154	0.2308
UW (l/day)	0.505	1.01	2.02
TU (kg/day)	2.56	1.28	0.64
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	1.8590	1.6070	1.3550
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—reponse model; and AW = parameters for Weibull dose—response model.

TABLE 5

Arsenic concentrations for sensitivity analysis

Segment	Power plant present		Background level	
	Dissolved	Total	Dissolved	Total
1	1.44	1.60	1.58	1.60
2	1.48	1.60	1.58	1.60
3	1.70	1.80	1.58	1.60
4	1.72	1.80	1.58	1.60
5	1.73	1.80	1.58	1.60
6	1.75	1.80	1.58	1.60
7	1.76	1.80	1.58	1.60
8	1.77	1.80	1.58	1.60
9	1.78	1.80	1.58	1.60
10	1.78	1.80	1.58	1.60
11	1.74	1.76	1.58	1.60
12	1.74	1.76	1.58	1.60
13	1.74	1.76	1.58	1.60
14	1.73	1.75	1.58	1.60
15	1.71	1.73	1.58	1.60
16	1.68	1.70	1.58	1.60

the sensitivity analysis began at the surface water transport model. In this model, which has been used in other studies, the only highly uncertain and sensitive parameter is K_d, the distribution coefficient. It measures the ratio of arsenic concentration in the sediment to concentration dissolved in water. When the value of K_d was reduced by a factor of 10 from the nominal, as shown in Table 4, the pollutant concentrations predicted by SERATRA were as shown in Table 5.

Net risk for the sensitivity cases was calculated using the dissolved arsenic

TABLE 6

Net risk results for sensitivity analysis on plant lifetime exposure

Variable	Value	Dose–response model			
		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

concentrations shown in Tables 2 and 5 with the parameter variations shown in Table 4. 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 6 and 7 and are shown in Fig. 5. These two calculations demonstrate the extremes of the range in risk expected to be encountered using the various models with a reasonably high degree of confidence. Over this wide confidence band, the resulting risks

TABLE 7

Net risk results for sensitivity analysis on human lifetime exposure

Variable	Value	Dose–response model			
		One-hit	Multhit	Probit	Weibull
All	Nominal	0.252	0.0364	0.00377	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

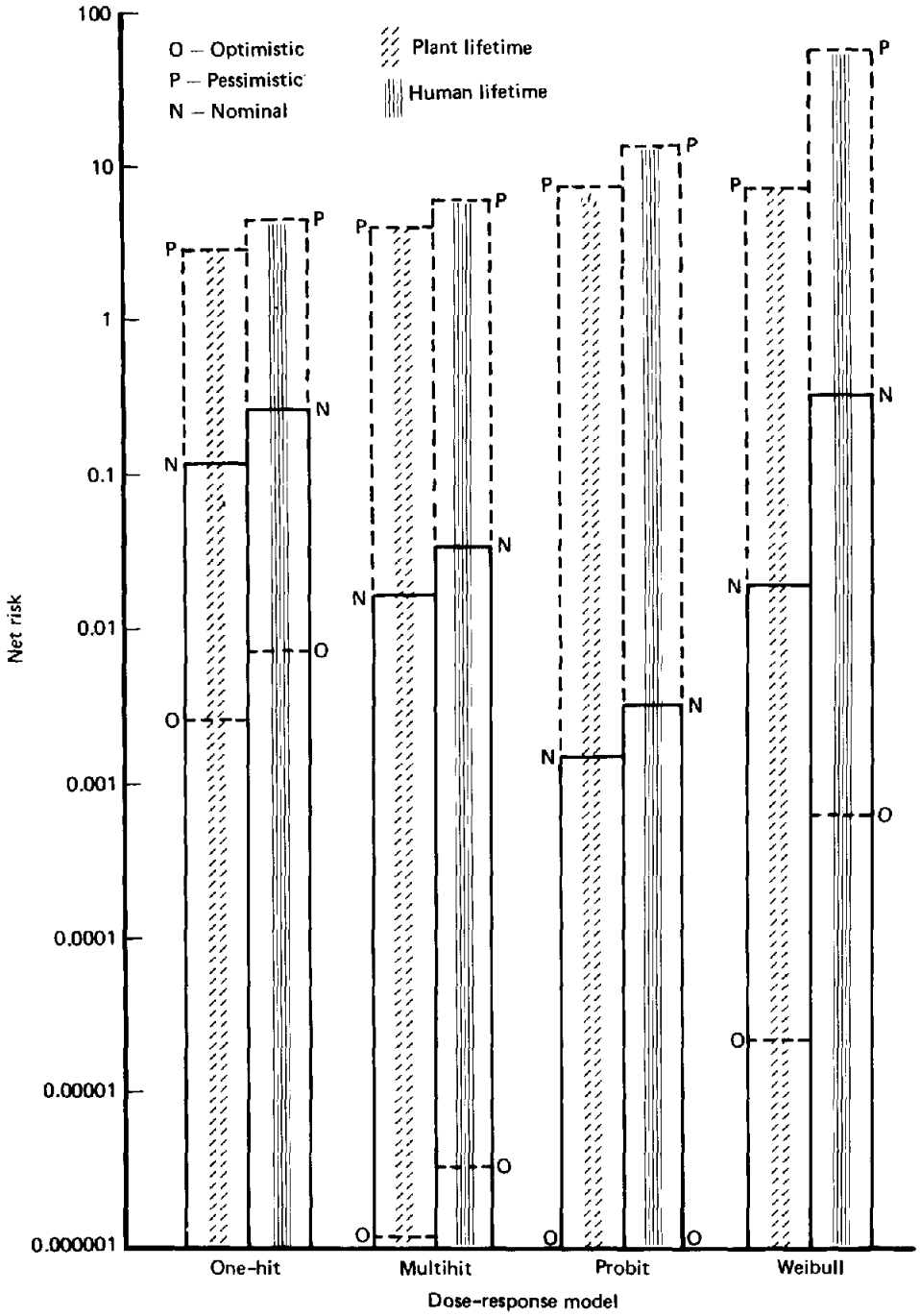

Fig. 5. Net risk under nominal, optimistic, and pessimistic assumptions.

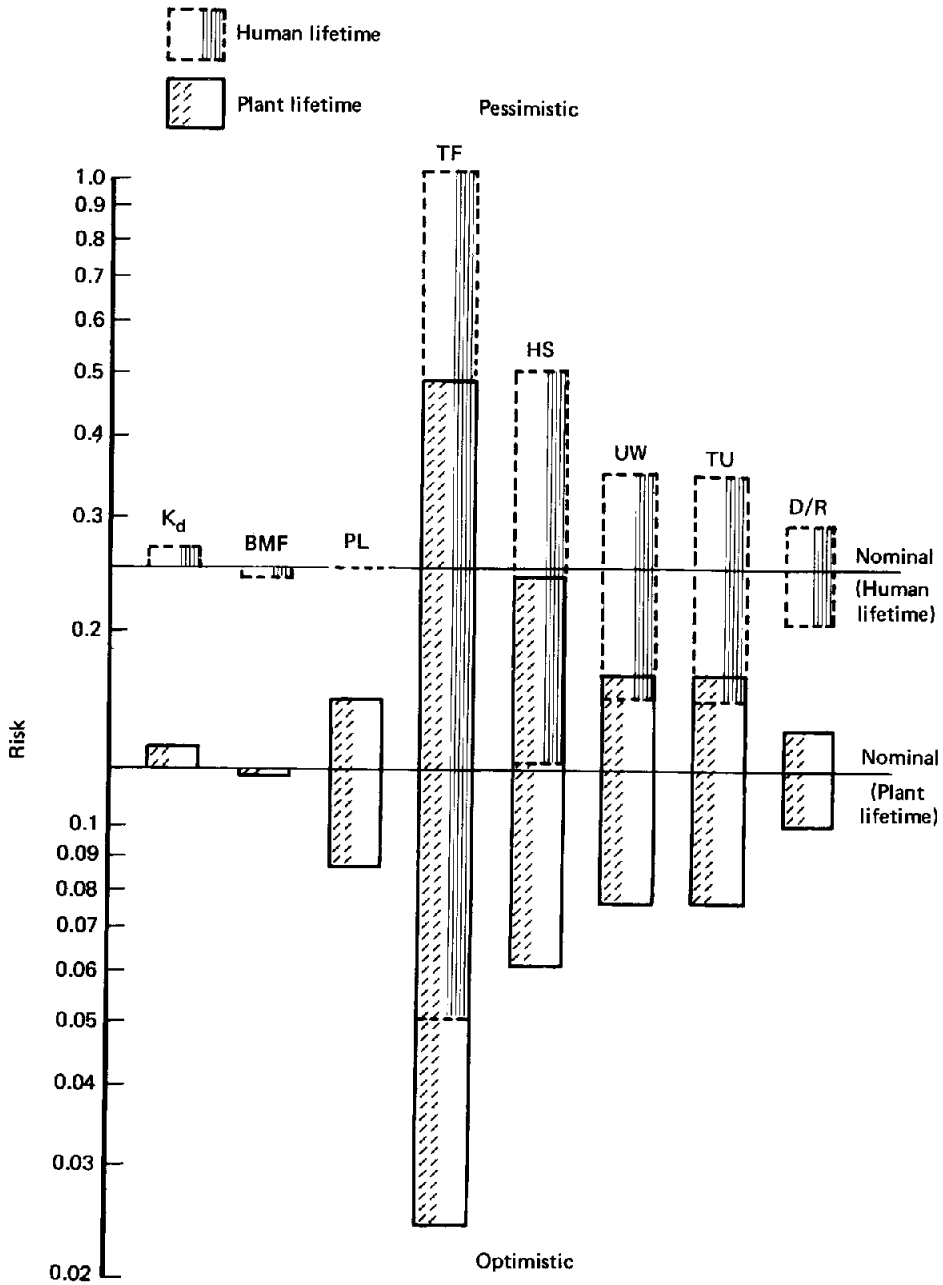

Fig. 6. Sensitivity of risk to single parameter variation (one-hit model).

vary by as much as a factor of 10^6 for the multihit model and by as little as 10^3 for the one-hit model. Using the probit model, the optimistic case estimates essentially zero net risk. The results of these best and worst cases

demonstrate such a wide range that the numbers in themselves do not provide very much useful information.

To investigate the effect of each parameter, we ran a series of calculations varying each parameter individually between optimistic and pessimistic values. These results are presented also in Tables 6 and 7. The numbers in these tables can be difficult to visualize. They are much easier to understand when presented as shown in Fig. 6, which gives the single-parameter sensitivity results for the one-hit dose—response model. Because of space limitations, we will not show similar graphs for the other dose—response models. In general, considering the results for all four dose—response models, the most sensitive parameter is TF and the least sensitive K_d and UW.

The sensitivity of the net risk estimates clearly depends on the choice of dose—response model. Results from the probit model are much more sensitive to parameter variations than results from the other models. This model is extremely sensitive to the overall background risk level (arsenic consumption level), which may be an undesirable characteristic in an analysis. Normally, one would prefer that net risks be relatively independent of the absolute background risks.

Some two-parameter sensitivity cases were run to assist in determining the linearity of combining parameters variations. These results are also shown in Tables 6 and 7 and plotted for the one-hit dose—response model in Fig. 7. These results suggest that simultaneous variations in parameter values are at most mildly synergistic, but more likely only produce effects that are the sum of their individual effects on the results.

Selenium case study

In the second case study the power plant was placed on a river (called the XYZ River) near three small towns (X, Y, and Z) in central Nebraska. Although not a major river, the XYZ has sufficient flow to meet the demands of the power plant, particularly if the plant uses a storage reservoir to hold surplus water from flood periods. The general area is rural, characterized by rolling hills, irrigated and dry land agriculture, and grazing. For reasons similar to those discussed earlier with reference to arsenic, we chose to study selenium emissions in this case. In addition, selenium has frequently been mentioned as a potential pollutant in groundwater supplies. Unfortunately, the chronic health effects of excessive selenium exposure have not been quantitatively documented.

Unlike the arsenic study, this case investigated population exposure through inhalation and all types of ingestion. The existing populations of towns X, Y, and Z were augmented to 1,000 (to compare risks between them) but the population of the remaining area was left at about 17,000. The region was partitioned into 40 subregions defined by eight 45-degree angular sectors and five 10-km radial divisions. As before, the population was divided into four groups: infants, children, teenagers, and adults. In

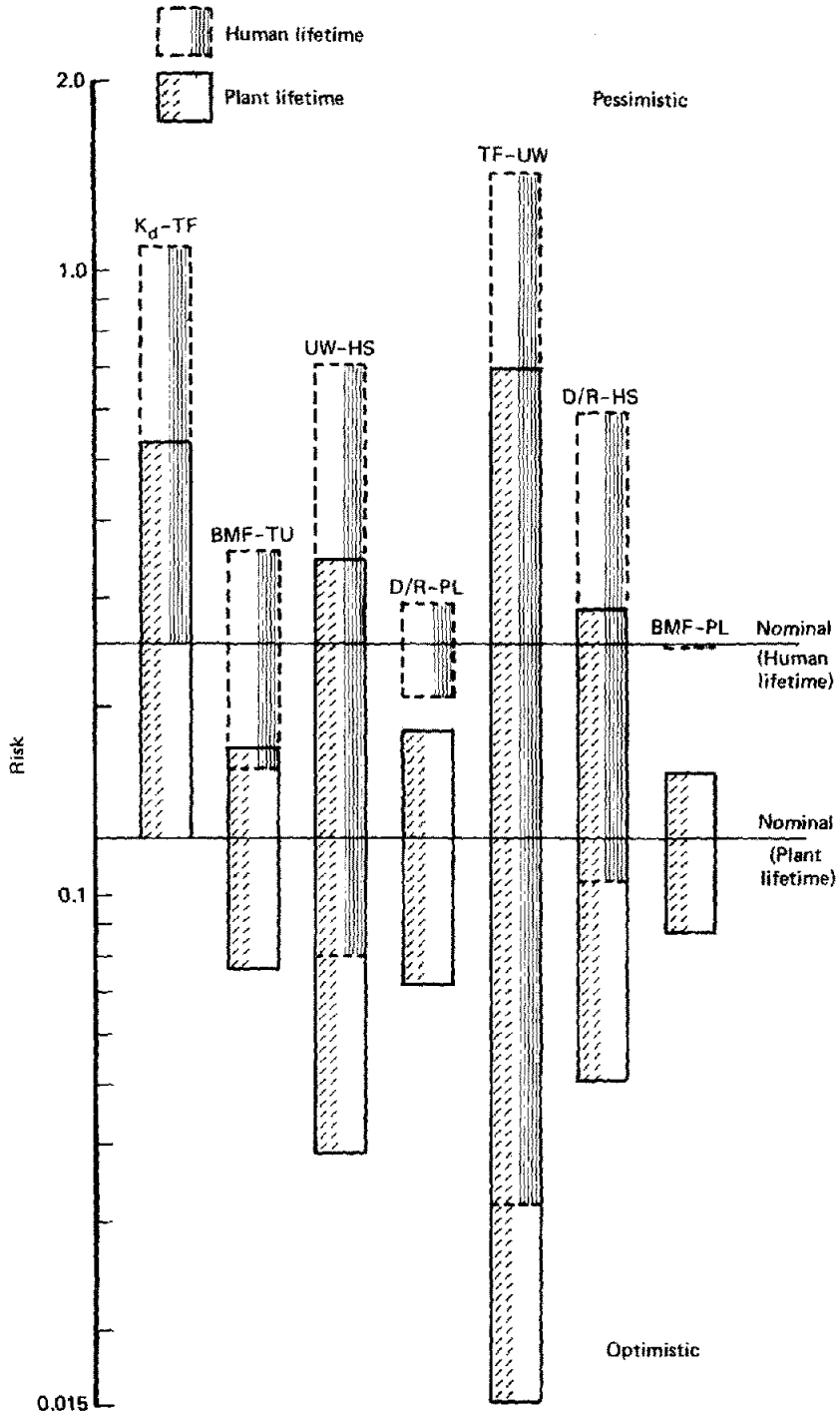

Fig. 7. Sensitivity of risk to double parameter variation (one-hit model).

addition, this study also considered potential adverse effects on the aquatic environment of the XYZ River.

The environmental transport analysis was performed at two levels. Level I analyses were done for atmospheric transport of stack emissions, for overland runoff of deposited pollutants, and for the surface water transport of direct discharges and overland runoff. The simplified models used in this analysis were those incorporated into the RATE program. First-order analysis of the area revealed that there would be no significant transfer of pollutant between surface water and groundwater bodies. A Level II analysis was done for the leaching of selenium from the plant disposal pond and landfill and subsequent transport through the soil and ground water.

The atmospheric transport analysis was performed using calculated stack emissions and characteristics with the measured distribution of wind frequency and mean speed by direction and atmospheric stability class. The model calculated mean annual pollutant concentrations and deposition rates as a function of subregion. We assumed a background selenium concentration of 5.0×10^{-9} g/m^3 and found that the incremental selenium concentrations from plant emissions were three orders of magnitude smaller.

The overland transport analysis calculated the pollutant runoff into the various reaches of the XYZ River and the distribution of concentrations in the region. The model used input information about the atmospheric deposition of selenium, irrigation rates, and pollutant concentrations in the surface water and groundwater used for irrigation. Assuming an average selenium concentration of 0.5 mg/kg in surface soil layers, we found that, under most circumstances, the incremental concentration from plant emissions was less than 0.1%. In the worst case, the concentration increased by an average of less than 2% over the life of the power plant.

Surface water transport calculations were performed with a simplified transport model assuming the river was partitioned into five reaches. Three reaches received plant emissions only through overland runoff, while the remaining two reaches were affected by the direct discharges of the power plant. The analysis assumed annual mean river discharge as the nominal flow rate, but varied this in the sensitivity studies. Other sensitivity analysis variables included the pollutant decay rate and the variables affecting the amount of overland runoff. In none of the cases did the incremental concentration from plant emissions in a reach ever exceed the background concentration of 2.0 μg/l. Under all but the "worst case" assumptions, incremental concentrations remained below 15% of background in the reaches receiving direct discharges. In those reaches affected only by runoff, the incremental concentrations normally remained below 1% of background and were always below 12%. The results of this analysis are summarized in Table 8, which shows the average incremental selenium concentration over each reach.

Clearly, the effects of plant discharges into reach 4 dominate the results.

TABLE 8

Computed incremental selenium concentrations (μg/l)

Case	Reach				
	1	2	3	4	5
Background level	2.0	2.0	2.0	2.0	2.0
Nominal case	0.00527	0.01206	0.01425	0.2547	0.2403
Slow decay	0.00537	0.01250	0.01495	0.2584	0.2547
Fast decay	0.00017	0.00015	0.00008	0.0406	0.0004
Minimum runoff	0.00036	0.00079	0.00097	0.2547	0.2102
Worst case, nominal	0.02364	0.05543	0.06696	0.3203	0.3642
Slow decay	0.02411	0.05740	0.07016	0.3277	0.3868
Fast decay	0.00077	0.00081	0.00047	0.0415	0.0017
Low flow	0.08774	0.19850	0.23940	1.1560	1.6310
Low flow/slow decay	0.08868	0.20240	0.24540	1.1700	1.6910
Best case, nominal	0.0	0.0	0.0	0.2421	0.2480
Slow decay	0.0	0.0	0.0	0.2449	0.2617
Fast decay	0.0	0.0	0.0	0.0413	0.0000
High flow	0.0	0.0	0.0	0.0819	0.0825
High flow/fast decay	0.0	0.0	0.0	0.0142	0.0000

Runoff from atmospheric and irrigation deposition processes contribute almost negligible small amounts of selenium to the stream, compared with the background levels found in regional waters. Reducing the decay coefficient increases pollutant levels only slightly, indicating that the nominal value of 8.0×10^{-7} s^{-1} is conservative. Under the worst-case conditions, with the atmospheric and overland model parameters designed to maximize runoff, selenium concentrations under nominal flow conditions are increased by less than 17% in the worst case. Only with minimum flow rates do the plant discharges increase background levels by more than 75%. Such flow rates are rare and do not persist, lasting only for a few weeks before increasing significantly. For chronic health effects, such transient variations should not be important.

The exposure analysis included inhalation and ingestion of (1) particulate air pollutants, (2) drinking water, (3) five types of animal products, and (4) one type of vegetable product. Ingestion of aquatic organisms was not included because the primary aquatic species in the area are not consumed by humans, but the study did consider how these species might be affected by plant emissions.

The analysis assumed that drinking water for all subregions came equally from surface water and groundwater. The XYZ River was the sole surface water source. The groundwater analysis determined that the population of only one subregion consumed contaminated groundwater. As before, all drinking water was given standard coagulation and filtration treatment.

For the ingestion analysis, it was necessary to determine the primary animal and vegetable products grown in the region. For the six general products selected (grains, beef, pork, milk, poultry, and eggs), we calculated production rates by county. Assuming uniform distribution of production within counties, these data could be used to estimate production rates by subregion. For each category, we also assumed that food and water used in production came from sources within the producing subregion and thus contained selenium concentrations characteristic of that subregion. Thus, grains and grasses contained selenium levels determined by multiplying the surface soil concentrations by appropriate bioconcentration factors. Consumption rates for each product were estimated from national data and are shown in Table 9.

TABLE 9

Consumption rates for the selenium case study

Product	Population group			
	Infant	Child	Teenager	Adult
Inhalation (m^3/day)	3.84	10.14	21.92	21.92
Water (l/day)	0.55	0.71	0.71	1.01
Food (kg/day)	0.55	1.19	1.50	1.28
Beef (kg/day)	0.00	0.03	0.04	0.07
Pork (kg/day)	0.00	0.03	0.04	0.07
Milk (l/day)	0.55	0.47	0.55	0.30
Poultry (kg/day)	0.00	0.01	0.02	0.03
Eggs (kg/day)	0.00	0.02	0.03	0.05
Grains (kg/day)	0.00	0.12	0.15	0.12

The health risk to the population could not be calculated using dose–response models. Although the literature contains lengthy discussions of acute and chronic health effects [16, 17], selenium toxicity cannot be easily analyzed. Selenium is probably an essential element for man, and its toxicity depends strongly on the chemical form of the element in the diet. Although selenium has been implicated in a number of chronic health conditions, including dental caries and vascular diseases, not enough quantitative information exists to fit any kind of dose–response model.

As described in Bolten et al. [15], one can use animal data to derive a threshold dose level for chronic toxicity in man. This dose level is found to be 50 μg/kg of body weight or about 3500 μg Se/day for a 70-kg individual. Similar analysis for dietary insufficiency leads to a possible minimum requirement of about 48 μg Se/day for an average human. Based on EPA [17], the average U.S. resident consumes about 132 μg Se/day. Because the study region is highly seleniferous, mean selenium intake for the exposed population is probably much higher. Using data from a variety

of sources, summarized in Bolten et al. [15], we can calculate a mean selenium intake of between 285 and 870 μg/day for the local population.

Because they live in different areas, have varying dietary preferences, and obtain food from different sources, certain elements of the population will consume more or less selenium than the average. For some individuals at the extreme high end of the distribution, the additional contribution of selenium from plant emissions might be sufficient to cross the threshold of selenosis. The selenium dietary distribution can be crudely estimated from measurements of Smith and Westfall [18]. These data can be fit to a log normal distribution and rescaled to an appropriate mean ingestion rate. Accordingly, the probability, P, that an individual will have a dietary intake exceeding the toxic threshold, I_t = 3500 μg/day, is

$$P = 1 - \frac{1}{2\pi} \int_{-\infty}^{\log I_t} e^{-z^2/2} dz$$

where $z = (\log I - \langle \log I \rangle)/\sigma_{\log I}$ and $\sigma_{\log I}$ = standard deviation of $\log I$. For the range of mean intake rates from 285 to 870 μg/day, the probability varies between 0.04 and 3%. In general, the upper figure represents a worst-case estimate which should considerably overestimate the true rate.

The basic exposure analysis was performed using nominal exposure parameter values with the soil and water pollutant concentrations found in three cases, the nominal environmental transport case and cases where transport parameter values were modified to maximize pollutant concentrations in either the soil or surface waters. These last two cases were chosen to illustrate the relative importance of the various potential exposure pathways, because specific exposure routes are not well understood, Moreover, these calculations assumed, conservatively, that all of the selenium would be in a form available for human absorption*. The average and maximum (subregion) background and plant-related exposure rates for the three cases are shown in Table 10.

TABLE 10

Exposure rates for alternative cases (μg/day)

Case	Background		With Plant	
	Mean	Maximum	Mean	Maximum
Nominal case	289.2	289.5	289.4	290.0
Worst soil			292.7	293.4
Worst water			289.8	290.4

*In general, measurements indicate the most of the selenium found in the environment is in elemental form, which is virtually insoluble in water and therefore has no significant toxicity.

Although these calculated background exposure rates agree well with the previously estimated rates discussed above, this should be considered coincidental in view of the many uncertainties involved in the calculations. The probability that an individual will exceed the toxicity threshold, given this mean exposure rate of 289.2 μg/day, becomes 0.041%, corresponding to about 8.6 people in the regional population.

The effect of plant emissions can be determined from the results shown in Table 10. In the nominal case, mean exposure rates increase by less than 0.1%. This corresponds to an increased probability of exceeding the toxic threshold of less than 0.00014%. For the regional population of 20,850, this is equivalent to an expected increase of 0.03 people. These results do not change significantly for the two worst-case transport analyses. Maximizing soil pollutant concentrations increases exposure rates by only 3.5 μg/day (about 1.2%), causing an increased probability of 0.0025% or 0.5 people. When surface water pollutant concentrations are maximized, the mean exposure rate rises by 0.6 μg/day (0.02%). This exposure leads to an increase of 0.00042% in exceedance probability or 0.09 people.

The table presents both mean and maximum exposure rates for all cases. The maximum exposure rate occurs in the subregion exposed to contaminated groundwater from ash pond leaching. The increase in exposure rate caused by this contamination is less significant than it appears to be for several reasons. First, to magnify the effects, we assumed all drinking water for that subregion was obtained from the contaminated source, rather than using the normal division between surface and groundwater sources. Second, because the groundwater has a higher background concentration of selenium, the maximum background exposure rate also occurs in this subregion. Accordingly, the maximum exposure rate for all cases should be compared with the maximum background exposure rate rather than the mean background rate. Third, the incremental selenium from plant leaching does not reach the well in significant amounts for more than 75 years, by which time the entire scenario will have changed and the power plant may no longer be operating. Fourth, the groundwater emissions used in these calculations represent a worst-case condition — an unlined pond and landfill with pessimistic model parameters. In other situations, the resulting pollutant concentrations are an order of magnitude smaller.

Finally, note also that these exposure rates are probably higher than what we would expect. First, the worst-case soil analysis assumes soil concentrations reach their maximum levels (corresponding to thirty years of accumulation) immediately and remain there for the life of the plant. Second, all cases assume the population consumes locally grown animal and vegetable products as much as possible, before consuming food produced nationwide, most of which will contain less selenium than the levels we have been considering. This is substantiated by EPA data showing the average daily per capita selenium intake in the U.S. to be only about 132 μg/day [17].

As with the arsenic study, a simple sensitivity analysis was performed for the selenium calculations. In this case, however, the analysis was more complex, because the case study included environmental transport analysis for air, soil, and surface water as well as the complex exposure pathways discussed above. As discussed earlier, the results in Table 10 represent extreme environmental transport cases with nominal exposure analysis parameters. To supplement this, we considered a number of cases using variations on the parameters in the exposure analysis models. These parameters and their nominal, optimistic, and pessimistic values are shown in Table 11.

TABLE 11

Values for parameters in selenium sensitivity analysis

Parameter[a]	Optimistic	Nominal	Pessimistic
TF	0.05	0.20	0.90
HS	0.0577	0.1154	0.2308
UW (l/day)	0.5 N	1.0 N[b]	2.0 N
UV (kg/day)	0.5 N	1.0 N[b]	2.0 N
UA (kg/day)	0.5 N	1.0 N[b]	2.0 N
UI (m^3/day)	0.5 N	1.0 N[b]	2.0 N
MF (day/kg day l)	0.5 N	1.0 N[c]	2.0 N
ACB (mg/kg)	0.2 N	1.0 N[d]	2.0 N
CV	0.13	1.30	8.00
VCB (mg/kg)	0.005	0.05	0.50
TL	0.05	0.20	1.00

[a]TF = fraction of pollutant retained after drinking water treatment; HS = ingestion exposure dose scale factor; UW = consumption rate for drinking water; UV = consumption rates for vegetable products; UA = consumption rates for animal products; UI = inhalation rates; MF = fraction of daily pollutant intake in animal products; ACB = background pollutant content of animal products from outside region; CV = bioconcentration factor for vegetable products; VCB = background pollutant content of vegetable products from outside region; TL = translocation factor to edible parts of vegetation.
[b]Nominal values given in Table 9.
[c]Nominal values: beef = 0.03 day/kg; pork = 0.20 day/kg; milk = 0.002 day/l; poultry = 4.00 day/kg; eggs = 10.00 day/kg.
[d]Nominal values: beef = 0.02 mg/kg; pork = 0.25 mg/kg; milk = 0.012 mg/l; poultry = 0.30 mg/kg; eggs = 0.10 mg/kg.

The identified parameters were varied individually and in groups, using the environmental concentrations of the nominal case and the worst-case soil and surface water variations on the nominal case. Because exposure rates varied so little in Table 10, only pessimistic cases were used in the exposure sensitivity analysis. The sensitivity cases and their results are shown in Table 12.

These results demonstrate two clear trends. First, changing parameter

TABLE 12

Exposure rates for sensitivity cases (μg/day)

Case	Parameters	Background		Power plant	
		Mean	Maximum	Mean	Maximum
Nominal	TF	291.2	292.1	291.3	294.1
	HS	289.3	289.5	289.4	290.0
	UW/UV/UA/UI	426.0	426.5	426.2	427.5
	MF	430.1	430.4	430.4	431.0
	ACB	297.0	297.3	297.1	297.8
	MF/ACB	437.9	438.1	438.1	438.7
	CV	1087.2	1087.7	1087.5	1088.3
	VCB	289.3	289.5	289.4	290.0
	TL	290.6	290.9	290.7	291.4
	TL/VCB/CV	1088.6	1089.1	1088.8	1089.7
Worst-case surface water	TF	291.2	292.1	292.3	294.4
	TF/UW	293.6	295.3	295.5	299.6
	UW/UV/UA/UI	426.1	426.5	427.1	428.1
	MF/ACB	437.9	438.1	438.9	439.4
	TL/VCB/CV	1088.6	1089.1	1089.2	1089.9
	TL/VCB/CV/MF/ACB	1628.7	1629.3	1629.7	1630.6
	TL/VCB/CV/MF/ACB/TF	1630.6	1631.8	1632.2	1634.6
Worst-case soil	HS	289.3	289.5	292.8	293.4
	UW/UV/UA/UI	426.0	426.5	432.5	433.8
	MF/ACB	437.9	438.1	443.8	444.4
	TL/VCB/CV	1088.6	1089.1	1104.3	1105.2
	TL/VCB/CV/MF/ACB	1628.7	1629.3	1635.5	1654.4

values in the exposure assessment can strongly affect exposure rates. Mean rates vary between 289.3 and 1630.6 μg/day, more than a factor of five. Second, in spite of this overall variation, incremental exposure rates from plant emissions remain small. In the most extreme cases, worst-case soil accumulation, mean exposure rates increase by less than 1.5% over background levels. Thus, although the overall level of exposure may be quite uncertain, the incremental contribution of plant emissions is relatively unaffected by the sensitivity variations. In the most extreme case, the expected increase in the probability of an individual exceeding the exposure limit is 0.48%. Although this probability may seem large, it represents not only a worst-case analysis for both environmental transport and exposure calculations, but also pessimistic assumptions about selenium availability and accumulation and consumption of locally grown produce.

The sensitivity results have important implications for the analysis. The cases involving the worst-case soil accumulations all show an increase in exposure rate of between 1.2 and 1.5%. Yet, because of the change in

background levels, these exposure rates correspond to increases in the toxicity exceedance probability of between 0.002 and 0.48%. Clearly, it is important in this situation to have the best data available for the overall selenium background exposure rate. A single study of selenium levels or consumption rate in the regional population might be sufficient to determine the magnitude of the background rates and the related risk. Because plant-related exposure differs so little from background rates in the sensitivity analysis, extensive study of most of the uncertain exposure analysis parameters may not be necessary if the overall exposure rates can be estimated for the region. If background rates are sufficiently low, the small increment of selenium to the local environment should have negligible effect.

In addition to considering the potential human health effects of selenium exposure, the analysis also investigated how increased selenium concentrations in surface waters might affect aquatic organisms in the region. Selenium in water can have both acute and chronic effects on fish. However, acute toxicity has not been recorded below concentrations of 340 μg/l, far above the levels predicted in the analysis. Chronic effects include not only life cycle survival, but also growth reduction, decreased blood iron, and reduction in egg hatchability. These effects occur at the lowest concentrations in rainbow trout [17], where hatchability can be reduced at 28 μg/l and life cycle survivability can be affected at 88 μg/l.

If the highest reported background concentration of selenium in the region (20 μg/l) is combined with the worst-case surface water concentrations found in Table 10, the result is still below the 28 μg/l which represents the lowest concentration found to have measurable effects on rainbow trout. Consequently, unless the aquatic species found in the XYZ River are far more sensitive to selenium than trout (the most sensitive species known), plant-related selenium emissions should have no significant impact on local aquatic life.

Conclusions and recommendations

Application and limitations of toxic risk analysis

Risk analysis for emissions of toxic materials has only recently become an important area. Consequently, although a 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. Besides complicating the analysis, they contribute to the overall uncertainty in risk estimates that makes regulation of toxic emissions so complex and controversial.

Risk analysis is not an exact science that can develop accurate and precise risk estimates. Instead, it 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 thus subject to limitations and potential problems. Some of these are general, relating to the selection and application of models and procedures for risk calculations. Others are more specific, in that they may be true for some analyses but not for others. These problems arise from the characteristics of a particular pollutant or situation. In this section, we first consider some of the general problems associated with risk assessment, then discuss more specific conclusions from the case studies.

No risk analysis framework or methodology should be used without a thorough understanding of the various models and their application. No one can construct a foolproof package of models that can be properly used by someone unfamiliar with the basic problem and general situation. "Cookbook" risk analysis cannot be done. The analyst must also have access to adequate information about (1) all models used in the analysis, (2) the operation and characteristics of the power plant (or other emission source) and its waste streams, (3) regional geography, geology, hydrology, and meteorology, (4) regional population, (5) the behavior and chronic health effects of the pollutant, and (6) regional production and consumption of animal, vegetable, and aquatic organisms (if relevant). This is fundamental information, although the level of detail required will vary significantly between problems. Because there are many other potential sources for most of the toxic pollutants, and regional background pollutant levels will vary greatly, the information used in an analysis must be as site-specific as possible. Use of national averages or "typical" values may lead to significant unknown errors in the results.

Environmental transport

In this type of analysis, multimedia environmental modeling is potentially the most difficult task. The composite approach attempts to produce realistic results while retaining flexibility for application to a variety of situations. Model selection can be tailored to problem requirements, data availability, user resources, and other constraints. Unfortunately, the most appropriate models for each medium may not be particularly compatible. Intermedia transfers and coupling may be difficult because the models have been designed for independent operation with differing requirements and purposes. Moreover, the models are frequently difficult and expensive to set up and operate, even for those who are already familiar with them.

As a consequence, the risk analyst must attempt to simplify the problem as much as possible without compromising the results. An initial Level I analysis should be performed to determine problem boundaries, isolate specific areas for more detailed analysis, and generally define the overall risk assessment. Then the Level II analysis can be performed as needed. At this stage, however, the analyst must accept that data collection and reduction, as well as other activities will be difficult for most practical problems, and that certain intermediate data manipulations and conver-

sions will be needed if more than one model is to be used. As individual experience with this process increases, these problems will become less important and the conversions less difficult.

Health effects

A primary weakness and source of uncertainty in risk assessment lies in the determination of health effects. This uncertainty arises from incomplete knowledge about how toxic pollutants behave in the human body and from a lack of understanding about which dose–response and extrapolation models are most realistic for any particular pollutant. Even for arsenic, a relatively well documented pollutant, dose–response calculations are limited by lack of acceptable health effects data. For the large majority of the toxic trace elements (including selenium) and organic pollutants, there is even less information, qualitative or quantitative. Moreover, as the arsenic case study showed, the choice of dose–response model can significantly influence the results. Unfortunately, there is as yet no scientific basis for preferring one model rather than another.

Although one may have appropriate models for all stages of an analysis, these may not be sufficient. Risk analysis will always be limited by data availability and understanding of how pollutants behave in the environment and human body. Therefore, the analyst should first determine whether sufficient information is available to perform a quantitative risk assessment. If health effects data are inadequate, it may be necessary to do environmental transport and exposure calculations only, comparing alternative scenarios on the basis of these results instead of actual health risk predictions. With limited data, it may be possible to perform a restricted health effects analysis, as was done for the selenium case study.

All toxic pollutants may have both acute and chronic health effects. Acute effects are caused by short-term exposure to high concentrations. Such effects would normally not be expected from exposure to power plant discharges into surface waters. Chronic effects, on the other hand, are caused by long-term exposure to low concentrations. These types of effects are much more likely. Short-term exposure to higher concentrations may contribute to chronic effects, however. Thus, for some pollutants, it could be necessary to consider both short and long-term variations in emissions and environmental concentrations. The transport and exposure calculations for this type of analysis would be complex and expensive. Generally, however, the theoretical models and data do not exist to perform the dose–response calculations that would be needed.

Time frame

Our analyses have been limited to long-term average conditions and emission levels, thus assuming that plant operations and emissions will be reasonably constant and continuous. Although discharges and environmental conditions may vary significantly over time periods less than a

week, if the resulting pollutant concentrations in the environment are below acute levels, these variations can be averaged out over the course of a year. It may sometimes be necessary, though, to consider time periods of a month or season in calculating exposure rates. Such might be necessary if plant operations are seasonal and environmental conditions (such as river flows or wind patterns) are strongly correlated with discharges. Under these conditions, the distribution of pollutant concentrations could vary strongly over time. The need for this type of analysis is conditional on the ability of the health data and models to utilize the resulting exposure variations.

By restricting the analysis to long-term conditions, one can use steady-state models and assumptions in the calculations. Variations from long-term average emissions or environmental conditions may generate transients in pollutant concentrations, but these transients will not be important. In the atmosphere and surface waters, processes occur relatively rapidly, meaning that pollutant concentrations in the environment will generally reflect plant emissions within a short time. For soil and groundwater transport, this may not be true. Processes occur much more slowly, from leachate discharge through retention and transport in saturated or unsaturated soils. Consequently, time delays become important. During the life of the power plant, groundwater concentrations may not only fail to reach a steady-state condition, but they may not even reflect plant emissions. Under these circumstances, the analyst must decide how to deal with the exposure and health effects calculations. If groundwater exposure does not become important until well after other exposure has ceased, the resulting risk must be carefully defined.

Toxic pollutants

The federal government regulates toxic emissions to surface water bodies primarily under the provisions of the Clean Water Act. This act lists 65 toxic substances or classes of substances subject to regulation. From this information, the EPA has developed a list of 129 specific priority toxic pollutants. Few of these pollutants have been studied in any detail. The pollutants differ widely with respect to their (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 pollutants have not been studied at all, in that virtually nothing is known about their behavior in the environment.

As a result, the exposure calculations must be highly uncertain. Even for the most direct exposure pathways, such as consumption of drinking water, more information is needed about how toxic organics and trace metals are affected by water treatment and distribution processes. Without this type of information, even the best computer models cannot produce results with acceptable uncertainty levels.

Sensitivity analysis

The sensitivity analyses were performed to determine reasonable upper and lower bounds on the calculation of net risk and to learn how much each model parameter affected the results. The procedure is limited in some ways, however. First, selecting high and low values around the best estimate for each parameter does not indicate the likelihood that these values will be achieved — 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 limitations, a thorough sensitivity analysis of this type can be useful. It can identify input parameters that are sufficiently important to warrant more careful consideration in the overall analysis, either by performing a more detailed analysis, using an alternative model, or investigating the value of the parameter more thoroughly. It can also indicate how much uncertainty there may be in the final results and by how much this can be reduced by improvements in the various parts of the analysis.

Case study conclusions

The case studies were designed to illustrate how part of the overall risk assessment methodology could be applied to a real problem. They were hypothetical examples, not related to any existing power plant, based on a combination of pessimistic assumptions and default values obtained from the literature. Moreover, in one instance, the background concentrations of pollutant in the affected surface water bodies were much higher than the recommended water quality criteria from the EPA [8]. The Columbia River has an arsenic concentration of 1.6 μg/l, compared with an EPA criterion of 22 ng/l, while the XYZ River has a selenium concentration of 2.0 μg/l, compared with an EPA criterion for selenium of 10 μg/l. Under these circumstances, one should not consider the case-study results to be definitive or typical of coal-fired power plants anywhere. Nevertheless, we can use these results and the sensitivity analysis to draw some limited conclusions about the risk analysis of arsenic and selenium emissions.

The arsenic and selenium results indicate that the incremental exposure and risk associated with the plant discharge are small. Arsenic concentrations in the Columbia River increased by 12.5% above background levels, which could lead to between 0.00164 and 0.329 additional cases of skin cancer in the regional population. These values correspond roughly to incremental skin cancer risks of between 0.016 and 3.29 in a million. Compared with 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, this analysis calculated final-risk estimates that could not be verified, implying that procedure verification must come from proper calibration and testing of the models at each stage of the analysis.

Similarly, the incremental selenium concentrations in the second case study do not lead to significant increases in exposure rates or risk. Atmospheric selenium levels increase by less than 0.1%. Selenium levels in surface soils increase by an average of 0.5% over the entire lifetime of the plant, under nominal circumstances, and by less than 2.0% using worst-case assumptions. Correspondingly, pollutant contributions to the surface water from runoff and atmospheric deposition are almost negligbly small compared with direct discharges from the power plant and existing background levels of selenium. Under all but the worst-case, low-flow assumptions, background concentrations in the surface water do not increase by more than a few percent.

As with the arsenic analysis, the health risk associated with the increased exposure to selenium is extremely small. The incremental risk for the nominal case is only 1.4×10^{-6}. Alternatively, maximizing soil concentrations or surface water concentrations leads to incremental risks of 2.5×10^{-5} and 4.2×10^{-6}, even assuming pessimistically that all emitted selenium will be in a form available for uptake by plants, animals, and man.

The arsenic sensitivity analysis indicated that net risk results can be extremely sensitive to the values of parameters and choice of dose–response model. The probit model is unduly sensitive to the background arsenic exposure rate and is probably not suitable for this risk analysis. For the other dose–response models, the results vary significantly with the assumption about how much arsenic is removed during drinking water treatment. This removal efficiency depends on several conditions and can be difficult to specify, as indicated by Sorg and Logsdon [19]. Thus, an actual risk analysis of this type should use measured values of the removal rate whenever possible.

Other potentially important parameters in the analysis could be more difficult to assess. Pollutant concentrations in foods are not frequently measured, although these levels can affect the incidence of chronic health effects when additional exposure occurs. Similarly, the pharmacokinetic parameters for arsenic exposure are not well understood and the data used in the dose–reponse model parameter estimation have been subject to intensive criticism in recent years. When problems such as these occur, and when literature or case-specific data are incomplete, uncertain, or inconsistent, sensitivity analysis must be used to determine or bound the effects of uncertainty in parameter values.

The selenium sensitivity analysis demonstrated that choice of parameter values could strongly affect estimates of overall exposure rate, but had little effect on the relative size of incremental exposure. Because of the modified procedure used to estimate an associated health risk, raising the background exposure rate will cause an increase in risk, even though in-

cremental exposure may remain constant. Accordingly, it becomes necessary to either evaluate parameters as accurately as possible, or to otherwise determine background exposure rates in the regional population. If these background rates are near estimated national averages, the incremental risk from power plant emissions is acceptably small. Only if these rates are extremely high do plant emissions become a possible hazard. In these circumstances, one would expect to find evidence of chronic selenium poisoning in a small, but measurable, fraction of the population.

As we have demonstrated, in general the risk assessment process demands a thorough understanding of the problem. One cannot expect to do an accurate or believable analysis without knowing the basic characteristics of the emissions, 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 might this cause the work to be more difficult, extensive, and costly than otherwise necessary, it will justifiably cast doubt on the validity of the entire study and its results.

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