ANL/CP--71887

DE91 014019

Stochastic Model for Estimating Personal Exposures in Contaminated Buildings at Superfund Sites

Donald J. Fingleton and Margaret M. MacDonell

Argonne National Laboratory
Environmental Assessment and Information Sciences Division
9700 South Cass Ave.
Argonne, Illinois 60439

Halûk Özkaynak, David A. Butler, and Jianping Xue

Harvard University
79 JFK Street
Cambridge, Massachusetts 02138

For Presentation at the 84th Annual Meeting of
the Air and Waste Management Association
Vancouver, British Columbia

June 16-21, 1991

INTRODUCTION

A hazardous waste site can pose threats to human health via transport of on-site contaminants through environmental media to human receptors. The U.S. Environmental Protection Agency (EPA) has developed a framework for assessing these potential threats in order to support Superfund cleanup decisions on the basis of risk. This framework consists of a baseline risk assessment, which is prepared to:

- Estimate risks that could occur either now or in the future if no cleanup action were taken at the site;
- Help focus the need for cleanup by highlighting the environmental media and locations that are associated with unacceptably high risk estimates (e.g., sludge pits and localized spill areas), as well as those that are not;
- Provide a basis for determining residual levels of chemicals that can remain on-site without adversely impacting human health;
- Permit risk-based comparisons between various alternatives considered for site cleanup (by identifying incremental protectiveness relative to the baseline case); and
- Provide relative consistency with the evaluation of human health threats associated with other hazardous waste sites.

In preparing the baseline risk assessment for a site, the relationship between the sources of a pollutant and the potential receptors are developed through an exposure assessment process. Quantifying human exposures to contaminants in the environment requires information on (1) contaminant sources and concentrations, (2) fate and transport of these substances from the source to the human receptor(s), and (3) receptor activity patterns. When assessing exposures associated with Superfund sites, the first two factors may be well characterized because of the extensive sampling often conducted as part of the Superfund decision-making process. Receptor activity patterns, however, are usually less well defined. Only a few exposure scenarios are typically considered in a baseline risk assessment for a Superfund site and these scenarios often use set values for parameters such as the frequency and duration of exposure for each receptor. Furthermore, few Superfund sites are uniformly contaminated, and they may include large areas that are free from contamination. Hence, an individual traversing such a site would probably come in contact with areas that would contribute little to that person's total exposure and risk. Research indicates that, in fact, receptor dynamics and microenvironment concentrations can be important to the appropriate characterization of human exposures.[1-5]

The results of an exposure assessment are combined with toxicity information to provide an estimate of carcinogenic and noncarcinogenic health risk for a site. This impact characterization forms the basis for recommending cleanup criteria and focuses the selection of remedial action alternatives. Thus, the uncertainty associated with exposure estimates at Superfund sites can directly affect the ultimate disposition of the site and potential cleanup costs. Because of these concerns relative to estimating health risks at a Superfund site, we

have used a nonstandard approach that accounts for the uncertainty in some of the exposure parameters.

Cleanup activities are currently being conducted by the U.S. Department of Energy (DOE) at a contaminated site consisting of a chemical plant area that has been inactive for more than 20 years and a noncontiguous quarry that was used for waste disposal. This 220-acre site was used by the Army to produce nitroaromatic explosives during the 1940s and DOE's predecessor to process radioactive materials of the uranium and thorium decay series during the 1950s and 1960s. In addition to contaminated surface water impoundments and soil, the chemical plant area includes about 40 buildings -- some of which contain asbestos, spill residues (e.g., polychlorinated biphenyls [PCBs]), radon gas, and radioactively and chemically contaminated particulates. In 1989, the EPA included the site in its National Priorities List. Hence, site cleanup is being conducted in accordance with EPA's Superfund process under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA), as amended. Site activities are also being conducted in compliance with the National Environmental Policy Act (NEPA).

An integral part of the CERCLA/NEPA process is the preparation of a comprehensive baseline assessment of human health and environmental impacts to support decision making for site cleanup. This paper focuses on one component of the baseline assessment prepared for the site in order to highlight the potential effects on exposure estimates that result from varying the exposure assumptions. This component is the characterization of human health risks associated with potential exposures to PCBs in one of the contaminated buildings. Because of the heterogeneous nature of the PCB contamination, a stochastic approach was used to account for the variability in possible exposures. This paper describes the mathematical model that was used to quantify human exposures and risks associated with ingestion, inhalation, and dermal absorption of PCBs in air, dust, and spills on floors and benches in the building. The model explicitly accounts for contaminant heterogeneity and simulates the movement of a person through several microenvironments within the building. By this approach, data on human activity patterns relative to the time spent in a particular microenvironment can be combined with contaminant concentrations to calculate personal exposure profiles and potential risks.

METHODOLOGY

The site considered in this paper is located in a rural area surrounded by land owned by the federal and state government, including wildlife areas that total over 14,000 acres. The site is fenced, and security guards are posted such that access by the general public is restricted. Nevertheless, potential exposures of the general public are evaluated by assuming that a hypothetical trespasser, assumed to be an adolescent, enters the building and is exposed either while walking or playing. The potential routes of exposure are inhalation of airborne particulates, incidental ingestion of residues and dust, and dermal contact with residues and dust.

Exposure Point Concentrations

The contaminated building is a 60-m by 110-m, one-story cinderblock structure that is divided in two by a wall that extends from north to south. The west half of the building is subdivided into small work bays and offices; the east half contains a main storage area and several smaller rooms that formerly housed an automotive repair shop, rest rooms, decontamination areas, and a shipping dock. Sampling for PCBs was conducted using two methods: (1) swipe sampling of floor and bench surfaces and (2) bulk sampling of spill residues and dusts. The biased sampling strategy focused on areas where PCBs were expected to be found. Concentrations in 22 surface swipe samples ranged from about 2 to > 29,000 $\mu g/100\ cm^2$; however, almost 40% of the samples were below the analytical detection limit (DL) of 1 $\mu g/100\ cm^2$. Concentrations in 11 bulk samples ranged from about 40 to 13,000 ppm, with 15% below the DL of 2 to 5 ppm. About 85% of the building was determined to be uncontaminated with PCBs.

Exposure point concentrations were estimated from these data by developing a strategy to address the non-detects (NDs), i.e., samples for which PCB concentrations were below the DL. Several statistical methods have recently been recommended for substituting values for the NDs,[6-8] and these methods have been evaluated for application to soil data from the site by Özkaynak et al.[9] (a companion paper being presented at this conference, Paper No. 91-119.7). From this evaluation, it was determined that the maximum likelihood estimator method was suitable for most data sets for which the NDs ranged from a few percent to 50%. However, we found that, in this case, replacing the NDs with one-half the DL for the building PCB data was adequate and did not introduce much bias in estimating the exposure point concentrations. This was largely due to the highly skewed distribution of PCB concentrations in the building samples. Because the PCB contamination is limited to several discrete areas within the building, the measured value at each of these areas was used as a location-specific exposure point concentration.

No air samples were taken for PCBs, thus, a dust resuspension model[10] was used to estimate PCB concentrations in air that could result from the resuspension of contaminated residue and dust during human activity in the building. Airborne concentrations of PCBs were estimated for each contaminated area as follows:

$$C_{air} = \frac{C_{sur}\ \Omega\ A}{V\ a} \qquad (1)$$

where C_{air} is the PCB concentration in air (mg/m^3); C_{sur} is the surface contamination level (mg/m^2); Ω is the fraction of dust resuspended per hour (h^{-1}); A is the area of contamination within a room (m^2); V is the volume of the room (m^3); and a is the number of air exchanges per hour (h^{-1}).

Intake Equations

Human intakes of PCBs via various pathways -- that is, PCB exposures normalized for time and body weights -- were estimated consistent with EPA guidance.[11] Intakes resulting from exposures via the air pathway were estimated as follows:

$$I_{ihl} = \frac{(C_{air})\ (IR_{ihl})\ (PM_{10})\ (ET)\ (EF)\ (ED)}{(BW)\ (AT)} \quad (2)$$

where I_{ihl} is the normalized intake for inhalation of resuspended contaminated dust (mg/kg-d); IR_{ihl} is the inhalation rate (m^3/h); PM_{10} is the fraction of resuspended dust with an aerodynamic mean diameter of less than 10 μm; ET is the exposure time (h/event); EF is the exposure frequency (events/yr); ED is the exposure duration (yr); BW is the body weight (kg); and AT is the averaging time (d).

Because the extent of PCB contamination was characterized by both bulk sampling and surface swipe methods, two algorithms were needed to estimate intakes resulting from incidental ingestion of contaminated residues. For data reported in μg/g (i.e., bulk sample data), intakes were estimated following EPA guidance:[11]

$$I_{ing} = \frac{(C_{rsd})\ (IR_{rsd})\ (FI_{rsd})\ (ET)\ (EF)\ (ED)\ (CF_1)}{(BW)\ (AT)} \quad (3)$$

where I_{ing} is the intake for incidental ingestion of residue (mg/kg-d); C_{rsd} is the PCB concentration of the residue (mg/kg); IR_{rsd} is the residue ingestion rate (mg/h); FI_{rsd} is the fraction ingested from the contaminated residue; and CF_1 is a unit conversion constant (10^{-6} kg/mg). However, for data reported in μg/100 cm^2 (i.e., surface swipe data), the EPA guidance needs to be modified; for this case, intakes were calculated as follows:

$$I^*_{ing} = \frac{(C_{sur})\ (SA^*)\ (FR)\ (EF)\ (ED)\ (CF_2)}{(BW)\ (AT)} \quad (4)$$

where I^*_{ing} is the intake for incidental ingestion of residue (mg/kg-d); SA^* is the exposed skin surface area that could come in contact with the mouth (cm^2/h); FR is the fraction of contaminant removed during contact; and CF_2 is a unit conversion constant (10^{-4} m^2/cm^2).

Similar to the ingestion pathway, two approaches were used to estimate intakes from skin absorption as a result of dermal contact with contaminated residue and dust. For data reported in μg/g (bulk samples), intakes were estimated following EPA guidance:[11]

$$I_{der} = \frac{(C_{rsd})\ (FI_{rsd})\ (SA)\ (AF)\ (AB)\ (EF)\ (ED)\ (CF_1)}{(BW)\ (AT)} \quad (5)$$

where I_{der} is the intake via dermal absorption (mg/kg-d); SA is the exposed skin surface area (cm^2/event); AF is the residue-to-skin adherence factor (mg/cm^2); and AB is the absorption

factor. However, for data reported in $\mu g/100\ cm^2$ (surface swipes), intakes were estimated as follows:

$$I^*_{der} = \frac{(C_{sur})\ (SA)\ (FR)\ (AB)\ (EF)\ (ED)\ (CF_2)}{(BW)\ (AT)} \qquad (6)$$

where I^*_{der} is the intake via dermal adsorption (mg/kg-d).

Consistent with EPA guidance,[11] health risks are assessed by estimating the reasonable maximum exposure (RME) expected to occur at a site. The RME is estimated for individual pathways. For multiple pathway exposures, health risks are summed across pathways to provide an estimate of total exposure. Scenario-specific assumptions and intake parameters used to estimate the RME are given in Table I. In general, the parameter values selected are the 90th or 95th percentile upper bound value of the parameter cumulative distribution; however, for some parameters, the 50th percentile value is recommended.[11] The input parameters given in Table I have varying degrees of uncertainty associated with them. In addition, those parameters pertaining to exposure frequency and duration are often subjective judgments based on area demographics. Nevertheless, the RME approach ensures that the resultant health risk estimates will be conservative.

Stochastic Approach for Estimating Contaminant Intakes and Risk

The PCB contamination within the building is limited to a few discrete locations that represent only about 15% of the total building area. Because of the highly skewed distribution of exposure point concentrations in the building, a nonstandard approach was used for quantifying possible PCB exposures, in addition to following the standard EPA approach. That is, risks from PCB exposures were first estimated on the basis of both the arithmetic mean of the exposure point concentration and the upper (one-sided) 95% confidence limit on the arithmetic mean of the exposure point concentration (UL_{95}),[21] as recommended by the EPA.[11] The underlying assumption in this approach is that an individual will always come in contact with a contaminated area during each RME event. However, in this specific case, 85% of the building is free of contamination and, thus, a trespasser is more likely to come in contact with an uncontaminated area than a contaminated one.

For comparison, a stochastic model was then developed to explicitly account for the heterogeneous nature of the PCB contamination, as well as the uncertainty in defining the time spent at any specific exposure point. Equations 2 through 6 were implemented with the Monte Carlo spreadsheet program @RISK[22] to simulate the movement of a hypothetical adolescent trespasser through several microenvironments within the contaminated building. In the simulation, it was assumed that the trespasser comes in contact with a discrete area inside the building for one hour during each of five visits per year over a 10-year period. This internal area, that is, the exposure point, was selected on the basis of the probability of entering a given room and the probability of coming in contact with a contaminated area within that room. (Only some of the rooms in the building are contaminated with PCBs, and

Table I Input parameters for PCB exposure assessment.

Variable	Parameter Description	Value	Reference
AT	Averaging time, period over which exposure is averaged	2.56×10^4 d for carcinogens 3.65×10^3 d for noncarcinogens	--
AB	Absorption factor	0.032	12
AF	Residue to skin adherence factor	2.77 mg/cm^2	11
BW	Body weight, adolescent[a]	50 kg	13,14
ED	Exposure duration	10 yr	--
EF	Exposure frequency	5 events/yr	--
ET	Exposure time	1 h/event	--
FI_{rsd}	Fraction ingested from contaminated residue	1	--
FR	Fraction of contaminant removed during dermal contact	0.1	--
IR_{ihl}	Inhalation rate	1.2 m^3/h	15
IR_{rsd}	Residue ingestion rate[b]	10 mg/h	11
a	Number of air exchanges per hour	0.5 h^{-1}	--
Ω	Fraction dust resuspended per hour[c]	10^{-4} h^{-1}	10
SA	Exposed skin surface area[d]	2,800 cm^2/event	13
SA*	Exposed skin surface area that could come in contact with the mouth	475 cm^2/event	--
AE_{orl}	Oral absorption efficiency	0.95	16
SF_{ihl}	Inhalation slope factor	7.7 (mg/kg-d)$^{-1}$	17
SF_{orl}	Oral slope factor	7.7 (mg/kg-d)$^{-1}$	18
SF_{der}	Dermal slope factor[e]	8.7 (mg/kg-d)$^{-1}$	11
RfD_{orl}	Inhalation reference dose	8.6×10^{-7} mg/kg-d	19
RfD_{orl}	Oral reference dose	1×10^{-4} mg/kg-d	20
RfD_{der}	Dermal reference dose[f]	9×10^{-5} mg/kg-d	11

[a] Estimated averge body weight for an adolescent over the 10-year exposure period.
[b] EPA recommended value of 100 mg/d adjusted to account for the fact that a receptor may be present at contaminated area for only a certain portion of the exposure period.
[c] Assumes moderate activity within the building.
[d] Time-weighted average based on estimate of skin surface area and consideration of the type of clothing that would be worn during the exposure event.
[e] $SF_{der} = SF_{orl}/AE_{orl}$.
[f] $RfD_{der} = RfD_{orl}/AE_{orl}$.

not all surfaces in these rooms are contaminated.) The intakes estimated for each of the 50 visits were then summed to give the total intake for the trespasser scenario. A number of 50-visit runs (each of which constituted a single iteration) were combined to form a model simulation. Because cumulative exposure is estimated over a 50-visit scenario, the assumption that the trespasser spends the entire hour of any given visit at one location does not bias the estimate of total exposure. Conceptually, the trespasser could spend $1/50^{th}$ of one hour at each of the 50 locations selected in any iteration of a simulation.

As previously noted, substantial uncertainty can exist for each of the intake parameters given in Table I. Clearly, the assumptions used for these parameters are based on judgment and are inherently uncertain. The total extent of exposure at an exposure point is defined by three of the parameters, exposure time, frequency, and duration. The values selected for the length of each visit to the building and the number of visits per year are considered to be reasonable on the basis of current land use and demographics of the surrounding area. Security at the site reduces the likelihood of more frequent entry and limits the length of time a trespasser could remain on-site. The exposure duration of 10 years is considered reasonable for adolescent trespassing behavior, and it is also consistent with the time interval projected for site cleanup (which is already under way). This paper focuses on uncertainty related to the number of contaminated areas contacted during each visit and the time spent at a specific area during the visit.

The results of an exposure assessment are combined with toxicological information to provide an estimate of excess cancer risk and the potential for noncarcinogenic health effects associated with a site. Carcinogenic risks are assessed in terms of the increased probability that an individual will develop cancer over the course of a lifetime. To estimate excess cancer risk, the intakes calculated with Equations 2 through 6 are multiplied by chemical-specific slope factors. Oral and inhalation slope factors for several Group A, B1, and B2 carcinogens have been derived by EPA and represent the lifetime cancer risk per milligram of contaminant intake per kilogram body weight, assuming that the exposure occurs over a 70-year lifetime. With some qualification (depending on available toxicological data), dermal slope factors can be calculated by dividing the oral slope factor by the oral absorption efficiency.[11] For Superfund sites the EPA has identified incremental lifetime cancer risks in the range of 10^{-4} to 10^{-6} (and lower) to be "acceptably protective".[23]

The measure used to describe the potential for the occurrence of noncarcinogenic effects in an individual is not expressed in probabilistic terms. Rather, these effects are evaluated by dividing the pathway-specific intake over a specified period of time by the EPA-established reference dose (RfD) for a similar exposure period. This ratio is termed the "hazard quotient" for a contaminant. For multiple chemical exposures and/or pathways, the individual hazard quotients are summed to determine an overall "hazard index". This hazard index approach could, however, overestimate the potential for adverse health effects for compounds that affect different target organs, do not induce the same type of effect, or do not act by the same mechanism. Thus, this method is only used for screening purposes. Chronic and subchronic RfDs for the oral and inhalation routes have been derived by the EPA for several compounds. Dermal RfDs can be calculated, again with qualification, by

multiplying the oral RfD and the oral absorption efficiency.[11] A hazard index of less than one has been identified by EPA as being "acceptably protective" for Superfund sites.[11,22] Route-specific slope factors and RfDs used in this analysis are given in Table I.

RESULTS

For 100 separate simulations of 500 iterations, the incremental cancer risk estimated from the mean exposure estimate of each simulation ranged from 4×10^{-6} to 8×10^{-5}; the mean of the 100 simulations was 1×10^{-5}. The risk calculated from the arithmetic mean of the exposure point concentration was 1×10^{-4}, an order of magnitude higher than that determined by the stochastic approach. The 95% upper-bound value of the incremental cancer risk estimated from the cumulative distributions ranged from 2×10^{-6} to 1×10^{-4}, and the mean upper-bound value for the 100 simulations was 9×10^{-5}. The risk calculated using the UL_{95} was 4×10^{-4}, a factor of 4 higher than that determined from the average 95% upper-bound value predicted the 100 simulations.

For the noncarcinogenic hazard index, the mean estimated from exposure estimate of each simulation ranged from 0.4 to 1; the mean of the 100 simulations was 0.7. However, the hazard index calculated from the arithmetic mean of the exposure point concentration was about 1. The 95% upper-bound value of the hazard index from the cumulative distributions ranged from about 0.2 to 10 and the mean value for the 100 simulations was about 9. The hazard index calculated using the UL_{95} was 4, which is less than half the value obtained using the average 95% upper-bound value predicted from the 100 separate simulations.

Although the values for incremental cancer risk and hazard index estimated from the arithmetic means or the 95% upper-bound values from either the nonstandard stochastic approach or the standard EPA approach are within a factor of 10, it is obviously useful to characterize the entire distribution. Figure 1 shows a typical example of a cumulative distribution plot for the incremental cancer risk from one simulation. In this case (and in others examined), the distribution of risk is highly skewed because of contaminant heterogeneity. The risks estimated from the upper-bound values in the 90 to 99% range are very sensitive to the percentile selected. For the case shown in Figure 1, the 95% upper-bound value of risk from the distribution is 1×10^{-4} whereas the 94% upper-bound value is 3×10^{-6}. That is, the estimate of risk decreased by a factor of about 30, with a 1% decrease in the cumulative percent at which the risk was evaluated. The results were similar for other simulation runs.

Figure 2 presents results for 10 of the 100 simulation runs for estimating incremental cancer risk. In this example, the estimated mean values of the distributions from each simulation are relatively stable, with a coefficient of variation (CV) of about 30%. However, significant fluctuations can occur in the estimate of the 95% upper-bound value of the distribution, with the CV increasing to about 70% because of the highly skewed data set. Increasing the number of simulations from 10 to 50 or 100 can significantly reduce the CV to about 15% for the mean and about 30% for the 95% upper-bound value.

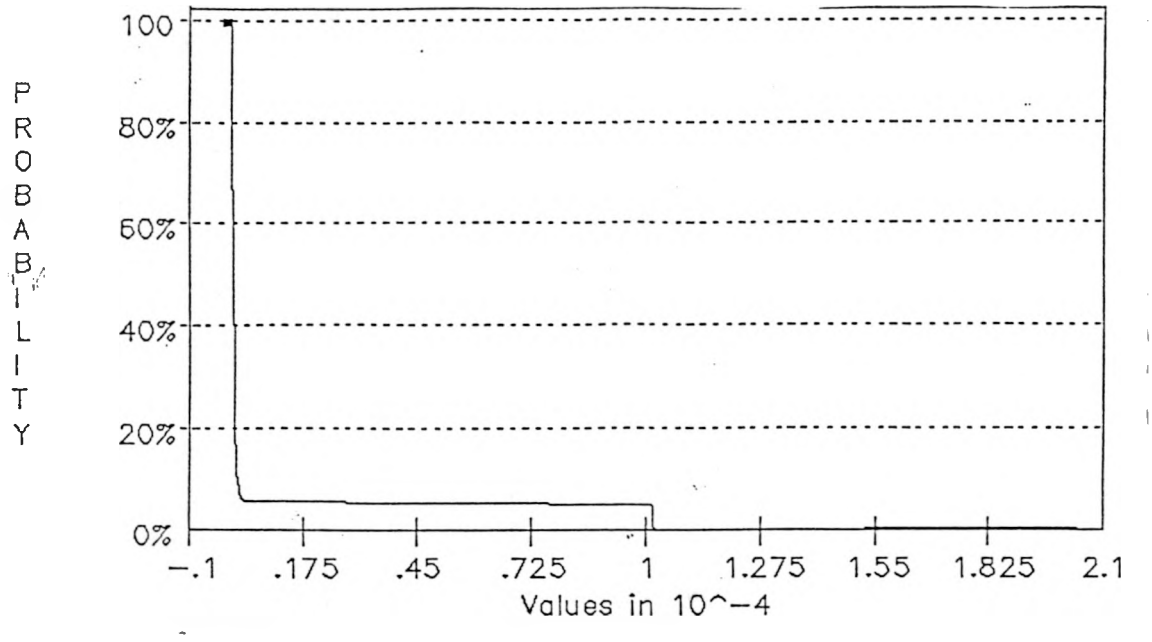

Figure 1. Cumulative distribution plot of incremental cancer risk from one 500-iteration simulation run.

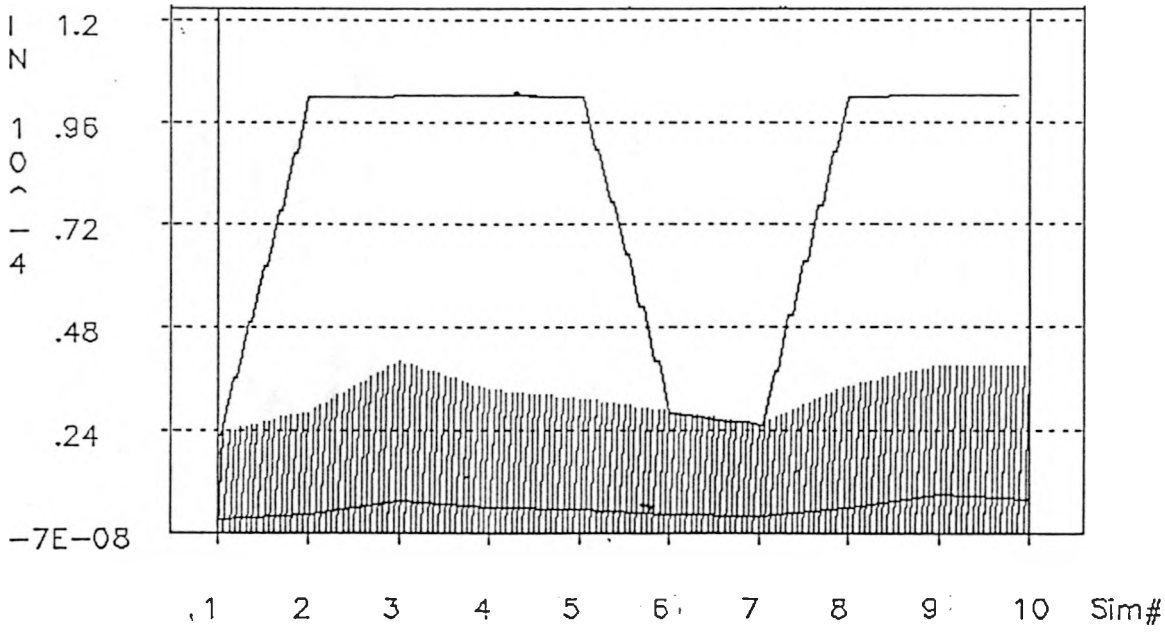

Figure 2. Incremental cancer risk predicted from ten 500-iteration simulation runs.

CONCLUSIONS

From this analysis, we conclude that simulation methods can be very helpful in understanding potential variability in risk estimates when dealing with stochastic processes that are complicated and involve data that are not normally distributed. This is especially true when the exposure scenarios that are being modeled involve a relatively small number of potential encounters with source(s) of contamination; in such cases, using the central limit theorem and relying on the analytical solutions may be inappropriate. Moreover, numerical solutions, like those presented here, offer the advantage of readily incorporating parameter uncertainties, varying model inputs, and explicitly characterizing the nature (stability) of the various percentiles that may be considered to predict health risks. Our analysis did not indicate a substantial difference between the 95% upper-bound value of the predicted risk distribution and the simpler, EPA-recommended approach for calculating the RME risks, which is based on the upper (one-sided) 95% confidence limit of the arithmetic average contaminant concentration. However, the uncertainties associated with both of these estimates were found to be quite significant.

By using the stochastic model and implementing Monte-Carlo techniques, we were able to estimate (and iteratively reduce) the magnitude of these prediction errors and to show that they were due primarily to the unique exposure conditions that exist inside the site buildings. We note, however, that such widely differing conditions of contamination and potential exposures are not that unusual. That is, contaminant concentrations in other media (e.g., water, soil, food, and game) and the potential for human contact with contaminants in these media also typically require dealing with data that are widely and nonuniformly distributed. As demonstrated by the application presented in this paper, we recommend that stochastic methods be applied to predict the full distribution of health risks and associated uncertainties for population groups exposed to contaminants near a Superfund site. By this approach, risk management decisions could be determined from a broader information base than the standard approach may provide. For example, decisions based only on the UL_{95} may overstate site risks and lead to less cost-effective use of the limited remedial action funds available. Alternatively, as in the case presented for the hazard index, the EPA recommended methodology may provide a less conservative estimate of the potential risks than the stochastic approach. Even if the cleanup decision is not changed as a result of considering stochastic methods, it is important for the decision maker to be provided with such information to support the discussion of the conservative nature of this decision.

ACKNOWLEDGMENTS

The authors would like to express their appreciation to Lynne A. Haroun, the Argonne National Laboratory Project Leader for the site risk assessment, for her direction on the comprehensive baseline assessment and helpful comments on this manuscript. The work was supported, in part, by the U.S. Department of Energy, Office of Environmental Restoration and Waste Management, under Contract W-31-109-Eng-38.

REFERENCES

1. National Research Council, Epidemiology and Air Pollution, National Academy Press, Washington, D.C., 1985.

2. National Research Council, Human Exposure Assessment, National Academy Press, Washington, D.C., 1991.

3. W. Ott, L. Wallace, D. Mage, G. Akland, R. Lewis, H. Sauls, C. Rodes, D. Kleffman, D. Kuroda, and K. Morehouse, "The Environmental Protection Agency's research program on total human exposure," Environ. Int. 12:475 (1986).

4. W.R. Ott, "Total human exposure: Basic concepts, EPA field studies, and future research needs," J. Air Waste Manage. Assoc. 40(7):966 (1990).

5. L.A. Wallace, E.D. Pellizzari, T.D. Hartwell, R. Whitmore, and C. Sparacino, "Total exposure assessment methodology (TEAM) study: Personal exposures, indoor-outdoor relationships, and breath levels of volatile organic compounds in New Jersey," Environ. Int. 12:369 (1986).

6. M.A. Cohen and P.B. Ryan, "Observations less than the analytical limit of detection: A new approach," J. Air Pollut. Control Assoc. 39(3):328 (1989).

7. C.N. Haas and P.A. Scheff, "Estimation of averages in truncated samples," Environ. Sci. Technol. 24(6):912 (1990).

8. C.C. Travis and M.L. Land, "Estimating the mean of data sets with nondetectable values," Environ. Sci. Technol. 24(7):961 (1990).

9. H. Özkaynak, J. Xue, D.A. Butler, L.A. Haroun, M.M. MacDonell, and D.J. Fingleton, "Addressing data heterogeneity: Lessons learned from a multimedia risk assessment," Paper No. 91-119.7, presented at the 84th Annual Meeting of the Air and Waste Management Association, Vancouver, British Columbia (1991).

10. J.W. Healy, Surface Contamination: Decision Levels, LA-4558-MS, Los Alamos Scientific Laboratory, Los Alamos, 1971, pp 30-34.

11. U.S. Environmental Protection Agency, Risk Assessment Guidance for Superfund, Volume I: Human Health Evaluation Manual (Part A), EPA/540/1-89/002, Office of Emergency and Remedial Response, U.S. Environmental Protection Agency, Washington, D.C., 1989.

12. U.S. Environmental Protection Agency, Development of Advisory Levels for Polychlorinated Biphenyls (PCBs) Cleanup, EPA/600/6-86/002, Office of Health and Environmental Assessment, Washington, D.C., 1986.

13. U.S. Environmental Protection Agency, Exposures Factor Handbook, EPA/600/8-89-043, Office of Health and Environmental Assessment, Washington, D.C., 1989.

14. U.S. Environmental Protection Agency, Development of Statistical Distributions or Ranges of Standard Factors Used in Exposure Assessments, Final Report, EPA\600\8-85-010, Office of Health and Environmental Assessment, Washington, D.C., 1985.

15. International Commission on Radiological Protection, Report of the Task Group on Reference Man, ICRP Publ. 23, prepared by a Task Group of Committee 2, 1975.

16. B.A. Owen, "Literature-derived absorption coefficients for 39 chemicals via oral and inhalation routes of exposure," Regul. Tox. and Pharm. 11:237 (1990).

17. California Air Pollution Control Officiers Association, Air Toxics "Hot Spots" Program, Risk Assessment Committee, California Air Pollution Control Officers Association, Sacremento, 1990.

18. U.S. Environmental Protection Agency, Integrated Risk Information System (IRIS): Online, U.S. Environmental Protection Agency, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, December 1990.

19. Commonwealth of Massachusetts, The Chemical Health Effects Assessment Methodology and the Method to Derive Allowable Ambient Limits, Commonwealth of Massachusetts, Department of Environmental Quality Engineering, Boston, June 1989 (Draft).

20. P.F. Hurst, U.S. Environmental Protection Agency, Office of Research and Development, Environmental Criteria and Assessment Office, Cincinnati, personal communication to D. Wall, U.S. Environmental Protection Agency, Region VII, August 29, 1990.

21. R.O. Gilbert, Statistical Methods for Environmental Pollution Monitoring, Van Nostrand Reinhold, New York, 1987, pp 132-151.

22. Palisade Corporation, @RISK: Risk Analysis and Modeling for the PC, Palisade Corporation, Newfield, New York, 1989.

23. U.S. Environmental Protection Agency, "National Oil and Hazardous Substances Pollution Contingency Plan; Final Rule (40 CFR Part 300)," Fed. Regist., 55(46): 8666-8865 (March 8, 1990).