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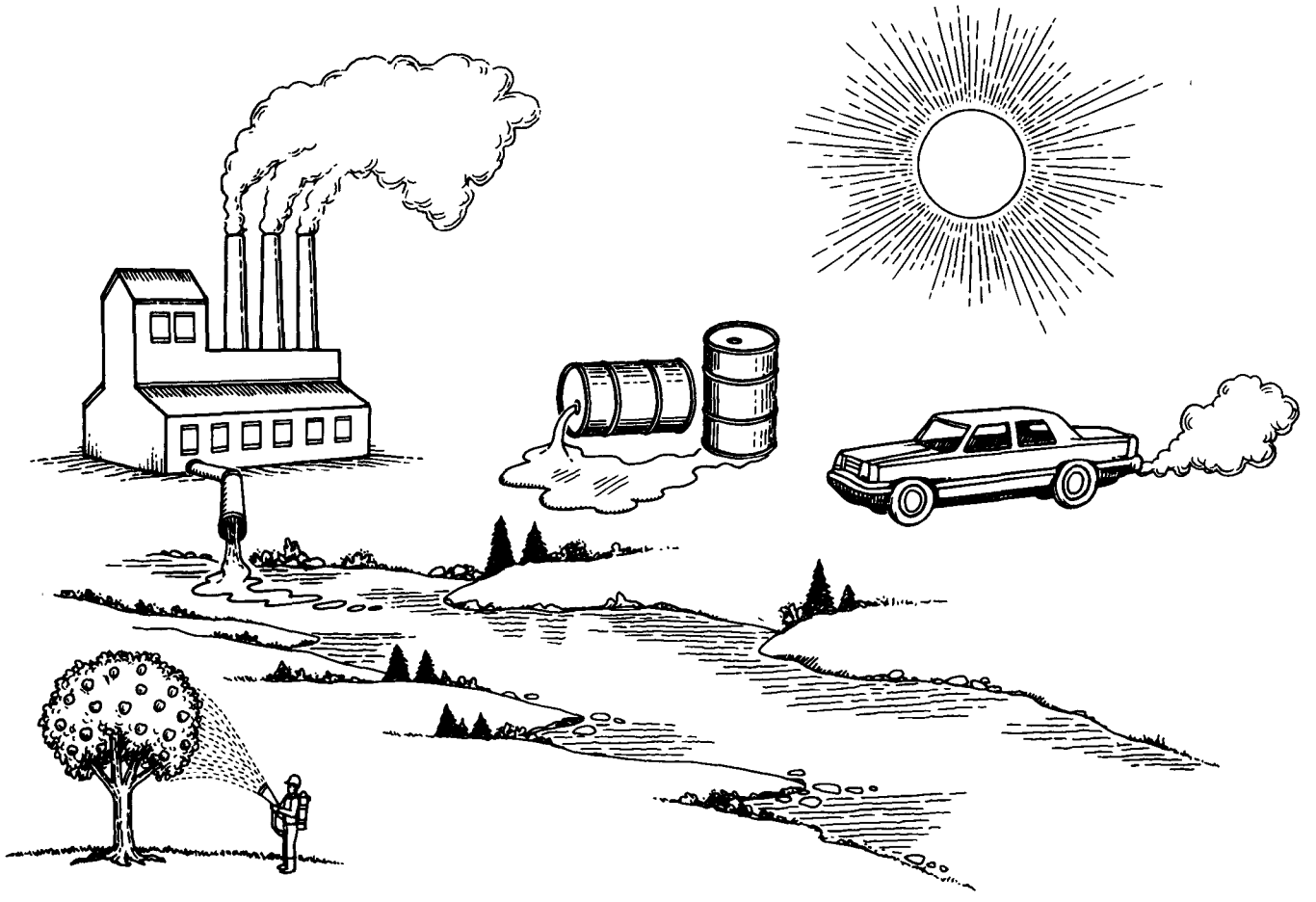
Unfinished Business: A Comparative Assessment of Environmental Problems

Appendix I
Report of the Cancer Risk Work Group



COMPARATIVE RISK PROJECT

REPORT OF THE CANCER WORK GROUP

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INTRODUCTION

The Cancer Work Group was directed to evaluate the cancer risk associated with 31 environmental problem areas under EPA's jurisdiction, and then to rank them according to the relative magnitude of their cancer risk. The work group was composed of senior managers and technical staff representing all Agency program offices (Air and Radiation, Pesticides and Toxic Substances, Solid Waste and Emergency Response, and Water). Individuals were chosen largely on the basis of their familiarity with cancer risk assessment methods, particularly with respect to such analyses undertaken within their programs. In addition to the program offices, the Office of Research and Development, Office of Policy, Planning and Evaluation, and the Regional Offices were represented on the work group.

This report discusses the process we used for ranking the environmental problems, identifies the difficulties we had in evaluating cancer risks, and presents the relative rankings. It also highlights several issues that affected the ranking of specific environmental problems. The final section of the report provides some observations on the difficulty of evaluating and ranking cancer risk. Appendix A presents further detail on the methods used to estimate cancer risks, and Appendix B presents quantitative estimates of cancer incidence and individual risks for each environmental problem for which such estimates exist.

The ranking should not be regarded as a list establishing priorities for regulation. Our assessment was limited to only one dimension -- that of cancer risk. We did not evaluate a host of factors important in regulatory decision making, such as how extensively risks can be reduced through regulation, the cost of reducing cancer risk, EPA's statutory authority, and the extent of ecological, welfare, and non-cancer health effects. In addition, because available data were often limited and did not allow for comparison between problems, we relied heavily on our professional judgment, rather than on quantitative methods. Nonetheless, the information we developed will be useful in setting EPA's priorities.

THE RANKING PROCESS

We did not conduct new research as a part of this effort. Instead, we extracted information from risk assessment work performed in support of other regulatory activities.

During the first phase of this project, we compiled information on the cancer risks of each environmental problem. In general, this information was based on EPA risk assessments performed in support of specific regulatory activities. We presented the information on the cancer risks of each environmental problem in the form of "data sheets," which indicated the substances examined and the extent of exposure. In most cases, these assessments used cancer potency estimates developed by EPA's Carcinogen Assessment Group (CAG), adhering to the methods outlined in EPA's Guidelines for Carcinogen Risk Assessment (51 FR 33992). Exposure data were generated either by using exposure models or by extrapolating from monitored data. (Appendix A of this report discusses these methods in detail.)

After a detailed review of the data sheets, we met to discuss the information on cancer risks, to review the methods program offices used to estimate the cancer risks, and to rank the environmental problems as to the relative magnitude of their risks. We first placed the 31 environmental problem areas into five categories. In addition, the work group chose not to rank two environmental problems to avoid counting the same risks twice. After reaching a consensus on this grouping of the problems, we ranked each problem ordinally by comparing the environmental problems within each category.

As a starting point for our rankings, we relied on the information presented in the data sheets. It soon became clear that we could not base our rankings solely on this information. For example, for some problem areas, only a few chemical assessments were compiled, while for others, many were compiled. This led to large differences in how well the problem areas were quantified. At best, for some problems, most of the toxicologically well-characterized chemicals were covered. Thus, the best coverage still could not consider the large number of substances that have insufficient toxicity data. For other problems, only one or two chemical assessments were compiled -- more as examples of the potential extent of the risk from the problem area than as attempts at quantification.

In addition, the methods and assumptions used to estimate exposure and risk were not consistent across environmental problem areas. This introduced a potential bias in comparing risks. The degree of uncertainty about estimates of cancer incidence varied considerably among environmental problems. Finally, the methods we used to develop national estimates of cancer incidence from available data were not always comparable, nor was it possible to make them so. As a result, we considered these qualitative factors in our deliberations on the final ranking.

After this meeting, the ranking results were circulated for the work group to review and to reach a consensus on. In addition, work group members prepared summaries of the data sheets for each environmental problem, and then circulated

them to the other members for review. These appear in Appendix B of this report.

As a final step, we reevaluated our rankings and discussed the observations that appear in this report.

RELATIVE RANKING OF ENVIRONMENTAL PROBLEMS

Table 1 displays the results of the relative ranking of environmental problem areas on the basis of cancer risk. Problem areas have been placed into five categories of decreasing magnitude, with the fifth category containing problem areas for which no risk was identified. In addition, problem areas were ranked numerically within each category. The second column indicates the substances or sources of exposure that are considered in the quantitative analyses for each environmental problem, as presented in Appendix B. The third column summarizes the rationale for the individual rankings and other comments not directly related to the ranking itself (such as the presence of particularly high individual risks).

TABLE 1

Consensus Ranking of Environmental Problem Areas
On the Basis of Population Cancer Risk*

CATEGORY 1

RANK	PROBLEM AREA	SUBSTANCES/ EXPOSURES INVESTIGATED	COMMENTS
1 (tied)	Worker Exposure to Chemicals	Formaldehyde Tetrachloroethylene Asbestos Methylene chloride	Ranked highest of any single environmental problem, along with Indoor Radon -- based on work group consensus. About 250 cancer cases were estimated annually from four substances, but workers face potential exposures to over 20,000 substances. Individual risk can be very high.
1 (tied)	Indoor Radon	Radon and its decay products	Also ranked the highest. Current estimates are 5,000 to 20,000 lung cancers annually from exposures within homes. Some of these are a consequence of the joint action of radon and tobacco smoke. Individual risks can be very high.
3	Pesticide Residues on Foods	1 Herbicide 3 Fungicides 1 Insecticide 1 Growth regulator	Cancer incidence estimate of about 6,000 annually, based on exposure to 200 potential oncogens (one-third of total pesticides in use) -- extrapolated from seven known oncogens. Assessment does not account for so-called inert materials in pesticides.
4 (tied)	Indoor Air Pollutants Other than Radon	Tobacco smoke Benzene p-Dichlorobenzene Chloroform Carbon tetrachloride Tetrachloroethylene Trichloroethylene	Quantitative assessment estimates 3,500-6,500 cancers annually. Environmental tobacco smoke is responsible for the majority. Risks from organics estimated on the basis of monitoring 600 U.S. homes. Individual risks can be very high. Potential for some double counting with Consumer Exposure to Chemicals and with Drinking Water.

* The five categories represent decreasing magnitude of cancer risk, with Category 1 representing problem areas with the highest relative risk, and Category 5 representing problem areas for which no cancer risk has been identified. Problems are also ranked numerically within each category.

RANK	PROBLEM AREA	SUBSTANCES/ EXPOSURES INVESTIGATED	COMMENTS
4 (tied)	Consumer Exposure to Chemicals	Formaldehyde Methylene chloride p-Dichlorobenzene Asbestos	The risk from these four chemicals is about 100-135 cancers annually. There are an estimated 10,000 chemicals in consumer products. Even though exposures are generally intermittent, risks are believed to be high, given the concentrations to which individuals are exposed. Consumers are exposed through such products as cleaning fluids, pesticides, particleboard and other building materials, and numerous asbestos-containing products. Considerable double counting with Indoor Air and Other Pesticide Risks.
6	Hazardous/ Toxic Air Pollutants	20 substances, classes of substances, or waste streams	A quantitative assessment of 20 substances estimates approximately 2,000 cancer cases annually. This is a subset of the large total number of pollutants to which people are exposed in ambient air. Individual risks can be very high. Potential for some double counting with Active Hazardous Waste Sites Municipal Hazardous Waste Sites, and Contaminated Sludge.
	Category 2		
7	Depletion of Stratospheric Ozone	Increased UV radiation (Chlorofluorocarbons, Halon 1301, Chlorocarbons)	Current nonmelanoma and melanoma skin cancer deaths at 10,000 annually. Ozone depletion projected to result in steadily increasing risks, with an additional 10,000 annual deaths projected for the year 2100. This problem is ranked in Category 2 because of the considerable uncertainties concerning estimates of future risk. If estimates are correct, would rank higher. Needs further research.
8	Hazardous Waste Sites - Inactive	Trichloroethylene Vinyl chloride Arsenic Tetrachloroethylene Benzene 1,2-Dichloroethane	Nationwide cancer incidence from six chemicals estimated at just over 1,000 annually. Considerable uncertainty, since nationwide risk estimates are based on extrapolating from 35 sites to about 25,000 sites nationwide. Individual risks can be very high. Potential for some double counting with Drinking Water.

RANK	PROBLEM AREA	SUBSTANCES/ EXPOSURES INVESTIGATED	COMMENTS
9	Drinking Water	Ingestion and/or inhalation of 23 substances	Quantitative assessment estimates about 400-1,000 cancer cases annually, based on home surveys of public water systems. Most cases are from radon and trihalomethanes. Potential for some double counting with Indoor Radon, Indoor Air Pollution, and several categories related to contaminated ground water.
10	Application of Pesticides	1 Herbicide 3 Fungicides 1 Insecticide 1 Growth regulator	Approximately 100 cancers annually estimated by a method analogous to that used for Pesticide Residues on Food. Small population exposed, but uniformly high individual risks.
11	Radiation Other than Indoor Radon	Occupational exposures Consumer products Industrial emissions	Risks associated with medical exposures and natural background levels excluded; would rank higher if these were included. Two-thirds of assessed risk of 360 annual cancers results from building materials. Individual risks can be very high. Nonionizing radiation not considered due to lack of data.
12	Other Pesticide Risks	Consumer use Professional exterminator use	Few quantitative estimates available. Consensus estimate of 150 cancers annually, based loosely on discussion of termiticide risks. Less data here than for other pesticide areas.
13	Hazardous Waste Sites - Active	Several carcinogens from each of the following: Hazardous waste storage tanks Hazardous waste in boilers/ furnaces Hazardous waste incineration Waste oil	No nationwide risk estimates are available, but probably fewer than 100 cases annually. Risk estimates are sensitive to assumptions regarding the proximity of future wells to waste sites. Solid waste management units were excluded from analysis. Individual risks can be very high. Possible double counting with Drinking Water and Hazardous/Toxic Air Pollutants.

RANK	PROBLEM AREA	SUBSTANCES/ EXPOSURES INVESTIGATED	COMMENTS
14	Nonhazardous Waste Sites - Industrial	Arsenic 1,1,2,2-Tetrachloroethane Chloroform Benzene	No analysis of cancer incidence. Instead, based on the consensus of the work group. Judged less severe than hazardous waste, worse than municipal. Potential for some double counting with Drinking Water.
15	New Toxic Chemicals	None	Very difficult to assess future uses of new chemicals and the risks of using chemicals never manufactured. Consensus was that this problem poses moderate risks.
	Category 3		
16	Nonhazardous Waste Sites - Municipal	Several pollutants/waste streams from the following: Municipal landfills Municipal sludge incineration Municipal waste incineration	Quantitative estimate of about 40 cancers annually. This estimate does not include risks from municipal surface impoundments. Potential for some double counting with Hazardous/Toxic Air Pollutants, Contaminated Sludge, and Drinking Water.
17	Contaminated Sludge	Up to 22 carcinogens from the following: Land application Distribution and marketing Landfilling Incineration Ocean disposal	Analyses and regulatory development are ongoing. Preliminary results estimate approximately 40 cancers annually. Most of this risk comes from incineration and landfilling. Potential for some double counting with Hazardous/Toxic Air Pollutants, Nonpoint Source Discharges to Surface Water, and Nonhazardous Waste Sites - Municipal.

RANK	PROBLEM AREA	SUBSTANCES/ EXPOSURES INVESTIGATED	COMMENTS
18	Mining Waste	Arsenic Cadmium	Estimate of 10-20 cancer cases annually largely due to arsenic. Severity of problem is relatively low because remote locations expose a relatively small population. This assessment excludes oil and gas operations. Individual risks can be very high. Potential for double counting with Drinking Water.
19	Releases from Storage Tanks	Benzene	Preliminary analysis suggests relatively low cancer incidence (< 1 annually), but exposure modeling not as conservative as several other solid waste problems (behavior that limits exposure is assumed). Potential for double counting with Drinking Water.
20	Nonpoint Source Discharges to Surface Water	None	Judged to be more serious than other surface water categories, but no quantitative analysis is available.
21	Other Ground-Water Contamination	Methylene chloride from septic systems	Generally, risks from other ground-water contamination are not estimated due to a lack of information with respect to sources, their locations, and concentration levels. Individual risks generally less than 10^{-6}, with rough estimate of population risk well under 1 case per year. However, this is an estimate of a small portion of total risk, as we examined one chemical at just one of many sources (septic systems). Potential for some double counting with Drinking Water.
22	Criteria Air Pollutants	Lead Ozone Particulate matter Nitrogen oxides Sulfur oxides Carbon monoxide	This assessment excludes carcinogenic particles and VOCs (controlled to reduce ambient ozone), which are considered under Hazardous/Toxic Air Pollutants. Ranked relatively low because none of the criteria pollutants has been adequately shown to cause cancer. If any are shown to be carcinogenic (e.g., lead), or if VOCs and carcinogenic particles are included in the definition of Criteria Air Pollutants, this problem would move to a higher category.

Category 4

RANK	PROBLEM AREA	SUBSTANCES/ EXPOSURES INVESTIGATED	COMMENTS
23	Direct Point Source Discharges to Surface Water	None	No quantitative assessment is available. Only ingestion of contaminated seafood was considered, since the impact of drinking water was covered elsewhere.
24	Indirect, Point Source Discharges to Surface Water	None	Same as above.
25	Accidental Releases - Toxics	None	Because of the short duration of personal exposure to accidental releases, cancer risk judged to be very small. Long-term effects on ground-water exposures were not considered here. Non-cancer health effects are of much greater concern. Nature of substances and exposures ranks this problem above oil spills.
26	Accidental Releases - Oil Spills	None	See above. Oil spills will be of greater concern for welfare and ecological effects.

|Category 5| (Listed Alphabetically)

PROBLEM AREA	SUBSTANCES/ EXPOSURES INVESTIGATED	COMMENTS
Biotechnology	None	Dilemma of ranking this problem is similar to that for new chemicals, but even less information is available. No known instances of carcinogenic bioengineered substances.
CO_2 and Global Warming	None	Cancer is not considered a significant aspect of this environmental problem. No assessment was undertaken.
Other Air Pollutants	None	By definition, carcinogenic pollutants in the outdoor air are considered under Hazardous/Toxic Air Pollutants. Therefore, no cancer risk was assessed here.

|Not Ranked|

Discharges to Estuaries, Cosatal Waters and Oceans	None	This category represents a conglomeration of other categories. The work group chose not to rank it to to minimize double counting.
Discharges to Wetlands	None	See Discharges to Estuaries, Coastal Waters, and Oceans, above.

GENERAL DIFFICULTIES WITH THE RANKING PROCESS

Generating a relative ranking of environmental problem areas would have been a simple quantitative exercise if we could have obtained consistent methods and complete information for all of the problem areas. And even though the 1986 Guidelines for Carcinogen Risk Assessment were helpful in establishing a consistent set of ground rules, the quality, consistency, and completeness of information on the different environmental problems was highly variable. The risk assessment guidelines warn that estimates of cancer risk generated from those methods are not indicative of true risk. Estimates of true risk are currently not possible, given our limited knowledge of carcinogenesis.

Quantitative Estimates Were Based on Existing Analyses

The quantitative risk estimates we compiled for this project were adapted from analyses previously undertaken by EPA program offices. In most cases, we developed these estimates only to consider the relative merits of different regulatory options. While uncertainties in such an analysis are often great in the context of a well-defined regulatory objective, they become much greater when comparisons are made between the outcomes of diverse analyses.

Ranking Reflects Population Risk Rather Than Individual Risk

Another area of judgment involved the consideration of population versus individual risks. In general, regulation of environmental problems may be warranted in either of two situations. On one hand, exposure of large numbers of individuals to a relatively small cancer risk may result in an unacceptable number of "expected" cancers associated with an environmental problem (high population risks). On the other hand, very high excess cancer risk to a few individuals (high individual risks) may also prove unacceptable, even if the expected number of cases is small.

For the purposes of this report, the rankings of environmental problems were based primarily on population risk. However, where problems pose particularly high risks to individuals, we highlighted this fact. Unfortunately, estimating the size of the population exposed to these high risks was beyond the scope of this project.

Ordinal Ranking May Confer False Accuracy

Several work group members were very uneasy about ranking each of the environmental problems ordinally. They believe that this approach would confer a false degree of accuracy on the ranking when, in fact, the differences between two or three adjacently ranked problems might be impossible to discern. Alternatively, other work group members saw the ordinal ranking

as desirable, so long as the high degree of uncertainty in the process was clearly stated. The work group agreed to present an ordinal ranking, with problem areas organized into five general categories according to the magnitude of population risk.

Comparative Ranking Is Based on Work Group Judgment

Because of the many uncertainties associated with developing a quantitative risk assessment for each of the 31 environmental problems, it was impossible to conduct a highly quantitative analysis with a great deal of confidence. We relied extensively on our collective professional judgment to rank the problem areas and used the quantitative results presented by each program office to supplement this judgment. Therefore, the ranking relies primarily on the opinion of well-informed EPA professionals. Indeed, while we believe that the ranking would not change dramatically, a work group composed of different members may well have arrived at somewhat different conclusions. Thus, this ranking should be interpreted cautiously, taking into consideration the constraints the data base imposes on this project.

DIFFICULTIES IN RANKING PROBLEM AREAS

For various reasons, ranking the problem areas was very difficult. For example, data were lacking in many areas, risks were not identifiable for some areas, and variations in coverage, analytical methods, and levels of confidence complicated the ranking.

Ranking May Reflect Inadequate Data

The information compiled for this project has been taken from cancer risk assessments previously conducted by EPA. Thus, we do not present quantitative information for several problems that are of primary interest to EPA because of factors other than cancer. The consensus of the work group was that these environmental problems pose relatively low cancer risks. However, this assessment may be biased by a lack of quantitative information.

For Some Problems, Risks Were Unidentifiable

We could not identify cancer risk for three environmental problems: Other Air Pollutants, CO_2 and Global Warming, and Biotechnology. We have not considered Other Air Pollutants in this analysis because we assumed that the risk associated with carcinogenic air pollutants, by definition, would be captured under Hazardous/Toxic Air Pollutants. And, though CO_2 and Global Warming may have large ecological and welfare effects, no mechanism by which this problem may increase cancer incidences is known. Finally, there are no data to indicate that cancer risks are associated with biotechnology.

Variations in Coverage Complicated Ranking

The extent to which the available quantitative information estimated total cancer risk varied widely among environmental problems. In general, incomplete coverage complicated the ranking in two situations:

1. Not all carcinogens in a problem area were covered. The assessments for a few environmental problems, such as Indoor Radon, theoretically considered all the cancer-causing substances associated with those problems. In other cases, such as Pesticide Residues on Foods, we extrapolated from a few suspected carcinogens to the universe of potential carcinogens within the problem area. For most problem areas, the quantitative analysis addressed only a subset of the total number of pollutants. An extreme example of this is Worker Exposure to Chemicals, where only four substances were examined, when workers may be exposed to as many as 20,000 substances.

2. Not all routes of exposure were covered. Because of intermedia transfer of carcinogenic substances (e.g., air and water pollution from hazardous waste sites), a given problem may pose cancer risks through several routes of exposure. However, in trying to address the most important routes of exposure, quantitative assessments were rarely based on more than a single exposure pathway. As a result, risks in problem areas in which intermedia transfers can take place may be understated relative to others.

Definitions and Boundaries of Problems Influenced Ranking

The boundary assigned to the definition of each problem area strongly influenced its ranking. For example, two classes of air pollutants -- volatile organic compounds and particulate matter -- have some carcinogenic components. Both classes are controlled by EPA regulations developed to reduce criteria air pollutants. However, we chose to examine these carcinogenic pollutants in the context of Hazardous/Toxic Air Pollutants. Thus, if we had defined this problem differently, Criteria Air Pollutants would rise considerably in the ranking.

We recognized at an early stage that there is considerable overlap in risks included in some problem areas. As a result, we did not consider Wetlands and Estuaries to avoid double counting of risk. The cancer risks that could be attributed to these environmental problems, which result largely through the consumption of contaminated seafood, are included in the other problem areas dealing with discharges into surface and ground waters. The "comments" section of Table 1 identifies problem areas where such double counting of cancer risk was a factor.

Using a Variety of Analytical Methods Complicated Ranking

The carcinogenic potencies of substances were estimated from two types of data. For the majority of substances suspected to cause cancer, potency estimates were based on the results of animal bioassays. Human epidemiologic data are available for only a few substances. This difference in the type of data used to assess risk probably introduces a bias between problem areas, particularly with respect to problem areas that rely entirely on human data (e.g., Indoor Radon).

Various methods of assessing exposure may also have biased comparisons of different problem areas. Not all analyses made exposure assumptions with the same degree of conservatism. Although we attempted to rank problem areas under a consistent set of assumptions, we could not always do so, given the wide variety of exposure situations. An example of this relates to the issue of mitigating behavior. For some environmental problems, people may take actions to reduce their exposure once they know they are at risk, even if there is no regulatory program to protect them. For example, people may stop drinking water that tastes bad or is known to be polluted. However, with the exception of the evaluation of risks from Underground Storage Tanks, no mitigating behavior is assumed to occur when we estimated cancer risks from the problem areas.

A similar situation exists in the evaluation of Drinking Water. Given that the majority of risks attributed to Inactive Hazardous Waste Sites result from drinking contaminated ground water, it initially may appear somewhat contradictory that Drinking Water ranks below this problem area. However, the Drinking Water calculations were based on contaminants actually detected in public water systems, while calculations for Inactive Hazardous Waste Sites were based on extrapolation from a small number of investigated sites to the total population of sites. (On the other hand, Inactive Hazardous Waste Sites did not account for inhalation or dermal exposures.) Clearly, if the cancer risks associated with all sources of ground-water contamination were considered under Drinking Water, the risks associated with this problem area would increase.

Different Levels of Confidence Complicated Ranking

Finally, the quality of information for the various environmental problems varied considerably. While there remains a high degree of uncertainty for any cancer risk assessment, we felt much more comfortable with our understanding of a problem area such as Radiation than we did for Stratospheric Ozone Depletion. It is unclear how this uncertainty may have biased our rankings, but it is likely to have had an effect.

OBSERVATIONS

The final ranking of the 31 environmental problems is heavily influenced by judgment. As such, it cannot be defended purely on scientific grounds. Rather, this final ranking represents the consensus of a work group composed of the senior technical staff and managers at EPA who are best qualified to make such judgments.

In this section we present several observations on the ranking itself, and on the ranking process.

EPA's Role Is Limited

Four of the six problem areas that ranked in the highest category are areas for which EPA has limited regulatory programs. For two of these, Worker and Consumer Product Exposures, EPA shares jurisdiction with other federal agencies which have primary jurisdiction in most cases. And, though EPA is the lead agency on the other two, Indoor Radon and Indoor Air Pollution, neither program lends itself to a conventional regulatory approach.

Inhalation Is The Major Exposure Route for High-Risk Problems

With the exception of Pesticide Residues on Food, the major route of exposure for all problem areas in the highest risk category is inhalation.

High Population Risk Often Means High Individual Risk

Although the problem areas were ranked on the basis of population risk, four of the six that ranked in the highest category also pose high individual risks. The two that do not are Dietary Exposures to Pesticide Residues on Food and Consumer Product Exposures.

Risks from Acute and Indirect Exposure Rank Low

Two types of problems ranked in Category 4 (the lowest category for which there is evidence of cancer risk): those related to accidental releases and discharges from point sources to surface waters. The problems related to accidental releases were ranked relatively low because exposure to carcinogens is acute, rather than chronic (that is, exposure is of short, rather than long, duration). It is possible that these problems would rank in a higher category if potentially chronic exposures related to ground-water contamination were considered. The two problem areas related to discharges to surface waters were ranked low mainly because people are only indirectly exposed to them, primarily through the relatively minor pathway of contaminated seafood.

Ordinal Rankings Are Not Precise

Ranking environmental problems was complicated by a lack of information, uncertainties in estimating exposures, the diversity of methods used to assess different problems and to project national cancer incidence from smaller-scale studies, and differences in the degree of coverage of potential carcinogens. The ranking is thus best described as a consensus judgment. Therefore, we do not believe the ordinal rankings have great precision. Rather, they generally indicate the relative cancer effects for each environmental problem. For this reason, the work group warns against placing too great a reliance on the ordinal ranking, particularly when similarly ranked problems are compared.

Dividing Lines Between Risk Categories Are Fuzzy

We believe that the risks associated with environmental problems grouped in one category are different from those grouped in another category. However, the precise location of the dividing lines between the categories was somewhat arbitrary. Often, the risk associated with a problem ranked at the bottom of one category was similar to the risk of a problem at the top of the next category.

Quality of Exposure Data Is Highly Variable

In general, the quality of human exposure data for the 31 environmental problem areas varies greatly, making comparisons difficult. Though in all cases exposure estimates are less than ideal, they are particularly lacking for problem areas relating to surface waters, ground water, solid waste, and new chemicals.

Relatively Few Substances Have Been Tested for Carcinogenicity

In general, the number of substances for which we have reasonably good cancer data (e.g., animal bioassay or human epidemiology) is a small subset of the number of chemicals to which the public is exposed. Because so little is known about the vast majority of chemicals, it is difficult to compare problem areas with only a few substances (e.g., Indoor Radon) with those represented by many substances (e.g., Worker Exposure to Chemicals).

RECOMMENDATIONS

Because this ranking is based only on cancer risk, we have not made recommendations regarding policy or resource allocation. We believe such recommendations should appear only in the Overview Report, which will contain the results of the four separate work groups for this project. However, we do have the following recommendations based on the experience of working on this project.

Data Base Needs Expansion

Significant improvements in the precision of the ranking will be possible only if a greatly expanded data base is available. In general, developing new data on carcinogenic substances and human exposures to carcinogens in the environment takes a considerable amount of time. Thus, it is not likely that there will be enough new information to warrant a new attempt to rank these problems for several years.

Next Step Should Consider Addressability

An important step in evaluating how to use the information presented here will be to analyze each environmental problem to ensure that suitable action can be taken, that taking such action is within EPA's mandate, and that the prospective improvements in health and/or welfare warrant the expenditure of resources. As a follow-up to this project, we recommend that EPA more comprehensively evaluate the environmental problem areas examined here.

APPEDNIX A

GENERAL PROCESS FOR ESTIMATING CARCINOGENIC RISK

Appendix A

General Process for Estimating Carcinogenic Risk

This appendix describes the way EPA generally assesses cancer risk. The first section outlines the methods EPA uses to estimate cancer potency for suspected or known human carcinogens. Included in this section is a list of the carcinogens we considered in making quantitative estimates of cancer risk, as well as the potencies that have been computed for each of these. Following this is a general discussion of how exposures are estimated in EPA analyses. Exposure assessments for individual problems areas are outlined in Appendix B.

We evaluated both chemical and physical (i.e., radon and other radiation sources) carcinogen hazards in this comparative ranking. The risks from radiation-induced cancer are addressed in Appendix B in the section on Radiation (page B-49). The risk estimates for chemical carcinogens were prepared by EPA's scientists according to procedures described in EPA's Guidelines for Carcinogen Risk Assessment (51 FR 33992, September 24, 1986).

ASSESSING CARCINOGENS

Selecting Data

In selecting experimental data to use to estimate potential cancer risks to humans, the quality of the data, its relevance to human modes of exposure and other technical details must be examined. Where possible, estimates were based upon human epidemiologic data (see Table A-1). In the absence of human data, data from animal species were used. Often, several studies were available for a given agent that involved different animal species, strains, and sexes, at several doses and different routes of exposure. When this was the case, tumor incidence data were separated according to organ site and tumor type and all biologically and statistically acceptable data sets were examined. The range of the risk estimates were calculated, giving due regard to biological relevance (particularly in the case of animal studies) and the appropriateness of the route of exposure. It was assumed that human sensitivity is as high as the most sensitive responding animal species unless there was evidence to the contrary. Therefore, the greatest evidence is generally given to the biologically acceptable data set from long-term animal studies showing the greatest sensitivity, again with due regard to biological and statistical considerations.

Where a single study revealed two or more significantly elevated tumor sites or types, extrapolations were conducted on sites or types selected on biological grounds. To obtain a total estimate of carcinogenic risk, data from animals with one or more significantly elevated tumor site or type were pooled and used for extrapolation. If the tumor sites or types actually occur independently, this procedure is the same as summing the risks from the several kinds of statistically significant tumors. The pooled estimates generally were used in preference to risk estimates based on single sites or types.

Benign tumors were usually combined with malignant tumors for risk esti-

mates unless the benign tumors were not considered to have the potential to progress to the associated malignancies of the same morphologic type, as in the case of tumors associated with exposure to formaldehyde.

Choosing a Mathematical Extrapolation Model

Because risks to low exposure levels cannot be measured directly by either animal experiments or epidemiologic studies, several mathematical models have been developed to extrapolate from high to low dose. However, while different extrapolation models may fit the observed data reasonably well, they may lead to considerable differences in the projected risk at low doses. No single mathematical procedure is yet recognized by the scientific community as the most appropriate for low-dose extrapolation in carcinogenesis.

To adequately protect human health, EPA has adopted a procedure whereby it strives to define an upper bound of the risk, rather than estimating the true risk -- a task that EPA believes is normally undefinable. Although mechanisms of carcinogenesis remain are largely unknown, at least some elements of the process have been elucidated -- e.g, linearity of tumor initiation. Thus, a linear multistage model has been adopted to define the plausible upper bound for risk. In further support of a linear model, it has been shown that if a carcinogenic agent acts by accelerating the same stages of the carcinogenic process that lead to the background occurrence of cancer, the added effect of the carcinogen at low dose is virtually linear. Thus, a model that is linear at low dose is plausible.

Therefore, the linearized multistage model low-dose extrapolation procedure was used in the risk estimates compared for this project. We emphasize that the linearized multistage model leads to a plausible upper limit to the risk, which is consistent with some likely mechanisms of carcinogenesis. Such an estimate, however, does not necessarily give a realistic prediction of the risk.

In certain cases, the linearized multistage model cannot be used successfully with the observed data. For example, it is unsuitable when the data are not monotonic or flatten out at high doses. In these cases it may be necessary to adjust the procedure to achieve low-dose linearity. In addition, a different low-dose extrapolation model might be conidered more appropriate when pharmacokinetic or metabolism data are available, or when other substantial evidence on the mechanistic aspects of the carcinogenesis process exists.

Extrapolating Animal Exposures to Humans

Low-dose risk estimates derived from laboratory animal data extrapolated to humans are complicated by a variety of factors that differ among species and potentially affect the response to carcinogens. Included among these factors are differences between humans and experimental test animals with respect to life span, body size, genetic variability, population homogeneity, existence of concurrent disease, such pharmacokinetic effects as metabolism and excretion patterns, and the exposure regimen. For many suspected or known carcinogens, it is not currently possible to account for these factors.

The approach for making interspecies comparisons was to use standarized scaling factors, such as mg per kg body weight per day and per lifetime, ppm

in the diet or water, and mg per m^2 body surface area per day. In the absence of comparative toxicological, physiological, metabolic, and pharmacokinetic data for a given suspect carcinogen, the extrapolation of body weight weight to the 0.67 power (approximating surface area) was considered to be appropriate.

Ultimately, the risk extrapolations were made and were presented in any of several forms, called a potency or unit risk estimate. Under an assumption of low-dose linearity, the unit cancer risk is expressed as the excess lifetime risk due to a continuous lifetime exposure of one unit of carcinogen concentration. Several types of units used for analyses are shown in Table A-1 and A-2. Risks from airborne carcinogens are typically defined in terms of lifetime exposes of ug/m^3 or ppm. Drinking water risks are expressed as either lifetime risks of exposure to a ug/l or mg/kg body weight/day. Other ingestion risks also use this type of expression. Typically, these estimates are characterized as being upper-limit values in that the risks are not likely to be higher than these values and may be significantly lower.

The listing of potency values derived from the same methodologic approach are comparable only to a certain point, which at best may be characterized as suitable only for prioritization purposes. This limited confidence in comparing potency estimates stems from the inherent requirement for judgment and differing qualities of individual data sets which were ultimately combined to produce a specific risk estimation.

Potency Estimates Used For This Report

Tables A-1 and A-2 present the potency estimates used in the regulatory risk assessments summarized in Appendix B. In addition to the potency estimate (expressed in the units cited in the original assessment), information is presented on the route of exposure, whether the estimate is based on human (epidemiologic) data, and which environmental problem area(s) relied on a given potency.

In general, differing potency estimates for the same chemical result merely from differing units used in analyses. Occasionally, potencies for a given substance will vary with the route of exposure (e.g., vinyl chloride). In at least one case, that of chloroform at Municipal Nonhazardous Waste Sites, an outdated potency estimate is cited, because we could not easily incorporate the most recent estimate into the analysis.

Table A-1

Cancer Potency Estimates Used To Generate Quantitative Risk Estimates: Nonradiation-Based Estmiates

Substance	Potency Estimate	Route Of Exposure	Problem Areas	Human Data?
Acenapthene	11.5 mg/kg/day	Ingestion	Hazardous Waste - Active	No
Acrylamide	3.7 mg/kg/day	Ingestion	Drinking Water	No
Acrylonitrile	2.4×10^{-1} mg/kg/day	Ingestion	Hazardous Waste - Active	No
Alachlor	1.0×10^{-1} mg/kg/day	Ingestion	Drinking Water	No
			Pesticide Residues	No
			Pesticide Application	No
Alar (Daminozide)	2.3×10^{-2} mg/kg/day	Ingestion	Pesticide Residues	No
			Pesticide Application	No
Arsenic	15 mg/kg/day	Ingestion	Mining Waste	Yes
			Nonhaz. Waste -- Industrial	Yes
			Hazardous Waste -- Inactive	Yes
			Hazardous Waste -- Active	Yes
			Drinking Water	Yes
			Contaminated Sludge	Yes
	4.3×10^{-3} ug/m^3/lifetime	Inhalation	Haz./Toxic Air Pollutants	Yes
			Nonhaz. Waste -- Municipal	Yes
Asbestos	7.6×10^{-3} ug/m^3/lifetime	Inhalation	Haz./Toxic Air Pollutants	Yes
	Not reported		Worker Exposures	Yes
Benzene	8.3×10^{-6} ug/m^3/lifetime	Inhalation	Haz./Toxic Air Pollutants	Yes
	2.9×10^{-2} mg/kg/day	Ingestion	Hazardous Waste - Inactive	Yes
			Storage Tanks	Yes
			Nonhaz. Waste -- Industrial	Yes
			Hazardous Waste -- Active	Yes
			Drinking Water	Yes
			Indoor Air	
Beryllium	8.4 mg/kg/day	Ingestion	Drinking Water	Yes
		Inhalation	Contaminated Sludge	Yes

Substance	Potency Estimate	Route of Exposure	Problem Areas	Human Data?
1,3-Butadiene	2.8×10^{-4} ug/m^3/lifetime	Inhalation	Haz./Toxic Air Pollutants	No
Cadmium	1.8×10^{-3} ug/m^3/lifetime	Inhalation	Haz./Toxic Air Pollutants	Yes
	6.10 mg/kg/day	Ingestion	Hazardous Waste -- Active	Yes
		Inhalation	Nonhaz. Waste -- Municipal	Yes
			Contaminated Sludge	Yes
Carbon Tetrachloride	1.5×10^{-5} ug/m^3/lifetime	Inhalation	Haz./Toxic Air Pollutants	No
			Indoor Air	No
	1.3×10^{-1} mg/kg/day	Ingestion	Drinking Water	No
Chlordane	1.6 mg/kg/day	Ingestion	Drinking Water	No
			Contaminated Sludge	No
Chlordimeform	1.28 mg/kg/day	Ingestion	Pesticide Residues	No
	9.4×10^{-1} mg/kg/day	Ingestion	Pesticide Application	No
Chloroform	2.3×10^{-5} ug/m^3/lifetime	Inhalation	Haz./Toxic Air Pollutants	No
			Indoor Air	No
	8.1×10^{-2} mg/kg/day	Ingestion	Hazardous Waste -- Active	No
	7.0×10^{-2} mg/kg/day	Ingestion	Nonhaz. Waste -- Industrial	No
Chromium (hexavalent only)	41 mg/kg/day	Both	Hazardous Waste -- Active	Yes
		Both	Nonhaz. Waste -- Municipal	Yes
		Both	Contaminated Sludge	Yes
	1.2×10^{-2} ug/m^3/lifetime	Inhalation	Haz./Toxic Air Pollutants	Yes
Coke Oven Emissions	6.2×10^{-3} mg/kg/day	Inhalation	Haz./Toxic Air Pollutants	Yes
Dibromo-chloropropane	1.4 mg/kg/day	Ingestion	Drinking Water	No
para-Dichlorodi-benzene	6.0×10^{-3} mg/kg/day	Inhalation	Consumer Products	No
			Indoor Air	No
1,2-Dichloroethane	2.6×10^{-5} ug/m^3/lifetime	Inhalation	Haz./Toxic Air Pollutants	No
	9.1×10^{-2} mg/kg/day	Ingestion	Hazardous Waste -- Inactive	No
			Drinking Water	No

Substance	Potency Estimate	Route Of Exposure	Problem Areas	Human Data?
2,4-Dinitrotoluene	3.1×10^{-1} mg/kg/day	Ingestion	Hazardous Waste -- Active	No
Dioxin (2378-TCDD)	1.6×10^{-5} mg/kg/day	Ingestion	Drinking Water	No
Epichlorhydrin	9.9×10^{-3} mg/kg/day	Ingestion	Drinking Water	No
Ethylene Dibromide	2.0×10^{-3} ug/l/lifetime	Ingestion	Drinking Water	No
Ethylene Oxide	1.0×10^{-4} ug/m^3/lifetime	Inhalation	Haz./Toxic Air Pollutants	No
Ethylene Thiourea	6.3×10^{-1} mg/kg/day	Ingestion	Pesticide Residues Pesticide Application	No No
Foplet	3.5×10^{-3} mg/kg/day	Ingestion	Pesticide Residues Pesticide Application	No No
Formaldehyde	1.3×10^{-5} ug/m^3/lifetime	Inhalation	Haz./Toxic Air Pollutants Consumer Products Worker Exposures	No No No
Gasoline Vapors	3.1×10^{-3} ppm/lifetime	Inhalation	Haz./Toxic Air Pollutants	No
Methylene Chloride	4.1×10^{-6} ug/m^3/lifetime 7.5×10^{-3} mg/kg/day 1.4×10^{-2} mg/kg/day	Inhalation Ingestion Both	Haz./Toxic Air Pollutants Drinking Water Worker Exposures Consumer Products Hazardous Waste -- Active	No No No No No
Polycyclic Organic Hydrocarbons	3.6×10^{-7} ug/l/lifetime	Ingestion	Drinking Water	No
Polychlorinated Biphenyls	4.3 mg/kg/day	Ingestion	Drinking Water Contaminated Sludge	No No
Products of Incomplete Combustion	4.3×10^{-1} ug/m^3/lifetime	Inhalation	Haz./Toxic Air Pollutants	No

Substance	Potency Estimate	Route of Exposure	Problem Areas	Human Data?
Telone II	1.7×10^{-5} mg/kg/day	Ingestion	Pesticide Residues	No
			Pesticide Application	No
1,1,2,2 - Tetrachloroethane	2.0×10^{-1} mg/kg/day	Ingestion	Nonhaz. Waste -- Municipal	No
Tetrachloroethylene	5.8×10^{-7} ug/m^3/lifetime	Inhalation	Haz./Toxic Air Pollutants	No
			Indoor Air	No
	5.0×10^{-2} mg/kg/day	Both	Worker Exposures	No
			Hazardous Waste -- Inactive	No
			Hazardous Waste -- Active	No
Tobacco Smoke	2.0×10^{-2} mg daily tar/ 40 years	Inhalation	Indoor Air	Yes
Toluene Diamine	2.9×10^{-1} mg/kg/day	Ingestion	Hazardous Waste -- Active	No
Toxaphene	1.1 mg/kg/day	Ingestion	Drinking Water	No
			Contaminated Sludge	No
Trichloroethylene	1.3×10^{-6} ug/m^3/lifetime	Inhalation	Haz./Toxic Air Pollutants	No
			Indoor Air	No
	1.1×10^{-2} mg/kg/day	Ingestion	Hazardous Waste -- Inactive	No
			Drinking Water	No
TSDF Emissions	1.5×10^{-5} ug/m^3/lifetime	Inhalation	Haz./Toxic Air Pollutants	No
Vinyl Chloride	4.1×10^{-6} ug/m^3/lifetime	Inhalation	Haz./Toxic Air Pollutants	Yes
	2.3 mg/kg/day	Ingestion	Hazardous Waste -- Inactive	No
			Drinking Water	No
	1.8×10^{-2} mg/kg/day	Ingestion	Nonhaz. Waste -- Municipal	No

Table A-2

Cancer Potency Estimates Used to Generate Quantitative Risk Estimates: Radiation-Baded Estimates

Substance	Potency Estimate	Problem Areas	Human Data?
Radiation (Total Pop.)	3.0×10^{-4} fatal cancers/person-rem	Radiation	Yes
Radiation (Adults)	2.0×10^{-4} fatal cancers/person-rem	Radiation	Yes
Radium 226	1.0×10^{-5} pCi/l/lifetime	Drinking Water	Yes
Radium 228	5.0×10^{-6} pCi/l/lifetime	Drinking Water	Yes
Radon	1.2×10^{-3} cancers/person-WLM	Radiation	Yes
		Indoor Radon	Yes
	1.0×10^{-7} pCi/l/lifetime	Drinking Water	Yes
	1.5×10^{-5} person/yr/WLM	Mining Waste	Yes
Uranium	1.4×10^{-6} pCi/l/lifetime	Drinking Water	Yes

ASSESSING EXPOSURE

The main objective of any exposure assessment is to provide reliable data and/or estimates of exposure for use in a risk assessment. Exposure assessment may be defined as the determination or estimation (qualitative or quantitative) of the magnitude, frequency, duration, and route of exposure. Exposure assessments may consider past, present, and future exposures with different techniques used for each phase -- e.g., biological accumulation for past exposures, measurement of existing exposure, and modeling of future exposures.

The exposure asessments we used in this ranking process varied considerably. This was largely due to the availability of data for each particular environmental issue. Thus, Appendix B describes the information used to assess each of the environmental problems. Below, we describe the relevant sections of EPA's Guidelines for Exposure Assessment (51 FR 34042, September 24, 1986). In some cases, these guidelines were not taken into account because many of analysis were undertaken before the guidelines were developed. Further, the broad nature of the assessments used for this project make much of the detail outlined in the guidelines overly ambitious. Therefore, caution should be exercised in evaluating the exposure estimates.

The guidelines emphasize that risk assessments will be conducted on a case-by-case basis, giving full consideration to all relevant scientific information. The guidelines also stress that this information will be fully presented in EPA risk assessment documents, and that EPA scientists will identify the strengths and weaknesses of each assessment by describing uncertainties, assumptions, and limitations, as well as the scientific basis and rationale for each assessment. Finally, the guidelines are formulated in part to bridge gaps in risk assessment methodology and data. By identifying these gaps and the importance of the missing information to the risk assessment process, EPA wishes to encourage research and analysis that will lead to new methods of assessing risk.

The scope of exposure assessments may vary. It depends on a variety of factors, including available data, regulatory concern, resources available, degree of exposure, perceived toxicity. Assessments used in this project generally extrapolated to a national level, facilitating the use of a great many assumptions with considerable uncertainty.

Five major aspects of exposure should be addressed in any exposure assessment: the sources of the pollutant of concern; the exposure pathways and environmental fate; measured or estimated concentrations; exposed populations; and integrated exposure analysis. These five features are appropriate for exposure assessments in general, whether the assessments are of a global, national, regional, local, site-specific, work-place-related, or other scope. The topics are appropriate for exposure assessments on new or existing chemicals and radiation sources, as well as single-media and multimedia assessments. Since exposure assessments are performed at different levels of detail, the extent to which any assessment addresses these aspects will vary.

Sources

The points at which a substance is believed to enter the environment

(indoor or outdoor) should be described, along with any known rates of entry. The assessment should describe hte human activities related to the substance and the environmental releases resulting from those activities. Seasonal variations in environmental releases, if applicable, should also be examined. The environmental releases can be described in terms of geographic and temporal distribution, as well as the receiving environmental media.

Exposure Pathways and Environmental Fate

The exposure pathways section should address how a pollutant moves from the source to the exposed population or subject. For a less detailed assessment, broad generalizations on environmental pathways and fate may be made. In the absence of data -- e.g., for new substances -- fate estimates may have to be predicted by analogy with data from other substances. Fate estimates may also be made by using measurements and/or models and laboratory-derived process rate coefficients. At any level of detail, certain pathways may be judged insignificant and not pursued further.

Measured or Estimated Concentrations

Measurements are used to identify releases and to quantitatively estimate both release rates and environmental concentrations. Some examples of uses of measurements are: sampling of stacks or discharge pipes for emissions to the environment, testing of products for chemical or radionuclide content, testing of products for chemical or radioactive releases, sampling of appropriate points within a manufacturing plant to determine releases from industrial processes or practices, sampling of potentially exposed populations using personal dosimeters, and sampling of solid waste leachate for chemical content. These data should be characterized as to accuracy, precision, and representativeness. If actual environmental measurements are unavailable, concentrations can be estimated by various means, including the use of fate models (see previous section) or by analogy with existing chemicals.

Concentrations of pollutants should be estimated for all environmental media (air, surface water, etc.) that may contribute to significant exposures. Generally, the environmental concentrations are estimated from measurements, mathematical models, or a combination of the two. If environmental measurements are not limited by sample size or inaccuracies, then exposure assessments based on measurements have precedence over estimates based on models.

The concentrations must be estimated and presented in a format consistent with available dose-response information. For carcinogens, an estimate of long-term average concentration will usually be sufficient. Future environmental concentrations resulting from current or past releases may also be projected. Moreover, if the agent has natural sources, the contribution of these to environmental concentrations may be relevant.

When the estimates of the environmental concentrations are based on mathematical models, the model results should be compared to available measurements, and any significant discrepancies should be discussed. Reliable, analytically determined values must be given precedence over estimated values whenever significant discrepancies are found.

Exposed Populations

Populations may be selected before an analysis is undertaken, but frequently the populations will be identified as a result of the sources and fate studies. An analysis of the distribution of the pollutant will reveal which populations will be subject to potentially high exposure rates. This population will then form the focus of the exposure assessment. Census and other survey data may be used to identify and describe the population exposed to various contaminated environmental media. Site-specific assessments then had to be extrapolated to a national level. The methods for this step were chosen on a case-by-case basis.

Integrated Exposure Analysis

The integrated exposure analysis combines the estimation of environmental concentration (sources and fate information) with the description of the exposed population to yield exposure estimates. Data should be provided on the size of the exposed populations; duration, frequency, and intensity of exposure; and routes of exposure. To the extent possible, behavioral and biological characteristics of the exposed populations should be considered. In addition, an estimate of the uncertainty associated with them should be provided.

Depending on the scope of the exposure assessment, the total exposure may be fractionated into one or more "exposure scenarios" to facilitate quantification. For example, seven very broad scanarios are recognized: occupational, consumer, transportation, disposal, food, drinking water, and ambient exposures. Investigation of only one scenario may be necessary for the scope of some assessments, as was the case for most of the environmental issues we compared in this document. Ideally, however, all relevant exposure scenarios should be considered.

ASSESSING RISK

Risk estimates may be in three forms: (1) the dose corresponding to a given individual risk level; (2) excess individual lifetime risks; or (3) the number of cancers (or cancer deaths) produced per year in the exposed population -- e.g., population risk. In this project, we have focused our main attention on estimates of population risk, which we made by combining potency estimates (or unit risks) with human exposure data. Irrespective of the form we chose, however, the degree of precision and accuracy in the numerical risk estimates usually does not allow us to use more than one significant figure. In characterizing the risk due to concurrent exposure to several carcinogens, the risks can be considered to be additive, unless there is specific information to the contrary.

In every quantitative risk estimation the results are highly uncertain. For this reason, we are usually estimating the the upper limit of risk as opposed to an estimate of the true risk. In addition, the uncertainties due to experimental and epidemiologic variability as well as uncertainty in the exposure assessment can be important. There are major uncertainties in extrapolating both from animals to humans and from high to low doses. There are important differences in the way species and strains take in, metabolize, and distribute carcinogens among their organs, as well as their differences

in target site susceptibility. Similarly, human populations are highly variable with respect to genetic constitution, diet, occupational, and home environment, activity patterns, and other cultural factors. The hazard assessment should always be presented along with the risk estimates to ensure that there is an appreciation of the weight of evidence for carcinogenicity that underlies the quantitative risk estimates. These and other factors need to be examined when considering the results of quantitative cancer risk assessment.

APPENDIX B

RISK CALCULATIONS FOR SPECIFIC ENVIRONMENTAL PROBLEMS

Appendix B

Risk Calculations for Specific Environmental Problems

This appendix presents summaries of the cancer risk information compiled for all of the environmental problems investigated. Each of these summaries begins with a short definition of the nature of the environmental problem, describing its boundaries and giving examples of the problem to the extent possible. This is followed by a discussion of the contaminants assessed, as well as other information relevant to examining the extent of the total problem assessed for this project.

Each summary discusses the methods used for estimating quantitative risks. Where an approach differed from the approach taken by EPA's Carcinogen Assessment Group (CAG), these cancer potencies are presented, along with an explanation of why a different approach was taken. The methods used to estimate exposure will also be explained in this section, including the extrapolation from specific exposures to nationwide estimates, whether contaminant concentration estimates are based on measured or modelled data, and whether nonstandard assumptions are made with respect to human intake of air, food, or water.

The final section of each summary presents the results of the risk assessment for both population and maximum individual risk, where possible. Some summaries also comment on the extent to which risk estimates are likely to improve in the near future.

Throughout the summaries, uncertainties and caveats specific to each environmental problem are addressed as they arise in the discussion. Where particular uncertainties or caveats do not easily fit into the structure of the summary, they are addressed at the end of the summary.

The descriptions of the individual environmental problems are arranged in the order in which they were ranked.

CATEGORY 1

1: Worker Exposure to Chemicals (tied with Indoor Radon)

PROBLEM DEFINITION

Workers are exposed to chemical substances in a wide range of occupational settings. These include chemical manufacturing and processing, and the use of chemicals in industry and the trades.

According to 1977 figures, over 20,000 chemical substances are in commerce at greater than 10,000 kg in any one year. In addition, manufacturing and processing operations generate a significant number of by-product streams that are an additional source of potential exposure to workers.

Because of the diverse nature of processes and equipment and the great range their of physical and chemical properties, occupational exposure varies greatly in different settings. However, unlike most other environmental problems, exposure to chemicals in the work place is not mediated by environmental pathways and often occurs in confined indoor environments. Further, significant exposures can take palce to the workers even where relatively small amounts of the substance are involved.

Exposure to workers handling pesticides is discussed under Application of Pesticides. Exposure to workers involved in transporting and disposing of chemicals and wastes, as well as exposures to miners, are also not considered in this analysis.

EPA'S RESPONSIBILITIES IN PROTECTING WORKERS

The primary federal responsibility for occupational safety and health resides in the Occupational Safety and Health Administration. However, the Toxic Substances Control Act (TSCA) requires EPA, in evaluating chemical risks, to consider the full "life cycle" of chemical substances -- i.e., manufacturing, processing, using, and disposing of chemicals. Under Section 4 of TSCA, EPA must identify which chemicals should be tested for potential toxicity (including carcinogenicity) and require industry to test them. In doing so, EPA must take into account the full range of potential exposures, including exposure in the work place.

Under Section 5 of TSCA, EPA has evaluated potential occupational and other areas of risk from over 7,000 "new chemicals." EPA has taken action on at least 200 of these chemicals to protect workers from potential risks. A number of regulatory actions taken under Section 4(f) and referrals of identified problems to other agencies under Section 9 of TSCA have been driven by concern for occupational risks. Finally, under the new Superfund Amendments and Reauthorization Act and the recently passed asbestos abatement legislation (AHERA), EPA is given major responsibilities in protecting publicsector employees from certain occupational chemical hazards.

POLLUTANTS ADDRESSED IN THIS SUMMARY

Criteria for Selection of Occupational Hazards

Only limited data exist wiith respect to that portion of current cancer incidence that may be attributed to chemical exposure in the work place. Estimates of the contribution of occupational factors to national cancer death rates vary from a few percent to 20 percent. To provide some perspective on the comparative risks presented by occupational exposures to chemicals, as opposed to other exposures to chemicals, the cancer work group chose substances for which estimates of both cancer potency and exposure are available. These include formaldehyde, tetrachloroethylene, methylene chloride, and asbestos.

Evidence of the carcinogenicity of the first three of these substances is relatively recent. As such, exposures to these substances may be higher than exposure to substances for which evidence of carcinogenicity has been available for some time (e.g., benzene, vinyl chloride, asbestos).

Extent to Which Risks of Selected Chemicals Represent Total Work Place Cancer Risks to Workers

The exposure of workers to chemical substances is relatively high when compared with the exposure of other populations. Because most occupational settings are often indoors significant levels of airborne contamination can result. Unless steps are taken to specifically mitigate exposure, the close proximity of workers to the substances and processing equipment increases the likelihood and magnitude of exposure.

The greatest number of chemical substances are likely to be found in the occupational setting. This results from the fact that is because all commercial substances must be manufactured, and a majority are used, in commercial settings. While the fraction of commercial chemical substances that are carcinogenic is not known, it is highly likely that the risks associated with the four substances assessed for this project represent a very small fraction of the overall risks of cancer from chemicals in the work place.

RISK ASSESSMENT METHODOLOGY

Cancer Potencies

Cancer potencies (unit risks) used to derive estimates of individual risk and expected annual incidence of cancers are consistent with those developed by the Carcinogen Assessment Group. However, for tetrachloroethylene and for methylene chloride, unit risk estimates are derived from the risk assessment conducted by the Office of Toxic Substances. These assessments are currently being reevaluated by an other EPA work group. For methylene chloride, this effort includes reviewing additional data developed by industry.

Exposure Assessment

The exposure-related information used to evaluate occupational risks in cludes estimates of both the number of workers exposed and the magnitude of the individual exposure. The magnitude of exposure involves estimates of air

concentration (the degree of contact in the case of dermal exposure) and the duration of exposure. Unless otherwise indicated, individual workers are assumed to be exposed 8 hours daily, 5 days a week, over a 40-year working lifetime.

To evaluate worker exposure, the work group has relied on the predictive methodologies developed by the Office of Toxic Substances. It has also used available work-place monitoring data, typically developed by industry to monitor compliance with existing standards.

The exposure estimates used to for this project have a number of limitations. For example, estimates of the total number of workers exposed to a chemical agent are often based on the total number of workers known to be involved in a particular industry sector. The fraction of these workers actually exposed to the agent may not be well characterized. The actual magnitude of exposure will typically have a broad distribution, based on the range of equipment and practices in the user industry. In addition, it is often not known to what extent monitoring data are "representative" of exposures experienced by the majority of workers.

For these reasons, exposures are sometimes expressed as a range of values. In these cases, a single value -- which represents the average or mean of the actual exposures anticipated -- is used to estimate total occupational risks.

ESTIMATED RISK

The estimates of occupational risks are based on the exposure estimates and unit-risk estimates described above. The anticipated incidence of cancer based on 40 year exposure period for these four substances is shown below.

Substance	Estimated Occupational Annual Cancer Incidence
Formaldehyde	100
Methylene Chloride	90
Tetrachloroethylene	10
Asbestos	55

CATEGORY 1

#1: Indoor Radon (tied with Worker Exposure to Chemicals)

PROBLEM DEFINITION

Radon is a radioactive noble gas produced by the decay of radium, which occurs naturally in almost all soil and rock. Because radon is chemically inert, it is not retained by most body tissues and poses very little direct health risk. However, inhaled radioactive decay products of radon, known as radon daughters, deposit in the air passages of the lung, and emit alpha particles that irradiate bronchial epithelium, possibly leading to lung cancer. Concentrations of radon daughters are ordinarily given in units of working levels (WL), which were first derived in occupational studies. Exposure (concentration x time) is usually expressed in working-level months (WLM).

The greatest health risks from radon occur when it moves through the soil into houses where it is trapped, producing a build-up of the radon daughters. Radon can also emanate from a house's building materials or enter a house through drinking water (see Drinking Water) or natural gas that is piped in.

RISK ASSESSMENT METHODOLOGY

That inhaled radon daughters can cause lung cancer is well documented for both laboratory animals and humans. Risk estimates are based on epidemiological studies of exposed cohorts of miners, primarily uranium miners. It is assumed that the risk is proportional to past WLM exposure (adjusted for a 10-year minimum latency period between exposure and cancer onset), with no threshold.

Laboratory experiments at the cellular level, as well as on whole animals, suggest that the risk from alpha particle irradiation is likely to be proportional to dose. However, at relatively high doses the risk per unit dose falls off because of cell killing. Human epidemiological evidence is consistent with this view.

National exposure estimates are derived from monitoring data collected in homes across the U.S. and extrapolated to the entire country.

In calculating risk, EPA uses a relative risk model. This model assumes that the risk of lung cancer is proportional to the baseline incidence rate of the disease in the population. Effectively, this means that as baseline lung cancer rates vary according to population characteristics (primarily age, sex, and smoking histories), estimates of risks of radon-induced lung cancer vary in parallel. The evidence in favor of a relative risk model is not conclusive, but studies of U.S. uranium miners strongly indicate that smoking and radon are more than additive and quite possibly multiplicative in their effects on lung cancer risk.

Based on an examination of the epidemiological evidence on miners exposed to various levels of radon, EPA has adopted a range of relative risk factors: 1%-4% per WLM (EPA, 1986). In other words, the chance of developing lung

cancer increases by between 1% and 4% for every WLM of exposure. In evaluating the risk to the general public, EPA adjusts the estimated exposure for reduced average breathing rates in the general population, as compared to miners, and for differences in the lung morphology of children (EPA, 1986). Consequently, EPA's risk estimates for the general public correspond to a range of about 0.6% to 2.4% per WLM, when expressed in standard units of exposure.

ESTIMATE OF RISK

The number of lung cancers induced by radon each year has been estimated to lie in the range of 5,000 to 20,000. The number of predicted cancers is approximately proportional to the average level of radon daughters in homes. This average level is still uncertain, but estimates have recently been rising as more homes are measured for radon. Moreover, if lung cancer estimates are calculated using a relative risk model, they will vary with changes in the baseline incidence of lung cancer. Because lung cancer rates have been increasing in recent years, the 5,000 - 20,000 estimate may have to be revised upward.

Some may question these estimates on the ground that when the fraction of excess lung cancers attributed to radon is added to the fraction attributed to smoking (about 85% according to the Surgeon General), the total may exceed 100%. There is actually no inconsistency, because both radon and smoking may be causal factors for a single lung cancer. Indeed, according to the relative risk model, for the great majority of radon induced lung cancers, smoking is also causal.

REFERENCE

U.S. EPA. Final Rule for Radon-222 Emissions from Licensed Uranium Mill Tailings: Background Information Document. EPA 520/1-86-009. August 1986.

CATEGORY 1

#3: Pesticide Residues on Foods

PROBLEM DEFINITION

EPA currently registers pesticide products for agricultural use that contain at least 600 active ingredients. These include chemicals used as insecticides, fungicides, herbicides, and rodenticides. Due to the wide use of these agents on food crops and their potential to induce deleterious toxic effects, including cancer, all require thorough assessment of their potential hazard to humans. EPA also registers several hundred other nonagricultural pesticide products. Although these may not result in significant dietary exposure to humans, they nevertheless require similar hazard assessment because of other modes of human exposure.

The spectrum of toxic effects capable of being induced by pesticides is great. It includes specific organ toxicity, reproductive/teratogenic effects, mutagenic effects, and cancer. Many of these changes are often produced by single pesticide chemicals in the controlled laboratory environment, but actually may result from additive and/or synergistic effects of exposure to multiple pesticides in concert under normal agricultural, as well as nonagricultural, use conditions. Thus, the scope of this environmental problem is wide, resulting from the use of a vast number of pesticides in the greater human environment.

As is the case for innumerable other chemicals to which humans are exposed, an area of great concern with respect to the use of pesticides is the induction of cancer. The actual number of pesticides that have the potential to induce a carcinogenic response is not known with certainty. The Office of Pesticide Programs is currently surveying its inventory of pesticide chemicals to determine which have had a sufficient number of valid rodent oncogenicity studies performed on them (usually two oncogenicity studies per chemical) to estimate the magnitude of this problem. To date, it is estimated that about 200 of the total of 600 agricultural use chemicals subject to EPA registration have been adequately tested for oncogenicity in animals. Of these 200 chemicals, about 60 have been identified as being potential oncogens. By extrapolation, therefore, the Office of Pesticide Programs considers it reasonable to assume that from the total of 600 agricultural-use pesticide active ingredients, as many as one-third, or 200, could be oncogenic.

RISK ASSESSMENT METHODOLOGY

Data Sources

EPA's two main sources for toxicologic data on pesticide chemicals are the regulated industry and the National Toxicology Program (NTP). The primary source of data is agricultural chemical companies in support of the field testing and registration of pesticides on crops within the United States. These companies have data on both new and existing pesticides. The Office of Pesticide Programs has already reviewed an extensive number of toxicity studies on such chemicals, but its inventory is far from complete, particularly in the case of older pesticides that were used domestically before the

initiation of more comprehensive testing for oncogenicity. To obtain this additional information, EPA has established a "data-call-in" procedure, whereby pesticide manufacturers must provide EPA all the data available on pesticides subject to EPA registration.

Through its Toxicology Research and Testing Program, the NTP has completed short- and long-term animal toxicology and mutagenicity tests on several pesticides. In addition, several other pesticides are being tested or are scheduled for testing. The NTP studies, which primarily involve older pesticide chemicals, are published as technical reports that are available to the general public and/or in the open scientific literature. The Office of Pesticide Programs is in close communication with the NTP on a routine basis and regularly receives newly generated data for evaluation and incorporation into its regulating activities.

Data Assessment

All toxicologic data received by the Office of Pesticide Programs in support of a pesticide application are independently reviewed by professional staff scientists employed in the Toxicology Branch. All toxicologic studies submitted by industry are evaluated in-house because of their critical nature in hazard identification. Similarly, all toxicologic studies obtained from the NTP or other public sources are reevaluated within the Branch as a matter of internal procedure.

For the following reasons, the data assessment or review process is extensive, complex, and time-consuming:

- The Office of Pesticide Programs is evaluating 600-1,000 active ingredients in pesticides for registration.

- Each of the active ingredients has been the subject of many complex biological studies that have been performed to characterize its toxicological profile. These studies commonly include several acute toxicity tests, mutagenicity studies, reproduction and teratology tests, pharmacokinetic assessments, subchronic and chronic tests, and one or more oncogenicity studies.

- Each study requires a thorough, detailed review by a staff scientist to assess its scientific validity and content. This review may require from 8 hours for a single acute toxicity test to 240 hours for a single oncogenicity study. Very often, 1,000 hours (0.5 person-year) are spent reviewing all of the studies submitted in support of the registration of a single pesticide ingredient.

- If oncogenic effects are apparent in the data base, additional work in reviewing the data base and in assisting regulatory activity is required, often involving 0.25 person-year/chemical.

Exposure Estimates

National exposure to pesticide residues is estimated either by using tolerances or by determining actual pesticide residues on food as it is consumed. The Office of Pesticide Programs uses tolerances to regulate the use of agricultural chemicals. They represent the legal maximum of pesticide

residues that are allowed to remain on raw agricultural commodities.

To estimate human exposure to pesticide residues on food crops, we added these tolerances, the average daily per capita food consumption figures for the crops, and the exposure figures for each crop. This addition provides the total maximum residue contribution (TMRC) for each particular pesticide chemical. The TMRC represents an upper-limit estimate of average human exposure to pesticide residues because it includes tolerances for all raw agricultural commodities on which the pesticide is used.

The estimation of pesticide-related human cancer risks within the Office of Pesticide Programs requires a determination of the actual residue levels on foods as they are consumed, and the effect of food processing on the residues that are contained in the foods. To obtain this information, the Office of Pesticide Programs has recently developed a food consumption matrix, known as the Tolerance Assessment System (TAS). This system provides data (1) on the primary foods consumed by particular segments of the U.S. population -- such as children, the aged, and ethnic or regional groups; (2) on the range of variation of consumption within any group; (3) on whether the food is consumed raw, cooked, or processed; and (4) on seasonal variations in food consumption.

In summary, TMRCs, based on levels of residue tolerances, provide a guidepost for dietary exposure assessments in relation to raw agricultural commodities. The TAS, however, facilitates the translation of residues on raw agricultural commodities into human exposure estimates by generating much more extensive and accurate residue data that can be matched to food items as they are consumed. The result is an estimate of actual residue consumption that can be used to address specific issues, such as human cancer risks related to dietary exposure of the U.S. population to pesticides.

The Office of Pesticide Programs has already used the TAS in regard to several oncogenic pesticide chemicals. It has found that, on the average, the actual exposure of humans to the pesticides in question is approximately 50 times less than described using tolerance methods.

Risk Calculation

The quantification of human cancer risk from pesticide residues is based on animal toxicologic data. The oncogenic dose-response data in an animal study, are extrapolated into regions of progressively lower exposure. Using mathematical models accepted by EPA yeilds a sliding scale linking risk probabilities to exposure. Potency estimates (expressed as risks per unit exposure in mg/kg/day) for humans are calculated for low doses. These methods are consistent with EPA's Guidelines for Carcinogenic Risk Assessment.

ESTIMATED RISKS

In the present survey, a sample of the human population risk from pesticide residues was obtained by evaluating exposure information, using TMRC or tolerance methods for seven oncogenic chemicals.

The average population risk for these seven chemicals from dietary exposure was estimated to be 100,000 people per lifetime for each chemical. Cal-

culations using TMRCs are known to represent an upper limit of the population risk, and calculations using TAS average exposure may be as much as 50-fold lower than those based on TMRCs. Thus, the value of 100,000 people/lifetime/ chemical was divided by 50 to yield a more representative risk estimate of 2,000 people/lifetime/chemical. Further mathematical manipulation of the figure to reach a risk estimate for each chemical on a yearly basis (i.e., dividing 2,000 people/lifetime/chemical by 70 years, the average lifespan) yields a value of approximately 30 people/year/ chemical as the expected risk. Since the Office of Pesticide Programs has estimated that as many as 200 pesticide chemicals may be oncogenic, the total annual population risk from dietary exposure to these chemicals is about 6,000 people/year (i.e., 30 people/year/chemical x 200 oncogenic chemicals).

UNCERTAINTIES

Several uncertainties are associated with human risk assessments resulting from exposure to pesticide residues. The prominent uncertainties include the following examples.

First, the actual amount of pesticide residues consumed by humans is not known with certainty. Estimations of consumption are usually made for residues contained on individual crops, and these are often added together to obtain "representative" values. However, the entire national crop is often not treated at the same time with a particular pesticide. In addition, food crops are not equally consumed by all Americans. There are usually significant differences in consumption for ethnic, age, sex, socioeconomic, and regional groups, and food processing may alter the levels of residues on foods. Finally, there may in fact be residues of multiple pesticides on crops. Factors such as these make determinations of actual residues consumed difficult to obtain. However, the TAS in use in the Office of Pesticide Programs takes most of these factors into account. It is assumed to be a good predictor of the types and amounts of pesticide residues consumed by humans.

Second, there is uncertainty in extrapolating observed toxicological effects in animals to expected effects in humans. For example, different species respond differently to toxic chemicals, experimental animals are usually exposed to pesticides on a continuing basis at high dosage levels in order to deliberately evoke and thus define toxic responses for chemicals, and some toxic effects occur in animals but are rarely seen in the human population (e.g., liver tumors in mice). To the extent that is possible, the Office of Pesticide Programs attempts to minimize these problems by thoroughly evaluating all available animal toxicity data on individual pesticides before extrapolating possible hazards to humans. It does this through the Toxicology Branch Peer Review Committee, which conducts "weight-of-the-evidence" meetings on pesticides and determines, using the EPA Guidelines for Carcinogen Risk Assessment, whether they are likely to be human oncogens.

Third, the mathematical low-dose extrapolation procedures used in animal studies to quantify human risks for pesticide residues are uncertain. This uncertainty stems largely from the fact that the mechanisms of action responsible for inducing oncogenic responses for pesticides are unknown. This situation, however, pertains to all chemicals reported to be oncogens.

CATEGORY 1

#4: Indoor Air Pollution (tied with Consumer Products Exposure)

PROBLEM DEFINITION

On average, people spend nearly 90 percent of their time indoors. Moreover, for many pollutants, indoor levels are considerably higher than outdoor levels. Therefore, for most persons, indoor exposures to air pollution predominate over outdoor exposures.

Indoor air pollution is an accumulation of contaminants in building air. The sources of these contaminants are primarily within the building, although outdoor sources also contribute. Building design, construction, and operation also affect the accumulation and dispersion of indoor air pollutants. The primary means of limiting this accumulation of indoor pollutants is air exchange between indoor and outdoor microenvironments. In general, concentrations of indoor pollutants are directly proportional to the number of sources per unit volume and inversely proportional to the the rate at which more polluted indoor air is exchanged with less polluted outdoor air. National trends toward energy conservation, increased use of synthetic chemicals, and ignorance of good ventilation and housekeeping practices have all led to a rise in indoor air pollution.

Indoor Air Pollution in Residential Buildings

There are about 100 million housing units in the U.S., with roughly two million new housing units built each year. The Department of Energy has estimated that air exchange rates in new construction are, on average, 50 percent lower than the national average. Given this trend in air exchange rates, the concentration of indoor pollutants will double if emission rates stay the same.

Carcinogenic indoor pollutants may be generated by a variety of sources. Some of these are outlined below.

- o New types of insulation: Urea-formaldehyde foam insulation came into widespread use in retrofitting older, uninsulated homes in the late 1970's. The indoor air pollution potential of this material, which emits free formaldehyde, was not well understood. As a result, many homeowners have been exposed to considerable indoor concentrations of formaldehyde.

- o Pesticides: An estimated 84% of U.S. households use pesticides in the home. Recently, EPA's Office of Pesticide Programs has begun regulatory action to address the carcinogenic potential of some pesticide products. In some cases, termiticides have been misapplied in homes by commercial applicators, whereby chlordane has been accidentally introduced into subslab heating ducts, contaminating the home.

- o Environmental Tobacco Smoke (ETS): The 1986 Surgeon General's Report has identified ETS as a cause of disease, including lung cancer, in nonsmokers. A 1986 report of the National Academy of Sciences has estimated that ETS in the home causes about a 30% increase in lung

cancer risk in nonsmoking spouses. Estimates of lung cancer mortality range from about 500 to 7,000 deaths per year.

- o Ventilation: Furnaces, hot water heaters, woodstoves, and fireplaces in overtight houses may require more combustion air than is available, resulting in negative pressures creating backdrafting of chimneys. Carcinogenic termiticides have accidentally been introduced into forced-air heating systems. Overtight homes may have high levels of humidity leading to overgrowth of molds and fungi, producing carcinogenic mycotoxins. Electrostatic precipitator air cleaners and filters that are not cleaned may yield carcinogenic volatile organic compounds (VOCs) from trapped tobacco smoke.

- o Asbestos: Friable asbestos on furnaces and pipes in older homes and misused asbestos shingles may increase cancer risks.

- o Organics: Emissions of volatile organic compounds from (1) oven cleaners and hairsprays, (2) arts and crafts materials and home workshops, (3) solvents from cleaning and waxing agents, and (4) paints and refinishing compounds may increase exposure to a variety of air toxics. EPA°s Total Exposure Assessment Methodology (TEAM) study has found such carcinogens as benzene, ethylbenzene, trichloroethane, trichloroethylene, tetrachloroethylene, carbon tetrachloride, chloroform, and meta- and para-dichlorobenzenes to be commonly present in indoor air. Wood preservatives have caused toxic levels of pentachlorophenol to accumulate in log dwellings wrongly built with treated logs. Emissions of VOCs from drinking water may increase exposure to carcinogenic air toxics. Emissions of benzene and other carcinogenic hydrocarbons may occur from gasoline tanks of automobiles in parking garages in residences and commercial buildings and become entrained in the building air by diffusion. Some limited attempts at assessing carcinogenic risks of indoor VOCs in residential structures have been made and are summarized on page B-15.

Indoor Air Pollution in Nonresidential Buildings

This discussion will be confined to nonindustrial nonresidential buildings. The carcinogenic risks of pollutants in such buildings generally have not been quantified, except for tobacco smoke. Indoor air pollution in industrial buildings is considered under Worker Exposure to Chemicals.

In new and remodeled buildings, paints, solvents, glues, caulking, new carpeting, emissions from particle board, and other sources, outgas, causing high initial levels of volatile organic compounds which generally diminish as the building ages. Buildings usually are not outgassed at high temperatures before occupancy, and may even be occupied before completion of construction or during remodeling. Further, ductwork may not be protected from entry of dusts, including roofing tar or asbestos generated during construction.

Ventilation systems designed primarily for thermal load control may leave the building unventilated for long periods of time, allowing pollutant loadings to build up. Ventilation rates designed for acceptable indoor air quality may not be required by local building codes, nor enforced if they are.

RISK ASSESSMENT METHODOLOGY

Since extensive measurements do not exist for the wide range of potential indoor carcinogens in buildings, modeling on the basis of limited measurements is at present the only method to for assessing the cancer risk of selected pollutants. A simple indoor air pollution transport model based on the mass-balance equation has been developed to relate the concentration A(t) of a pollutant at time t to the generation rate G, removal rate R, and the building volume V:

$$A(t) = [G/(VR)] \times [1 - e^{-Rt}] .$$

For pollutants that are emitted more or less continuously, the time-dependent part of this equation becomes unity, and the equilibrium concentration of the pollutant A_{eq} (e.g., in units of micrograms per cubic meter or ppm) is determined by the ratio of the source emission density (G/V) (e.g., in units of micrograms per hour per cubic meter) to the removal rate R (e.g., in units of air changes per hour; one air change per hour is 1.44 times the halflife for pollutant removal). The amount of pollutant inhaled, I, in units of mass inhaled per unit time is given by the equation:

$$I = A\ B\ T,$$

where A is the average concentration of the pollutant in the building during time T, B is the average breathing rate of the individual over time T, and T is the duration of time spent in the building. The lifetime risk, X, of exposure to a given pollutant is then estimated from the equation:

$$X = I\ D\ L,$$

where D is the dose-response function for the pollutant (e.g., cancer cases or deaths per 100,000 person-years per unit mass of pollutant inhaled per unit time), and L is the exposure lifetime in years. Using analogous methods, Wallace (1986), Tancrede et al. (1986), and Repace and Lowrey (1985) have published preliminary risk assessments on a limited number of indoor pollutants.

A summary of the quantitative information compiled for this report follows.

Environmental Tobacco Smoke (ETS)

There is a considerable amount of epidemiologic data on smokers, with smoking associated with cancers of the lung, larynx, oral cavity, esophagus, bladder, pancreas, and kidney. With respect to passive smoking, epidemiologic studies have definitely established a link between exposure to environmental tobacco smoke and lung cancer, with some reports suggesting increases in brain tumors and hematopoeitic cancers.

A potency estimate has been calculated by Repace and Lowrey (1985), using a phenomenological model. This model predicts five lung cancer deaths per 100,000 person-years per milligram of tobacco tar inhaled per day or, alternatively, 2×10^{-3} for of 40 years exposure (Repace and Lowrey, 1986). Using a potency estimate derived from a 1-hit or multistage model (extrapolating from risks in smokers) yields a potency estimate that is one-tenth

as large. However, these results are inconsistent with epidemiologic observations in nonsmokers. Repace and Lowrey (1986) have postulated that these underestimates result from the large doses received by smokers; the slope of the exposure-response relationship for somewhat lower exposures experienced by nonsmokers is steeper than predicted by extrapolations from smokers.

There have been several attempts to quantify the cancer risks from ETS exposure. According to the National Acadamy of Science (NAS, 1986), domestic exposure is expected to increase risk of lung cancer by 30%. The aforementioned phenomenological model of Repace and Lowrey estimates very similar results, on the order of 26%. Total estimated mortality from domestic exposure is 2,400 lung cancer deaths/year (NAS, 1986); total estimated mortality from domestic plus work place exposure is 5,000 lung cancer deaths/year (Repace and Lowrey, 1985). Based on Repace-Lowrey phenomenological model, estimated lifetime risk to most-exposed individual is 3% (3×10^{-2}).

Volatile Organic Indoor Pollutants

With respect to the nationwide U.S. population risks, the best estimate available has been developed by Wallace (1986). On the basis of monitoring results from 600 homes in four states (New Jersey, North Carolina, North Dakota, and California), Wallace estimates 1,240 deaths per year across the nation from six organic pollutants (benzene, para-dichlorobenzene, chloroform, carbon tetrachloride, tetrachloroethylene, trichloroethylene). These risks are based on potency estimates developed by EPA's Carcinogen Assessment Group (CAG).

Tancrede et al. (1986) calculated average lifetime risks of 2×10^{-3} on the basis of monitoring of four homes in the Netherlands. Risks at the 98th percentile from 45 selected indoor pollutants are on the order of 10^{-2}. This study used risk estimates based on "human, animal data, and analogy"; it is difficult to assess how these potencies compare to CAG estimates.

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CATEGORY 1

#4: Consumer Product Exposure (tied with Indoor Air Pollution)

RROBLEM DEFINITION

This problem area includes risks that are incurred as a result of the direct exposure of users to chemical substances in consumer products. There are over 10,000 chemical substances in consumer products. These substances are present in two categories of products: (1) in formulations and mixtures of various types (paints, solvents, glues, detergents, polishes, deoderizers, etc.), and in (2) manufactured articles (clothing, housewares, batteries, etc). While exposure to the chemicals in manufactured articles is usually limited, experience has shown that potential risks can sometimes be significant -- e.g., TRIS used on pajamas, friable asbestos in electrical products, DEHP in plastic articles mouthed by young children, and formaldehyde emissions from pressed-wood products.

This environmental problem area does not include the exposure of the general public that results from substances released from the product into the environment and transported beyond the immediate vicinity of the user-- e.g., contamination of drinking water or nonpoint source air pollution. Where consumer products, used indoors, contaminate the indoor air, any resulting risks are likely to be double counted as a consumer product risk and as an indoor air problem.

EPA'S RESPONSIBILITY TOWARD PROTECTING CONSUMERS

The Consumer Product Safety Commission has the major federal responsibility for ensuring that chemicals used in consumer products do not present health risks. However, EPA has been given several responsibilities related to identifying and reducing risks in consumer products. The Toxic Substances Control Act (TSCA) requires EPA to consider the full exposure to chemicals in regulating under the Act. Under Section 4 of TSCA EPA must identify which chemicals should be tested for potential toxicity (including carcinogenicity) and require industry to test them. A number of Test Rules published by EPA have been based, in whole or in part, on the concern for consumer risks.

EPA's proposed ban of certain asbestos products and phase-down of the manufacture and import of asbestos was based on concern for the life cycle exposure (including consumer exposure) to asbestos. EPA's priority review of methylene chloride under 4(f) of TSCA was based in large part on the potential carcinogenic risks presented to users of paint strippers containing this substance.

POLLUTANTS ADDRESSED IN THIS SUMMARY

Criteria for Selection of Consumer Hazards

Because of market pressures, carcinogenic substances in consumer products are often replaced by substitutes. However, the carcinogenic potential of these substitutes is often not well characterized.

To illustrate the risks consumer products can present, three substances were chosen for which information on their potential carcinogenicity is relatively recent: methylene chloride, para-dichlorobenzene, and formaldehyde. Asbestos was also included as there is a considerable amount of information available on consumer exposure to this carcinogen.

Extent to Which Risks of Selected Chemicals Represent Total Cancer Risks to Consumers

The potential exposure of consumers to chemical substances in consumer products is relatively high in comparison to typical environmental exposures. Numerous household products contain a great number of chemical substances. For example, hundreds of chemical constituents may be present in paints, solvents, thinners, and other related products. These products are typically used indoors, and are assumed to be safe by their users. Little effort is typically made to avoid or mitigate exposure to them.

While relatively few chemicals with established carcinogenic potential are found in these products, many of their consituents are in similar chemical/structural classes, but have not been tested in long-term animal bioassays. Because of the number of users of such products and the magnitude of exposures, the risks of the few selected substances presented here are probably only a fraction of those that are likely to be identified as a result of future testing.

RISK ASSESSMENT METHODOLOGY

Cancer Potencies

Cancer potencies (unit risks) used to derive estimates of individual risk and expected annual incidence of cancers are consistent with those developed by the Carcinogen Assessment Group. For methylene chloride, the unit risk estimate was derived from the risk assessment conducted by the Office of Toxic Substances. The assessment is currently being reevaluated by EPA in order to review additional data and a number of issues raised by industry.

Exposure Assessment

The exposure-related information used to evaluate consumer risks includes estimates of both the number of individuals exposed to the substance and the magnitude of the individual exposure. The magnitude of exposure involves estimates of concentrations of the chemicals in the air and statistics on both the frequency of use and the duration of exposure anticipated for each use. Estimates of air concentration are based on both monitoring data and an indoor air model.

The consumer exposure estimates used in these analyses reflect a number of uncertainties, stemming largely from lack of current data on product composition and use patterns. The models used to predict concentrations typically rely on assumptions on the details of the indoor environment.

ESTIMATED RISK

The estimates of risks to consumers from the four substances are based on the exposure estimates and unit risk estimates contained in agency risk assessment documents. These are presented below.

Substance	Estimated Annual Cancer Incidence
Formaldehyde	50
Methylene Chloride	30
Asbestos	5
para-Dichlorobenzene	9-50

CATEGORY 1

#6: Hazardous/Toxic Air Pollutants

PROBLEM DEFINITION

Approximately 60,000 to 70,000 known chemicals are in commerce today in the United States. An additional large number of chemicals are formed in chemical processes and by atmospheric reaction processes. A substantial portion of these substances can become airborne and, when inhaled by humans in sufficient concentrations, can be harmful to their health.

Toxic air pollutants are emitted from a wide variety of stationary and mobile sources. Obvious sources are large industrial facilities, combustion sources of various types and sizes, and motor vehicles. Some less traditional but equally important sources include commercial facilities that use solvents and facilities that treat, store, and dispose of hazardous wastes. While higher concentrations of toxic air pollutants are generally found in large urban or industrialized areas, the limited available ambient monitoring data indicate large variations within and between urbanized areas.

This report merges the terms "hazardous air pollutant" and "toxic air pollutant" to comprise a general category of particulate and gaseous pollutants in the ambient air. Strictly speaking, the hazardous air pollutants are pollutants that meet the statutory requirements of section 112 of the Clean Air Act. This section requires the EPA Administrator to establish emission standards for pollutants that "...cause, or contribute to, air pollution which may reasonably be anticipated to result in serious irreversible, or incapacitating reversible, illness...." "Toxic air pollutant" is a more general term for any airborne chemical substance that may pose a human health threat.

This problem area considers only routine, continuous emissions of pollutants. Further, it assumes that exposure to toxic air pollutants occurs only outdoors on a continuous basis for a lifetime (70 years). This assessment also does not consider the potential role of toxic air pollutants, such as carbon tetrachloride or chlorofluorocarbons in the depletion of stratospheric ozone. Both Indoor Air and Depletion of Stratospheric Ozone are considered under their own environmental problem areas.

POLLUTANTS ADDRESSED IN THIS SUMMARY

Criteria for Selection of Hazardous/Toxic Air Pollutants

Table 1 shows the toxic air pollutants selected for assessment. The initial procedures that the Office of Air Quality Planning and Standards (OAQPS) followed for setting priorities identified many hundreds of potentially toxic air pollutants. After reviewing available health literature and source information, OAQPS selected approximately 50 chemicals, many of which were potent carcinogens with the potential for significant human exposures. OAQPS then conducted more detailed analyses and developed estimates of cancer risk. Only those pollutants with an estimated cancer incidence in excess of one per year were retained for this analysis. As a result, this analysis estimates the cancer risk from 20 substances, or categories of substances.

Legislatively excluded from section 112 and this problem area are the six so-called criteria air pollutants (particulate matter, ozone, carbon monoxide, sulfur oxides, nitrogen dioxide, and lead). These pollutants are regulated under sections 108-110 of the Clean Air Act and are treated under the Criteria Air Pollutant environmental problem. Although toxic air pollutants and the criteria pollutants are classified separately, certain toxic air pollutants are constituents of particulate matter, and some are volatile organic compounds that may contribute to the formation of ozone.

RISK ASSESSMENT METHODOLOGY

Cancer Potencies

Most of the carcinogenic potency estimates for the pollutants listed in Table 1 were developed by EPA's Carcinogen Assessment Group (CAG). The potency estimates for some of the toxic air pollutants warrant special comment.

- The inhalation unit risk estimate for asbestos was derived by OAQPS staff, based on potency estimates developed by the Office of Health and Environmental Assessment (U.S. EPA, 1985a).

- The potency of the mixture of polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans was based on the CAG unit risk estimate for 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). The mixture was converted to TCDD equivalents, based on the relative potency of compounds in the mixture compared to the potency of TCDD (U.S. EPA, 1986a).

- The potency estimate for formaldehyde was based on the unit risk estimate for malignant tumor formation, rather than on total tumors (U.S. EPA, 1986b).

- The unit risk estimate for products of incomplete combustion (PIC) was derived from dose-response data that use benzo[a]pyrene (BaP) levels as a surrogate for this large category of BaP-related pollutants (U.S. EPA, 1985b).

- The potency of emissions from hazardous waste treatment, storage, and disposal facilities (TSDFs) was based on the unit risk estimate for carbon tetrachloride. It was assumed that the potency of the carcinogenic fraction of TSDF emissions may be as low as that of methylene chloride or as high as carbon tetrachloride. The unit risk estimate for carbon tetrachloride was used to estimate the upper end of a range of preliminary risk estimates (U.S. EPA, 1985c).

Exposure Assessment

Exposure assessments estimate concentrations of a pollutant and the numbers of people exposed to these concentrations. Two approaches for determining ambient air concentrations in the vicinity of emission sources are the use of monitoring data, which are direct measurements of pollutant concentrations in the ambient air, and the use of emission estimates coupled with dispersion modeling to predict concentrations to which humans are exposed.

Use of Monitoring Data

Monitoring data were used to estimate at least a portion of the risk associated with exposure to carbon tetrachloride, formaldehyde, products of incomplete combustion, and vinyl chloride. Using direct measurements of ambient concentrations to estimate risk avoids the problems of incomplete emission inventories, incomplete knowledge on current control status, a lack of knowledge concerning pollutants formed or destroyed in the atmosphere, as well as the errors associated with dispersion modeling. There is, however, significant potential for error in using monitoring data to estimate risk.

For example, monitoring data are usually available for a relatively small number of areas for each pollutant, so certain assumptions must be made in extending these data to the rest of the nation. Further, monitors are often located away from major sources, and the air quality data are subject to errors in sampling and analytical methods. Finally, ambient concentrations measured over a period of at least a year should be used in the estimation of the long-term exposure concentrations that are associated with the development of cancer, but ambient data are rarely collected for an entire year.

Use of Emission Estimates

To predict human exposure to a pollutant using dispersion modeling techniques, one must first estimate the emissions from a source. Emission estimates for point sources are located at a specific site, whereas estimates for area and mobile sources are apportioned over the entire area being considered. Sources of information for emission estimates include data gathered from industry under the authority of the Clean Air Act (section 114) or other regulatory authorities, material balance calculations, published information on emission factors, and actual measurements of emission rates. Given the diverse nature of the sources of information, the quality of the estimates may vary considerably.

Emission estimates for benzene, 1,3-butadiene, and formaldehyde in automobile exhaust were determined using the Mobile Sources Emissions Model (Mobile3) developed by the Office of Mobile Sources (US EPA, 1984). Mobile3 is a computer program that calculates emission factors for hydrocarbons (HC), carbon monoxide (CO), and oxides of nitrogen (NOx) from highway motor vehicles. Mobile3 calculates emission factors for eight individual vehicle types in two regions of the country. Emission estimates generated by Mobile3 depend on a variety of conditions, such as ambient temperature, speed, and mileage accrual rates.

Use of Dispersion/Exposure Modelling

Developed by OAQPS, the Human Exposure Model (HEM) was used to estimate human exposure for most of the pollutants in Table 1 (US EPA, 1986c). The HEM estimates the population exposed to various concentrations of air pollutants emitted from point and area sources and the carcinogenic risk associated with this exposure. The HEM consists of (1) an atmospheric dispersion model, (2) population distribution information based on 1980 Bureau of the Census data, and (3) a procedure for estimating risks due to the predicted exposure. The inputs needed to operate this model are such source data as emission rate, plant location, height of the emission release point, and the temperature of the off gases.

The model estimates the magnitude and distribution of ambient air concentrations of the pollutant at distances of 0.2 km to 50 km from the source. These concentration estimates are coupled with the population to estimate public exposure to the pollutant. The HEM then predicts population and individual lifetime risks if a unit risk number determined from health data is used as input for the pollutant.

The model relies on information provided in a data base developed by the U.S. Census Bureau. The HEM contains several simplifying assumptions and uncertainties, some of which may contribute to either an over- or underestimation of the health risk. The model assumes that most exposure occurs at populationweighted centers (centroids) of block group or enumeration districts (the locations of actual residences are not known), that people reside at these centroids for their entire lifetimes (assumed to be 70 years for calculating cancer risk), that no net population migration or growth occurs, that indoor concentrations of pollutants emitted from the sources being studied are assumed to be the same as outdoor concentrations, that plants emit pollutants at an average emission rate for 70 years, that the only source of exposure is the direct inhalation of ambient air (resuspension of pollutants via dust is not considered), and that all terrain is flat.

There are several areas of uncertainty associated with the input required for running the HEM. The meteorological data set selected for each facility may not be representative of the actual conditions at that site. There is often considerable uncertainty associated with the emission estimates and the plant parameters used to characterize the emission source. Finally, if the plant is not correctly located in the model calculation, there will be a problem matching population census data with concentrations.

Estimation of Risk

To estimate the annual cancer incidence associated with exposure to the toxic air pollutants in Table 1, the following equation was used:

$$Ia = \frac{\sum_{i}^{n} (Ci \times Pc_i) \times U.R.}{70},$$

where Ia = annual incidence;

C = ambient air concentration at a specific location (modeled annual concentration or monitoring data);

Pc = population exposed to a specific concentration (c); and

U.R. = cancer unit risk estimate for lifetime exposure (70 years).

A second measure of risk -- maximum individual lifetime risk -- was calculated to estimate the highest probability that an individual will develop cancer from ambient exposure to a pollutant over a 70-year period. This

was done by multiplying the unit risk estimate by the highest average annual ambient air concentration of a pollutant (usually nearest to a source) for all sources modeled.

ESTIMATED RISKS

The estimated number of people subject to the maximum individual risks for each pollutant in Table 1 varies considerably. This estimate of risk is best used along with the annual incidence estimate as a relative indicator of the risk that may be associated with exposure to a pollutant, rather than as an absolute measure of maximum risk.

As shown in Table 1, the estimated annual population risk or incidence of cancer for the toxic air pollutants in this analysis totaled 2,054. The maximum individual lifetime risks ranged from 2.4 out of 10 (2.4×10^{-1}) to 8.1 out of 100,000 (8.1×10^{-5}), with many maximum risks estimated to be in the 10^{-2} and 10^{-3} ranges. As an example for translating these figures, the maximum individual lifetime risk estimate of 1.1×10^{-2} for chloroform implies that one out of 100 people near a particular point source, breathing a given concentration of chloroform for 70 years, will develop cancer as a result of that exposure.

The exposure and risk assessments for most of these pollutants are continually being reviewed and updated. As such, the risk estimates presented in Table 1, although subject to change, reflect the most current estimates.

UNCERTAINTIES AND CAVEATS

Following are some of the important caveats surrounding this analysis:

- Only routine releases are considered in this section. Accidental releases are discussed under a separate environmental problem area.

- Quantitative risk estimates were generated for only 20 substances or categories of substances. Major factors preventing analysis of more pollutants were the limitations of exposure data and the lack of quantitative estimates of cancer potencies. As such, the risks outlined in Table 1 represent less than 100 percent of the total risk.

- No consideration is given to the possible synergistic or antagonistic effects of exposure to mixtures of chemicals. Urban air is characterized by the presence of hundreds of pollutants, and little is known about how these substances may interact once they enter the human body.

- Estimates of cancer risk for each pollutant in this analysis may change as more detailed analyses are conducted and as the risk assessment methodologies are refined.

- Atmospheric transformation of toxic air pollutants was not explicitly considered.

- The potency and exposure estimates used to estimate both annual population and individual lifetime risk assume a 70-year exposure to the compound in question. Obviously, people spend a substantial portion of their lives indoors in environments where exposures to toxic pollutants can be higher, lower, or similar to those that occur outdoors.

- Exposure estimation techniques employ several simplifying assumptions that may over- or underestimate exposure.

- Ambient air quality data, rather than modeled levels estimated by dispersion modeling, were used to estimate exposure concentrations for several compounds.

TABLE 1

ESTIMATED NATIONWIDE RISKS FROM HAZARDOUS/TOXIC AIR POLLUTANTS

Pollutant	Estimated Annual Incidence	Maximum Individual Lifetime Risk
Arsenic (inorganic)	2	1.9×10^{-3}
Asbestos	82	2.3×10^{-3}
Benzene	90[a]	1.5×10^{-3}
1,3-butadiene	223[a]	2.4×10^{-1}
Cadmium	7	2.8×10^{-3}
Carbon tetrachloride	69	5.7×10^{-3}
Chloroform	10	1.1×10^{-2}
Chromium (VI)	75	8.7×10^{-3}
Coke oven emissions	7	3.4×10^{-2}
1,2-Dichloroethane (EDC)	4	1.2×10^{-2}
Dioxins/dibenzofurans	10	1.4×10^{-3}
Ethylene oxide	58	1.9×10^{-3}
Formaldehyde	435[a]	1.6×10^{-4}
Gasoline vapors (marketing)	77	5.7×10^{-3}
Methylene chloride	35	2.8×10^{-3}
Products of incomplete combustion (PIC)	610[a,b]	-
Tetrachloroethylene	5	1.3×10^{-4}
Trichloroethylene	4	8.1×10^{-5}
Vinyl chloride	11	3.5×10^{-3}
TSDFc emissions	240	-
Total	2054	-

NOTE: Annual incidence and maximum individual risk should be regarded as rough estimates due to uncertainties in the unit risk values and exposure assessments. These estimates are subject to change. Annual incidence is rounded to the nearest whole number.

a Includes contribution from mobile sources. The Office of Mobile Sources is currently revising its component of the risk estimates for some of these pollutants.

b Potency estimate and annual incidence for PIC taken from US EPA, 1985a. These estimates were based on the use of benzo[a]pyrene as a surrogate for PIC.

c TSDF = hazardous waste treatment, storage, and disposal facilities.

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CATEGORY 2

#7: Stratospheric Ozone Depletion

PROBLEM DEFINITION

By preventing most potentially harmful ultraviolet radiation (UV-B) from penetrating to the earth's surface, the ozone layer acts as an important shield protecting human health, welfare, and the environment. The possibility that the production, use, and release of chlorofluorocarbons (CFCs) could deplete stratospheric ozone was first theorized in a 1974 article in Nature by Molina and Rowland.

The major consequence of ozone depletion would be an increase in harmful UV-B radiation, particularly that in the more damaging region of the UV spectrum. Under current atmospheric conditions, the latitudinal exposure to UV-B varies greatly, with more UV-B received by those closer to the equator. This variability in exposure takes place largely because of changes in the solar angle and a natural latitudinal gradient in ozone thickness.

On the basis of both epidemiological studies that relate the natural variation in UV-B exposure to skin cancer incidence and laboratory studies in which tumors have been induced and promoted by UV-B, researchers have conclusively demonstrated that both basal and squamous skin cancers are associated with cumulative exposure to UV-B (NAS, 1984). While infrequently fatal (somewhat less than 1 percent of cases currently result in fatalities), these are the two most common types of skin cancer, with approximately 500,000 cases per year (Scotto, 1986). A relatively good understanding exists of these cancers, with a 1 percent ozone depletion projected to increase basal skin cancer by 1 to 3 percent and squamous skin cancer by 2 to 5 percent (U.S. EPA, 1986, based on data in Scotto, 1986).

Melanoma is a less common but far more deadly type of skin cancer. In 1985, there were about 25,000 cases and 5,000 fatalities in the U.S. (Scotto, 1986). Recent studies of melanoma have tended to reinforce the hypothesis that UV-B is one of the causes of melanoma, although the relationship appears much more complex, and is perhaps related to peak and possibly youthful exposure (EPA, 1986). Recent efforts to quantify this relationship have produced the following dose-response relationship: a 1 percent ozone depletion is projected to increase melanoma incidence by 1 to 2 percent and melanoma fatalities by 0.8 to 1.5 percent (EPA, 1986, based on data in Scotto, 1986).

POLLUTANTS ADDRESSED IN THIS SUMMARY

The likelihood of ozone depletion will be influenced by the future concentrations of many trace gases. Aside from trace gases that have de minimis effects, potential ozone depleters include chlorofluorocarbons (CFC 11,12,113, 22, and Halon 1211); Halon 1301 (not a chlorofluorocarbon); and chlorocarbons (CCl_4 and CH_3CCl_3). Trace gases that may buffer ozone depletion include CO_2, CH_4, and, in some cases, N_2O.

Part B of the Clean Air Act charges EPA to consider "any substance" that may affect the ozone layer." EPA's Integrated Assessment Model was therefore

developed to assess the atmospheric effects of all trace gases likely to affect the stratosphere. The model incorporates each of the trace gases listed above.

RISK ASSESSMENT METHODOLOGY

EPA's Science Advisory Board is currently completing its review of the methodologies and results presented in this summary. Both the approaches and results are subject to revision.

Dose-Response Relationships

A strong consensus exists on the role of UV-B in the induction and promotion of non-melanoma skin cancer (basal and squamous). Dose-response relationships for basal and squamous skin cancer incidence are based on the results of epidemiological investigations undertaken by the National Cancer Institute (NCI)(Scotto, Fears, and Fraumeni, 1981). The relationships currently included in the model are power functions that express the change in incidence as a percentage change that can be multiplied by the baseline incidence to compute the increased age-specific incidence. An advantage of this formulation is that the change in exposure can be expressed as a change relative to baseline, thereby avoiding potential difficulties in specifying the absolute levels of changes in exposure to UV. The coefficients used in the models were derived from the regression coefficients presented in Scotto, Fears, and Fraumeni (1981). Results presented in the draft risk assessment are based on a measure of UV where wavebands are weighted by a DNA action spectrum.

A separate dose-response relationship for mortality from non-melanoma skin cancer has not been estimated. For purposes of modeling, the current relationship between mortality and non-melanoma incidence are assumed to apply.

The role of UV-B in melanoma skin cancer is more uncertain. The methodologies used to relate UV-B to melanoma were developed for use in the Integrated Analysis Model based on an extensive review of the literature and data analysis. The review was conducted with the cooperation and participation of other EPA offices (Carcinogen Assessment Group and Office of Standards and Regulations), government agencies (NCI and NASA), and academics (from Johns Hopkins University, University of South Carolina, and University of Cincinnati). The work is currently under review by EPA's Science Advisory Board.

The dose-response models for melanoma incidence are of the same form as for non-melanoma. The coefficients were derived from data presented in Scotto and Fears (in press) and are are assumed to be applicable for the DNA action spectrum.

A separate analysis of melanoma mortality has been performed by Pitcher (in press). The coefficients estimated as the result of that investigation are used to model the potential increase in mortality due to melanoma skin cancer. The coefficients are estimated using peak UV values, weighted by the DNA action spectrum.

Exposure Assessment

Projections of future emissions of potential ozone-depleting compounds were developed based on a range of long-term estimates prepared for EPA/ United Nations Environment Programme workshops on future demand. In the short term, the scenarios incorporate market-based analysis. For the long term, they are based on the application of historical relationships between production and economic and population growth. Estimates for compounds that may buffer depletion were based on a review of independent projections.

The effects of trace gas emissions on the stratospheric ozone layer are modeled based on a parameterized version of a state of the art, one-dimensional model (Connell, 1986). Consequent changes in UV are modeled based on a model of ozone and UV radiation developed in conjunction with NASA and National Oceanic and Atmospheric Administration.

Because the risks of skin cancer vary with race, age, and sex, the population at risk was characterized by these variables using Census Bureau data. To reflect differences in current UV exposure, the assessment split the population into three geographic regions. For each region, the current and expected size and age distribution were projected to the year 2000, based on estimates of migration and birth/death rates. After 2000, these distributions were held constant. Aggregate increases in population were based on estimates prepared by Keyfritz and used in previous EPA studies (Seidel and Keyes, 1983).

Estimation of Risk

Using the risk assessment methodology discussed above, estimates of UV-B effects on basal, squamous, and melanoma skin cancer were presented in the draft risk assessment, An Assessment of the Risks of Stratospheric Modification. For the central case scenario of ozone depletion, changes in non-melanoma and melanoma skin cancer for three cohorts of individulals alive today or born by the year 2074 are shown in Table 1. On the basis of this table, approximately 10,000 additional cancer cases annually will be attributable to ozone depletion by the year 2100. The sensitivity of these estimates to trace gas emission scenarios is shown in Table 2. The sensitivity to dose-response coefficients is shown in Table 3.

OVERALL ASSESSMENT OF UNCERTAINTY

A strong consensus exists that UV-B radiation induces and promotes basal and squamous skin cancer. The model and coefficients used in this analysis are within the range of reported results.

The effect of UV-B on melanoma is much more uncertain. The theories, methodologies, and data used in this analysis have been preliminarily reviewed by the Science Advisory Board.

For projections of future ozone depletion, the largest quantitative uncertainties involve assumptions concerning future emissions of CFCs and other trace gases. With respect to modeling the atmospheric consequences of trace gas growth, there exists the possibility that some overlooked or missing factor or oversimplified process has lead to over- or underpredictions

of changes in ozone. Because of these and other uncertainties complicating the evaluation of this problem area, we ranked it lower than the analytical results would indicate.

TABLE 1

Human Health Effects: Central Case
Additional Cumulative Cases and Deaths by Population Cohort

HEALTH EFFECTS	POPULATION ALIVE TODAY[a]	NUMBERS BORN 1985-2029[b]	NUMBERS BORN 2030-2074[c]
Non-Melanoma Skin Tumors			
Additional Basal Cases	630,600	5,012,900	17,630,500
Additional Squmaous	386,900	3,185,800	12,122,400
Additional Deaths	16,500	135,000	509,300
Melanoma Skin Tumors			
Additional Cases	12,300	109,800	430,500
Additional Deaths	3,900	32,200	115,100

a/ Analysis period for health effects: 1985-2074.

b/ Analysis period for health effects: 1985-2118.

c/ Analysis period for health effects: 2030-2164.

SOURCE: U.S. EPA (1986), An Assessment of the Risks of Stratospheric Modification, draft report. Washington, D.C.

TABLE 2

Human Health Effects: Emission Scenarios
Additional Cumulative Cases and Deaths Over Lifetimes of People in the U.S
Alive Today and Born in the Next 88 Years

HEALTH EFFECTS	EMISSIONS SCENARIOS			EXTREME CASES	
	Low	Central	High	Lowest	Highest
Non-Melanoma Skin Tumors					
Additional Basal Cases	1,599,500	23,274,000	83,755,700	-1,616,100	135,317,800
Additional Squamous Cases	823,400	15,695,100	71,808,100	-856,600	117,809,809
Additional Deaths	35,700	660,800	2,952,400	-36,700	4,837,400
Melanoma Skin Tumors					
Additional Cases	47,300	552,600	1,897,400	-40,500	3,079,500
Additional Deaths	12,100	151,200	502,800	-11,200	809,700

SOURCE: U.S. EPA (1986), An Assessment of the Risks of Stratospheric Modification, draft report. Washington, D.C.

TABLE 3

Human Health Effects: Sensitivity to Dose-Response Relationship
Additional Cumulative Cases and Deaths Over Lifetimes of People
in U.S. Alive Today and Born in Next 88 Years

HEALTH EFFECTS	SENSITIVITY OF EFFECT TO UV DOSE Low	Central	High
Non-Melanoma Skin Tumors			
Additional Basal Cases	14,046,400	23,274,000	34,130,500
Additional Squamous Cases	9,242,000	15,695,100	24,385,300
Additional Deaths	109,200	660,800	10,203,000
Melanoma Skin Tumors			
Additional Cases	384,300	552,600	732,100
Additional Deaths	134,300	151,200	168,500

SOURCE: U.S. EPA (1986), An Assessment of the Risks of Stratospheric Modification, draft report. Washington, D.C.

REFERENCES

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CATEGORY 2

#8: Hazardous Waste Sites - Inactive

PROBLEM DEFINITION

Although a wide variety of sites are covered under this subject, all are characterized by the presence of hazardous waste at the site and the absence of any current manufacturing, treatment, storage, or disposal activity at the site. This category includes inactive hazardous waste sites covered under the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA, known as Superfund) and the National Oil and Hazardous Substances Pollution Contingency Plan (NCP), which provides procedures for addressing the problem of inactive hazardous waste sites under CERCLA.

The NCP establishes a system for ranking the degree of hazard at sites identified by the Comprehensive Environmental Response, Compensation, and Liability Inventory System (CERCLIS), the master list of inactive hazardous waste sites that have been reported to EPA. These sites are found throughout the country and vary from mine tailings to pesticide spills to abandoned manufacturing facilities. The sites are investigated and ranked, and the ones presenting the most significant hazards are placed on the National Priorities List (NPL) and become eligible for money from the Superfund for remedial action to reduce the hazards.

Some sites do not meet the criteria for inclusion on the NPL, but may pose some hazard nonetheless. The cancer work group has included non-NPL sites in its extrapolation to the universe of potential inactive hazardous waste sites.

A wide variety of exposures are possible from hazardous waste sites. These include exposure to volatilized contaminants in air, inhalation of wind-entrained dust, inadvertent soil ingestion, dermal absorption from soil, contact and ingestion of surface water contaminated by leaching or running off from sites, and contaminants leaching into ground water. Exposure to surface or ground water can include dermal contact and inhalation of volatile organics, as well as ingestion of drinking water. Out of the wide array of possible exposure pathways, ingestion of contaminated ground water poses the overall highest individual and population risks for the sites included in this analysis.

Because consideration of cumulative risks due to all exposure routes would be very difficult, and because much less information is available for the other routes, only risks due to ground-water ingestion were considered for this problem area. This simplification, however, did not significantly distort estimated population risk because ingestion of ground water probably captures the majority of population cancer risk at the sites. This is not necessarily the case for individual risks.

POLLUTANTS ADDRESSED IN THIS SUMMARY

Initially, information on ground-water concentrations and carcinogenic risk was abstracted for all chemicals assessed at any of the 35 Superfund sites investigated in either public health evaluation or feasibility studies. From this list, six chemicals were taken as representative: arsenic, vinyl chloride, tetrachloroethylene, trichloroethylene, 1,2-dichloroethane, and benzene. Generally, these chemicals were associated with the highest concentrations, the highest individual risk, and high frequency of occurrence.

RISK ASSESSMENT METHODOLOGY

Cancer Potencies

The cancer potencies used in this section were all derived by the Carcinogen Assessment Group. They are subject to the interpretations and caveats to which all such values are subject.

Exposure Assessment

Approach and Data Sources

The basic analytical approach consisted of four steps: (1) estimate the mean individual cancer risk for each chemical, based on available risk assessment documents; (2) estimate the number of people exposed per site; (3) estimate the total number of sites; and (4) multiply individual risk times population times number of sites.

Baseline public health evaluations or endangerment assessments performed as part of the Remedial Investigation/Feasibility Study process for Superfund sites were the source of data used for estimation of individual cancer risk at uncontrolled hazardous waste sites. These assessments usually evaluate the site under an assumption of no remedial action. Thirty-five risk assessment or feasibility study documents for Superfund sites in several EPA regions were reviewed. Of these, 20 contained quantitative estimates of cancer risk via ground-water ingestion, and individual risk data were extracted from them. Ground-water exposure pathways were the focal point for analysis because most data were available for them.

Risk estimates were abstracted for a maximum of five carcinogens at each site (usually fewer than five carcinogens were evaluated in the assessments reviewed). Both maximum risk and average risk estimates were generally presented and abstracted in the assessments. The following six frequently occurring high-risk potential carcinogens were identified: arsenic, vinyl chloride, tetrachloroethylene, trichloroethylene, 1,2-dichloroethane, and benzene. The reported chemical-specific cancer risks were averaged across the sites at which that chemical was reported. This was combined with frequency of detection in samples from sites analyzed by the Contract Lab Program (CLP) to extrapolate to expected national cancer risks from inactive hazardous waste sites.

Extrapolation to Human Population

Population estimates were determined by estimating the number of inactive hazardous waste sites, and then multiplying it times the average number of people exposed at each site. The total number of sites was determined by combining the total expected number of NPL sites (1,800) with non-NPL inactive hazardous waste sites. (U.S. EPA, 1984) This non-NPL number was estimated from the projected CERCLIS site inventory of 25,000, minus the 1,800 NPL sites, and assuming that site investigations will find that two-thirds show no risk, which has been program experience. (U.S. EPA, 1984)

The exposed population at sites was based on statistics in the Hazard Ranking System data base. The mean number of ground-water users within three miles of NPL sites is 11,773. We assume that 10 percent are potentially affected by site contamination, because many ground-water users within three miles may be upgradient, too far away, or not hydraulically connected to site contamination. We further assume that the population exposed at non-NPL sites is half that at NPL sites, because of the population bias in getting listed on the NPL. Consequently, the total affected population (via ground water) from all inactive hazardous waste sites is estimated to be 6.8 million. This number was combined with chemical-specific individual cancer risks to estimate excess cancer risks on a per chemical basis.

Estimates of the concentrations of carcinogens to which individuals are exposed were based on a distribution of wells within three miles of the sites. Actual concentrations at these wells were used, rather than on-site concentrations.

Estimated Risks

Tables 1 through 3 summarize data from the public health evaluations and feasibility studies. Table 1 presents all chemicals reported in more than one of the public health evaluations or feasibility studies that were investigated for this analysis, with maximum and best-guess concentration data and risk predictions averaged for all sites having that chemical. Frequency of appearance in Public Health Evaluations (PHEs) refers to the number of times the chemical was found in the PHEs of the 35 sites investigated. These values were used as the basis for selecting the the six chemicals of concern. Percent frequency at sites from CLP samples refers to the percentage of sites whose samples have been analyzed by the CLP as part of the remedial investigation for the Superfund program at which a chemical was found. These values were used to estimate the number of sites at which a chemical may be of concern. The highest average individual risk was for vinyl chloride, with a maximum excess individual cancer risk of 0.2, and a best-guess cancer risk of 0.1.

Table 2 presents annual excess cancer cases estimated for the top six chemicals from Table 1. These estimates assume an exposed population of 1,173 at each site, as determined by the methodology above. These estimates refer only to the 35 sites investigated for the analysis, and not to the universe of all inactive hazardous waste sites. Vinyl chloride was associated with the highest number of excess cancer cases, with 30 and 15 excess cases for maximum and best-guess risks, respectively.

Table 3 presents annualized excess cancer cases for the top six chemicals, extrapolated to the entire potential universe of inactive hazardous waste sites. These values represent the estimated number of excess cancer cases among the estimated exposed population of 6.8 million, as determined by the methodology above. Maximum excess cancer cases per year for all sites range from 1,300 for vinyl chloride to 9 for benzene. Best-guess values range from 650 for vinyl chloride to 0.9 for benzene.

UNCERTAINTIES AND CAVEATS

This analysis attempts to capture the most significant risks from Superfund sites. Because this analysis is limited to only six of the literally hundreds of chemicals found at inactive hazardous waste sites, it may underestimate actual total risk from these sites. However, field experience at sites indicates that the majority of risks at most sites is due to a few chemicals.

In addition, only one of many possible exposure pathways has been analyzed. And, while it may be the most significant pathway, several sites used in this analysis did not have any reported exposure to ground water. On the other hand, the estimates of affected sites and exposed population may overestimate the actual number of cancer cases in the population.

Many assumptions are built into the numbers used from other sources and are inherent in these extrapolation methods. Taken as a whole, these assumptions place severe limitations on the accuracy of the projections. Major assumptions include the following:

- Site-specific risk assessment reports are both comprehensive and accurate. In some cases these were small-budget assessments based on very limited site data. Therefore, the uncertainty in the individual risk numbers is high. In general, these numbers are probably conservative because of conservative assumptions in the exposure and toxicity assessments (e.g., use of future-use scenarios, full lifetime exposure, upper-bound potency estimates) on which the risk analyses are based.

- Individual risk distributions and mean risks derived from the 35 sample sites adequately represent the overall population of thousands of uncontrolled waste sites. This is a very small sample size, and it is highly unlikely to be representative, given the great variability in uncontrolled waste site conditions and the sample site selection approach. (The only criterion for inclusion was ready availability of a risk assessment report.) This assumption also implies that non-NPL sites have comparable risk levels to NPL sites because all sample sites examined were NPL sites.

- There will be no interventions to eliminate or reduce risk at these sites.

TABLE 1

Potential Carcinogens That Are Found in Ground Water and That Appear in More Than One Superfund Public Health Evaluation/Feasibility Study

Chemicals	Frequency of Appearance in PHEs 1/	Percent Frequency at Sites (from CLP)	Mean Maximum Concentration 2/ (ug/l)	Mean Maximum Individual Risk 3/	Mean Best-Guess Concentration 4/ ug/l	Mean Best-Guess Individual Risk 5/
Trichloroethylene	10	27.9	40,000	2×10^{-2}	20,000	1×10^{-2}
Vinyl Chloride	9	6.8	5,500	2×10^{-1}	1,400	1×10^{-1}
Benzene	9	30.4	1,622	3×10^{-4}	280	3×10^{-5}
Tetrachloroethylene	6	22.6	12,000	2×10^{-2}	1,050	1×10^{-3}
Arsenic	4	40.8	95	2×10^{-2}	9.8	4×10^{-3}
1,2-Dichloroethane	4	9.4	9,000	2×10^{-2}	81	2×10^{-3}
Chloroform	3	27.8	2,800	7×10^{-3}	98	2×10^{-4}
PCBs	3	8.1	27	3×10^{-3}	4	1×10^{-3}
1,1-Dichloroethene	3	12.9	72	2×10^{-4}	5.6	2×10^{-5}
Methylene Chloride	2	76.7	1.2×10^{6}	4×10^{-2}	53,000	8×10^{-4}
1,1,2-Trichloroethane	2	4.4	1,500	3×10^{-3}	23	3×10^{-4}
N-nitrosodiphenylamine	2	8.9	90	9×10^{-7}	--	8×10^{-7}

1/ Out of a total of 35 sites.

2/ Data from 20 NPL sites. Averages are calculated with data from sites having that chemical reported in public health evaluations only.

3/ Data from 17 NPL sites. Averages are calculated with data from sites having that chemical reported in public health evaluations only.

4/ Data from 7 NPL sites. Averages are calculated with data from sites having that chemical reported in public health evaluations only.

5/ Data from 15 NPL sites. Averages are calculated with data from sites having that chemical reported in public health evaluations only.

TABLE 2

Projected Annual Excess Cancer Cases for Selected Chemicals at 35 Sites Having Public Health Evaluations[1/]

Chemicals	Maximum Excess Cancer Cases	Best-Guess Excess Cancer Cases
Trichloroethylene	4	2
Vinyl Chloride	30	15
Benzene	0.45	0.045
Tetrachloroethylene	2	0.1
Arsenic	1.3	0.3
1,2-Dichloroethane	1.3	0.13

1/ Assuming an average of 1,173 people exposed to ground water at each site. See text for method. Annual number of cases represents the lifetime number of projected cases divided by an average 70-year lifetime.

TABLE 3

Projected Annual Excess Cancer Cases for Selected Chemicals at the Entire Universe of Inactive Hazardous Waste Sites [1]

	Maximum Excess Cancer Cases	Best-Guess Excess Cancer Cases
Trichloroethylene	540	270
Vinyl Chloride	1,300	650
Benzene	9	0.9
Tetrachloroethylene	440	21
Arsenic	775	154
1,2-Dichloroethane	180	18

1/ Total population potentially exposed to inactive hazardous waste sites is estimated to be 6.8 million. See text for method. Annual number of cases represents the lifetime number of projected cases divided by an average 70 year lifetime.

- The number of actual uncontrolled sites will not increase significantly over that projected by EPA (i.e., almost all existing sites have already been discovered)(U.S. EPA, 1984).

- The mean exposed population via ground water contaminated by sites can be derived from the estimated number of ground-water users within three miles. On the basis of our past experience, we would not expect these numbers to correlate very well.

- The entire exposed population either is equally susceptible (i.e., no sensitivity variation), or the distribution of susceptibility is symmetric around the potency value used.

- There are no interactive effects (e.g., synergism) resulting from concurrent exposure to chemicals from sources other than uncontrolled waste sites, as well as several chemicals from one site.

- The distributions of individual risk and exposed population are independent (i.e., there is no association between population size and risk level). If these distributions are skewed (likely) and not independent (possible), the population risk estimates could be significantly in error.

Clearly, the number and nature of assumptions required to estimate population risks cast great doubt on the reliability of the numbers presented.

REFERENCE

U.S. EPA, Office of Solid Waste and Emergency Response. Extent of the Hazardous Release Problem and Future Funding Needs, CERCLA Section 301(a)(1)(C) Study. Final report. December 1984.

CATEGORY 2

#9: Drinking Water As It Arrives as the Tap

PROBLEM DEFINITION

Since 1975, EPA's Ofice of Drinking Water has conducted several national surveys. The 1975 National Organics Reconnaissance Survey and the 1975 National Organics Monitoring Survey primarily examined the presence of trihalomethanes in U.S. drinking water supplies. The 1977-81 National Screening Program and the 1978 Community Water Supply Survey have demonstrated the presence in surface waters of organic contaminants in drinking water, generally at levels lower than 10 micrograms per liter (ug/l).

The 1982 Ground Water Supply Survey (GWSS) examined approximately 1,000 drinking water supplies that used ground water as a source. Five hundred supplies were selected at random, and 500 were selected by states as having high potential for contamination by organic chemicals. Approximately 21 percent of the randomly selected systems had one or more volatile organic chemicals (VOCs) at detectable levels (primarily in the low ug/l range). Approximately 16 percent of the smaller systems (<10,000 people) in the random sample sample contained some concentrations of the VOCs at levels measurable at less than 1 ug/l, while approximately 28 percent of the large supplies (>10,000 people) contained these levels of VOCs. In the state-selected portion of the survey, higher frequencies of occurrence were found at all levels.

In addition, synthetic organic chemicals of industrial origin (including pesticides) have been detected with increasing frequency, especially in ground-water sources. Some surface waters are being contaminated with industrial and municipal wastes, although in many cases, application of pollution controls has apparently improved surface water quality in recent years. Contamination of surface water by other pesticides during runoff can be a significant problem in certain areas, such as in Ohio, where treated drinking water levels of locally used agricultural pesticides have been shown to parallel seasonal use.

POLLUTANTS ADDRESSED IN THIS SUMMARY

The Comparative Risk Project examined the pollutants that are the subject of regulation (see table). These pollutants are determined by the results of monitoring surveys, as well as by explicit direction given in the 1986 amendments to the Safe Drinking Water Act. They consist of a wide variety of volatile and synthetic organic contaminants, including pesticides; several organic chemicals falling under a group known as trihalomethanes; and several natural and man-made radionuclides, including radon. The regulations that will be proposed will eliminate most of the known risk of exposure to contaminated drinking water.

RISK ASSESSMENT METHODOLOGY

Evaluating the toxicology of potentially carcinogenic substances is a two-phase process. In the first phase, the toxicological data base for noncarcino-

genic toxic effects was evaluated in the same manner as that for noncarcinogens. This involves determining acceptable daily intakes, which are exposure levels that are estimated not to pose significant risk to humans when received daily over a lifetime.

In the second phase, an assessment is made of both the evidence of the carcinogenic potential of a substance (e.g., long-term bioassays in rodents and human epidemiology) and the information that provides indirect evidence (e.g., mutagenicity and other short-term test results). The objectives of this assessment are (1) to determine the strength of evidence that the substance is an animal or human carcinogen, and (2) to provide an upper-bound estimate of the possible risks of human exposure to the substances in drinking water.

One issue considered in assessing carcinogenicity is the data on inhalation and ingestion. If the data show the chemical to be carcinogenic through ingestion, then the chemical will be considered a potential carcinogen and evaluated based upon the carcinogenicity data. If the chemical has been shown to be carcinogenic through inhalation and not ingestion, it will not be be considered a potential carcinogen via drinking water. A third case consists of chemicals that are shown to be carcinogenic through inhalation, but the data on their carcinogenicity through ingestion are either not available or equivocal. In these situations, carcinogenicity will be determined on a case-by-case basis by examining the applicablity of the inhalation data to drinking water exposure.

Exposure Assessment

Drinking water exposures are estimated for three classes of substances.

Volatile Organic Chemicals. The results of six federal surveys were combined into estimates of national exposure using a multinomial distribution. This method estimates the proportion of the total exposures for a given range of concentrations on the basis of the observed relative frequency of that range in the surveys.

Synthetic Organic Chemicals (Pesticides). Data on the occurrence of SOCs in drinking water are quite limited. Estimates are based upon data on use of ground water and vulnerability of ground water to SOC contamination, and limited surface water data.

Radionuclides. Exposure is based upon various surveys and sources, including, to a limited extent, monitoring data for compliance with the Interim Regulations for Radionuclides. For example,

- for radium 226, exposure is based on a 1980-81 survey of 2,500 public ground-water supplies in 27 states by EPA's Office of Radiation Programs (ORP), as well as studies conducted in New England and in the Coastal Plain and Piedmont regions;
- for radon exposure is based on data from the above-mentioned New England and ORP studies; and

- for natural uranium. Exposure is based on data from the U.S.G.S. Natural Uranium Resource Evaluation Program, in which more than 55,000 ground-water samples and 34,000 surface-water samples were analyzed during the late 1970s.

ESTIMATED RISKS

The following table shows the cancer risks from different pollutants in drinking water.

Contaminant	Concentration Resulting in 10^{-6} Risk (ug/l)	Population Exposed (Millions)	Cases Per Year
Vinyl Chloride	0.015	0.9	40
Trichloroethylene	2.6	1.3	<1
Carbon Tetrachloride	0.27	0.5	12
1,2 Dichloroethane	0.38	0.023	<1
Benzene	1.3	0.033	<1
para-Dichlorobenzene	2.0[a]	0	0
Toxaphene	0.03	--	--
Chlordane	0.0218	0.9	0.2
Alachlor	0.15	4.5	3.4
Epichlorohydrin	3.54	--	--
Dioxin	2.2×10^{-7}	--	--
PAHs	2.8	--	--
PCBs	0.008	--	--
Phthalates	b	--	--
Acrylanide	0.01	--	--
DBCP	0.025	2.4	2.4
EDB	5×10^{-4}	2.4	8
Trihalomethanes	b	>45	322[c]
Heptachlor Epoxide & Heptachlor	.00065	7.0	42
Radium - 226	0.1 pCi/l[d]	1	3-60
Radium - 228	0.2 pCi/l	>1	3-60
Natural Uranium	0.7 pCi/l	>100	1-10
Radon - 222	10.0 pCi/l	>100	30-600

a/ Draft potency estimate.

b/ Under review by the Carcinogen Assessment Group (CAG).

c/ Current estimate for systems serving 10,000 or more people, having a concentration of 100 ug/l or more. May be different if CAG risk estimate for chloroform, currently under review, changes.

d/ Picocuries per liter.

CATEGORY 2

#10: Application of Pesticides

PROBLEM DEFINITION

The summary for Pesticide Residues on Foods (ranked third by the cancer work group) notes that about 200 pesticide chemicals are potentially oncogenic. These chemicals are professionally applied by workers to protect agricultural crops.

RISK ASSESSMENT METHODOLOGY

Data Sources and Assessment

The sources and assessment of data are the same as described under the summary for Pesticides Residues on Food. That is, data are usually generated by the pesticide companies and augmented by the NTP testing program. As indicated, data assessment is extensive, both because risk assessment of pesticides involves a very large number of chemicals, and the amount of data for each chemical often requires over 1,000 hours of basic review time.

Exposure Estimates

The exposure estimates for pesticide application pose the most complex issue in assessing risks from actual use. Generally, a matrix of exposure scenarios must be developed, depending on some of the following factors:

- the assignment of the worker in the crew, e.g., mixer/loader, pilot, tractor driver, flagman;
- the application protocol -- e.g., single or multiple applications, lb/acre needed for control;
- the formulation to be used -- e.g., liquid, dust, granular; and
- the protective equipment and clothing to be used -- e.g., closed systems, respirators, gloves, enclosed tractor cabs.

For the most frequently encountered exposure scenarios, actual data on the deposit of some chemicals on workers are available. This information is often used for modelling the exposure of other similarly applied chemicals, taking into account the different application rates between chemicals.

Exposure in the agricultural setting is usually limited to several days or weeks during the year and varies from chemical to chemical, even among uses of the same chemical. These determinations are generally linked to the use directions on the pesticide product -- e.g., apply to dormant fruit trees, apply at fruit set, or apply as needed when infestation of pest reaches a certain level.

It is obvious that the exposure scenarios can cover a multitude of combinations and permutations of the factors described. Usually the exposure

assessment is limited to several major scenarios, but for widely used chemicals there can be as many as 50 different exposure scenarios estimated.

Addressing all the variables, the exposure is calculated on both a daily basis and an annual basis -- i.e., daily exposure times expected use-days per year, and expressed as mg/kg body weight/year.

The major route of exposure of agricultural workers is through the skin. Especially for oncogenic risk assessment, it is desirable to know what proportion of the chemical deposited on the skin is expected to penetrate. Animal studies designed to answer this question can be carried out but often the results are not very satisfactory in a scientific sense and provide only a crude or partial answer for extrapolation to the human experience.

The estimate of the exposed population -- i.e., the number of individual workers involved with a particular pesticide application -- is in most instances not very accurate, and data on the number of workers are generally not readily available. The production volume of a chemical is sometimes used to predict the number of individuals exposed.

In a qualitative sense, the exposure of agricultural workers to pesticides is a reality. The quantitative estimate is difficult because of the diversified agricultural practices, application equipment, and protective equipment. A large number of farm workers may not be fully cognizant of the proper handling of pesticides. Thus, certification of pesticide applicators may provide a more uniform and careful use of pesticides.

The problem of determining the number of workers applying a certain pesticide is further compounded by the fact that work crews may use several chemicals in succession. However, a composite risk assessment is not performed, thus potentially underestimating the individual risk.

Similarly, a pesticide may be labeled for use once a year (e.g., herbicides). However, an application crew may treat a larger growing area for several days according to changing growing seasons and practices. Thus, the individual worker may experience a larger exposure than deducted from use directions.

Although the uncertainties are substantial for estimating risks to pesticide applicators, there is a growing concern over a higher than expected cancer rate among farm workers. While these findings cannot necessarily be solely attributed to pesticide use, it would seem prudent to keep farm worker risk at a minimum from all sources of potential oncogenic risks.

Risk Calculation

The extrapolation of long-term animal data to obtain a potency estimate in human equivalents is performed as described under the summary on exposure to Pesticides Residues on Foods. Since the animal data for determining oncogenic effects are usually based on a lifetime daily oral exposure, the exposure of agricultural workers is amortized as follows:

1. The yearly exposure is used for risk calculation, and the average daily exposure is calculated by dividing the yearly exposure by 365.

2. It is assumed that a worker is exposed for 40 years out of a lifetime of 70 years.

3. The difference between dermal and inhalation absorption versus oral absorption, if known, is factored in.

ESTIMATED RISKS

In the experience of the Office of Pesticides Programs, the individual lifetime risk for an applicator is usually much higher than the individual lifetime risk for the general population consuming pesticide residues on food. The difference in individual lifetime risk can be three to four orders of magnitude. Often the individual risk is calculated to be as high as one in ten. However, since the population of agricultural workers is very small as compared with the general population, the population risk becomes smaller for the pesticide applicator.

After analyzing several representative agricultural pesticides, it was determined that the average lifetime population risk is about 35 persons/lifetime/chemical. Thus the yearly risk/chemical would be 0.5 person/year/chemical (i.e., 35/70 with 70 years being the average lifetime). The total yearly risk from all pesticide chemicals estimated to be oncogens (200) would be 0.5 x 200 = 100 persons/year.

CATEGORY 2

#11: Radiation Other Than Indoor Radon

PROBLEM DEFINITION

Ionizing radiation was among the first environmental causes of cancer to be identified. Cancer may develop at any of several sites, largely determined by the type and location of exposure. From a collective dose standpoint, the largest sources of radiation, by far, are natural background radiation and medical treatments. Excluding doses due to radon, the average person in the United States receives about 100 mrem per year from natural radiation, plus a similar amount from medical procedures.

EXPOSURES ADDRESSED IN THIS SUMMARY

This section deals with exposures to ionizing radiation via occupational, consumer product, and industrial emissions. Exposure to indoor radon is mostly covered under the problem areas Indoor Radon and Drinking Water. Radon exposures included under this problem area are occupational exposures, and that small fraction of radon exposure resulting from industrial emissions.

Exposures to other types of electromagnetic radiation are not discussed here. The potential carcinogenic impact of enhanced exposure to ultraviolet light is treated under Depletion of Stratospheric Ozone. Although there are conflicting data from both animal and epidemiological studies suggesting that environmental exposures to microwaves and lower-frequency fields may cause cancer, this possibility is neither verified nor quantifiable as yet. Finally, because medical treatments are clearly outside EPA's purview and because natural background radiation is largely unavoidable, these exposures are not included in this section.

RISK ASSESSMENT METHODOLOGY

For the purpose of risk assessment, EPA's Office of Radiation Programs uses a risk factor of 2.8×10^{-4} fatal cancers per rad of whole-body low linear energy transfer (low-LET) ionizing radiation to the general population (EPA, 1984). In other words, exposure to one rad of low-LET radiation is estimated to increase one's chances of contracting a fatal cancer by appoximately one in 3,500. This estimate is primarily derived from epidemiological studies on the Japanese A-bomb survivors. It represents a linear extrapolation from doses above 50 rads and is an average of "absolute" and "relative" risk model projections (i.e., projections that assume that the excess absolute or relative risk per unit dose is constant over time).

The risk to adults is lower than for children. Consequently, for occupational exposures, a lower risk factor is used here: 2×10^{-4} (two out of 10,000) fatal cancers per rad. When the radiation is non-uniformly distributed over the body, the dose and associated risk for each organ must be calculated separately, as described in the reference cited above (EPA, 1984).

The methodology for assessing risk from exposure to radon daughters is discussed under Indoor Radon. For internal alpha emitters other than inhaled radon daughters, the risk per rad-absorbed dose to any organ is assumed to be eight times that for low-LET radiation.

All these risk factors reflect estimates of fatal cancers. Given current cancer survival statistics, roughly an equal number of nonfatal cancers should also be projected if the radiation dose is uniformly delivered to the whole body. Otherwise, the situation must be examined on an organ-by-organ basis. In particular, lung cancer is about 90% fatal. Hence, estimates of fatal cancers resulting from radon exposure can be used to approximate total cancers as well.

ESTIMATED RISKS

Following are estimations of the risk of dying from cancer from being exposed to occupational, consumer product, and industrial sources of radiation.

Occupational Exposures

EPA has published a review of occupational exposures to ionizing radiation in the United States (Kumazawa et al., 1984). The review treats four classes of occupational exposures. One of those classes, the enhanced exposure to cosmic radiation received by flight crews and attendants, is not considered here because it is outside EPA's purview.

The largest class of people occupationally exposed to radiation encompasses several major categories of workers, including those employed in industry, medicine, research, and defense. On the basis of monitoring data, about 1.7 million workers were potentially exposed to radiation in 1985, and about 850,000 received detectable doses of ionizing radiation. The collective dose estimated from the monitoring data was 175,000 person-rem. Assuming two out of 10,000 (2×10^{-4}) fatal cancers per person-rem, about 35 fatal cancers would be expected from this exposure. Most of the dose would be low-LET radiation delivered fairly uniformly to the whole body. Hence, in addition to the fatal cancers, a comparable number of nonfatal cancers would be projected. We estimate the maximum lifetime occupational dose to be about 100 rem, implying a maximum estimated lifetime risk of about 2%.

A second class of exposed people consists of students and visitors at Department of Energy facilities. In 1980, 41,500 people in this category received measurable doses. The collective dose was approximately 3,900 person-rem, implying about 0.8 (1.6) projected fatal (total) cancers.

The final class reflects radon exposures to miners. In 1980, 17,700 miners were exposed to radon in uranium mines (13,500) and nonuranium mines (4,200). About 60% of these miners received a measurable exposure of 0.7 WLM or more, and the collective exposure for all miners was about 7,500 person-WLM, based on monitoring data. (Concentrations of radon daughters are ordinarily given in units of working levels (WL). Exposure (concentration x time) is usually expressed in working level months (WLM).) If we assume that the baseline mortality from lung cancer among miners is about 7% (which is reasonable for an essentially all-male population), about 10 fatal lung cancers annually would be calculated on the basis of a 2% relative risk per WLM. The maximum

lifetime exposure may be about 60 WLM, corresponding to an estimated lifetime risk of about 9%.

The estimates relating to occupational exposures are summarized in Table 1. As shown in the table, for one year's occupational exposure, 46 fatal cancers and 82 total cancers are projected.

Exposures from Consumer Products

The largest source of collective dose in this category is from decay of naturally occurring radionuclides in building materials -- e.g., bricks used in construction of homes. Many people would regard this exposure as part of natural background. Obviously, it would be impractical to control much of this exposure in any way, particularly where construction is already complete. Some fraction of the exposure due to new construction might be limited (e.g., by restricting the use of phosphate mine tailings in wallboard or some types of slag in cinder block), but we have not tried to estimate the magnitude of this fraction. The remaining dose from consumer products is contributed by a variety of small sources, including television sets, radium dial watches and clocks, and smoke detectors.

As shown in Table 2, about 300 fatal cancers per year are attributed to radiation doses received from consumer products. If doses from building materials are excluded, only about 68 fatal cancers would be calculated. Roughly an equal number of nonfatal cancers would be projected in addition to the fatal cancers.

Exposures to Industrial Emissions

As background for standard setting, the Office of Radiation Programs has estimated the number of fatal cancers resulting annually from exposures to radionuclides emitted by various industrial sources (EPA, 1982; EPA, 1983; EPA, 1984). Since these documents were published, EPA has slightly revised its risk estimates for radon. However, those previous estimates of radon-induced lung cancers would be consistent with current central estimates derived on the basis of a 2% per WLM relative risk coefficient (EPA, 1986). Therefore, we have used the previously published estimates of fatal cancers induced by industrial emissions, without change, for radon emissions, as well as for emissions of other radionuclides.

The projected fatal cancers from the most significant classes of sources is summarized in Table 3. Not included is the impact of emissions from nuclear power plants, which is very small. The Nuclear Regulatory Commission has estimated the collective dose to the public from such emissions in 1982 to be about 130 person-rem (Baker and Peloquin, 1986). Based on a risk factor of 2.8×10^{-4}/rem, this would correspond to about 0.04 fatal cancers induced annually from this source. As shown in the table, about 10 fatal cancers are estimated to be induced annually in the general population by industrial emissions of radionuclides. Most of the projected cancers from these sources are lung cancers, so nonfatal cancers can be neglected.

SUMMARY

Based on the restricted classes of sources treated here, ionizing radiation may cause about 360 fatal cancers a year. Almost two-thirds of these, however, are from radiation emitted by building materials. In addition to these fatal cancers, a comparable number of nonfatal cancers would be projected. For perspective, employing the same risk assessment methodology, one would project roughly 10,000 fatal cancers induced by radiation each year from medical exposures and natural background (apart from radon).

Table 1

Annual Impact of
Occupational Exposures[a]

Occupational Class	Dose (Person-rem)	Exposure (WLM)	Fatal Cancers[b]	Total Cancers	Max. Individual Lifetime Risk
Major categories other than miners	175,000	---	35	70[c]	2%
Students and visitors	3900	---	0.8	1.6[c]	--
Miners	---	7500	10	11[d]	9%
Total	178,900	7500	46	82[c,d]	9%

a/Source of exposure data: Kumazawa et al., 1984.

b/Assumes a risk factor of 2×10^{-4} fatal cancers/person-rem for whole-body exposures to low-LET radiation and .02 x baseline lung cancer fatality rate/WLM for radon exposures.

c/Assumes approximately 1 nonfatal cancer for every fatal cancer induced by whole-body irradiation.

d/Assumes about 90% of all radon-induced lung cancers are fatal.

Table 2

Consumer Product Exposures to Ionizing Radiation

Source	Average Dose[a] (mrem/yr)	Collective Dose (Person-rem/yr)	Fatal Cancers[b] (per year)
Building materials	3-4	700,000 - 1,000,000	196-280
Televisions	0.5	120,000	34
Other	0.5	120,000	34
Total	4-5	approx. 1,000,000	approx. 300

a/References: National Academy of Sciences, 1980.

b/Based on a risk factor of 2.8×10^{-4} fatal cancers/person-rem.

Table 3

Fatal Cancers from Exposures to Radionuclides

Source of Emissions	Fatal Cancers/yr
Coal plants	1.3*
Phosphate industry	0.1*
Uranium mines	3*
Uranium mills	6**

*/U.S. EPA, 1984.

**/U.S. EPA, 1982, 1983.

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CATEGORY 2

#12: Other Pesticide Risks

PROBLEM DEFINITION

Two other sections on pesticides describe the problems associated with pesticide residues on food and exposure of agricultural workers to pesticides. Other uses of pesticides are generally designated as domestic, household, and industrial uses and includes hospital and household disinfectants, fumigants, termiticides, and wood presentatives. For most of these chemicals, testing for oncogenic effects is not available. Termiticides and some wood preservatives, however, have been implicated as posing an oncogenic risk.

The following considerations are mainly directed at these uses. However, the many chemicals of unknown oncogenic potential may significantly alter the comparative risk standing of "other pesticides."

RISK ASSESSMENT METHODOLOGY

Data Sources and Assessment

The data source for these consumer/household pesticides are the same as for the other pesticides -- i.e., registrant-generated data, National Toxicology Program data, and, to a small degree, data from the scientific literature data. For chemicals where no oncogenicity data are available, data call-in letters will be issued in the next few months. Thus, several years may pass until new studies will be completed.

For those chemical where oncogenicity data are already available, scientific evaluations similar to those performed on agricultural pesticides will be necessary. For chemicals not yet tested, a tiered testing regimen is proposed, which includes an assessment of oncogenicity based on exposure estimates, and an assumed value of cancer potency (Q_1*).

Exposure Estimation

For most consumer pesticides modeled, exposure estimates are not available. By their very nature, the uses are not strictly controlled by labelling. For example, hospital disinfectants are certainly used every working day but the of mg/person/day has never been estimated.

On the other hand, for termiticides, air montoring in treated houses has demonstrated that exposure is rather significant, both with respect to magnitude and duration. We also have determined that the use of home or farm wood perservatives results in significant exposure, even though these products are not used on a daily basis.

In summary, exposure estimation for these "other pesticide" uses is very complex and includes a large number of scenarios and contains many uncertainties.

RISK ESTIMATION

If better data existed, the basic risk calculations would be similar to those described elsewhere for pesticides. But since neither the exposed population nor the daily exposure is well defined, it is extemerly difficult to develop reliable figures. However, the cancer work group agreed that a yearly nationwide annual incidence of 150 cancer cases may be a reasonable assumption. This opinion was arrived at by a one-on-one comparison with other environmental problem areas, using professional judgment to determine which one of each set of two is expected to be worse.

UNCERTAINTIES

The following uncertainties are associated with estimating the risks of cancer from exposure to "other pesticides."

- The number of chemicals involved is large.
- Only a few consumer chemicals have adequate oncogenicity data.
- The exposed population is unknown, but is likely to be large for some chemicals.
- Daily and lifetime exposures are not known and are difficult to determine. For example, label directions are less likely followed by the general public than by agricultural workers.
- Exposure is by dermal contact or inhalation, and correlating these exposures to ingestion data on pesticides is difficult.

CATEGORY 2

#13: Hazardous Waste Sites -- Active

PROBLEM DEFINITION

This category includes the risks posed by a number of sources, including RCRA landfills and surface impoundments (both open and closed), hazardous waste storage tanks, hazardous wastes burned in boilers and furnaces, hazardous waste incinerators, waste oil, and solid waste management units (SMUs). The primary routes of exposure are ground water for landfills, surface impoundments, storage tanks, and SMUs and air for boilers and furnaces, incinerators, and waste oil.

POLLUTANTS ADDRESSED IN THIS SUMMARY

The pollutants evaluated for each source are as follows:

RCRA Landfills and Surface Impoundments

Pollutants were selected based on analyses of wastes disposed at 55 RCRA facilities that plan to continue operating. Constituents in wastes and concentrations are based on data from the waste, environment, and technology (WET) model. Constituents of major concern include 2,4-dinitrotoluene, arsenic, acenapthene, toluene diamine, benzene, and acrylonitrile. (An alternative modelling exercise examined the entire range of wastes and constituents included in the WET data base.)

Hazardous Waste Storage Tanks

One study (ICF/Pope-Reid, 1986) used methylene chloride and 1,1,1-trichloroethane as representative of the types and toxicities of wastes stored in tanks. Another examined 32 waste streams and 36 toxic chemicals.

Hazardous Wastes Burned in Boilers and Furnaces

The carcinogens evaluated include metals (chromium, cadmium, and arsenic), products of incomplete combustion (PICs: chloroform, tetrachloroethylene, benzene, and carbon tetrachloride), and polycyclic organic hydrocarbons (a weighted average of POHCs in high-heat-value liquid wastes).

Hazardous Waste Incinerators

Two waste streams were used as representative of the range of wastes incinerated. One consisted of 50 percent polychlorinated biphenyls (PCBs) (presumed to be 1254 substituted) and the other 50 percent ethylene dichloride. Both streams were assumed to contain four carcinogenic metals: arsenic, cadmium, chromium (VI), and nickel. PIC concentrations used were based on available test data.

Waste Oil

The four carcinogens in waste oil that were assessed are arsenic, cadmium, chromium (VI), and PCB-1254.

Solid Waste Management Units (SMUs)

No information is available on representative constituents.

The waste streams used in the various analyses summarized in this summary are representative of the range of wastes that are either disposed of or are burned. Consequently, any estimate of risk is unlikely to be underestimated. Conservative assumptions often were made regarding the concentrations of carcinogens in waste streams.

RISK ASSESSMENT METHODOLOGY, ESTIMATED RISKS, AND UNCERTAINTIES

Estimates of the risk posed by each source were developed as follows.

RCRA Landfills and Surface Impoundments

The liner location model was used to project ground-water contamination around 55 facilities with 67 units that plan to continue operating. The modelling extrapolated over a 400-year period. Risks were estimated assuming: (1) current well distributions, (2) people continue to drink contaminated water, (3) no degradation of contaminants, and (4) unlined facilities. Under these assumptions only two facilities appear to cause health risks (45 facilities had no drinking water wells within two miles).

Extrapolating these results to the 1,500 open and closed RCRA facilities yields an estimate of 0.3 cancers per year. An upper-bound estimate of 384 cancers per year was developed by assuming 1,000 people were drinking water from wells located at the facility boundary. (Since most of this risk was from arsenic, the estimate would drop by an order of magnitude if an updated CAG potency for ingested arsenic were used.)

The lower estimate may underestimate cancer incidence because facilities that plan to continue operating may have significantly less contamination problems than those that close. Further, the current distribution of wells may change considerably over time, possibly increasing the potential for future exposure. Other modelling work that was conducted to analyze the effects of the land disposal ban conservatively places cancer incidence around 30 to 40 cases per year.

Significant uncertainty exists regarding the distribution of future wells, actual migration of contaminants from RCRA facilities, the synergistic effects of multiple contaminants, and the potential for chemical reactions among and transformation of chemical constituents.

Hazardous Waste Storage Tanks

There are about 15,000 hazardous waste storage tanks and about 11,400 small-quantity-generator facilities with tanks. Health risks may arise if a tank fails and the subsequent leak contaminates ground-water drinking sources.

To define alternative scenarios, existing analyses of risks from leaking tanks assume a deterministic tank failure rate (e.g., failure in 20 years

with a specified release rate) or a probabalistic failure rate, and use various hydrogeologic settings and waste streams. Individual risks are estimated to be in the range of one cancer for every 100,000 exposed (10^{-5}). No estimates of annual cancer incidence are available.

Hazardous Wastes Burned in Boilers and Furnaces

Concentrations of metals, POHCs, and PICs were estimated within a 50-km radius for a representative sample of boilers and furnaces using the industrial source complex air dispersion model. Risk estimates were based on the assumption of 70 years of continuous exposure.

Aggregate cancer incidence was estimated to be about 0.3 cases per year. Individual risks occasionally are as high a 10^{-5} and, most often, lower than 10^{-6}.

Uncertainties in the analysis arise from lack of information on waste composition, destruction performance of boilers and furnaces, and potential clustering of devices, which would affect risks to most exposed individuals.

Hazardous Waste Incinerators

Air dispersion models were used to estimate concentrations of metals, POHCs, and PICs in a 50-km radius around four large commercial incinerators. Two waste streams were used to represent the range of wastes that are currently incinerated.

Cancer incidence was averaged for the four incinerators and was extrapolated to the 235 on-site and commerical incinerators across the country. Risk was adjusted downward by a factor of five to account for the large amount of waste assumed in the initial analysis and by a factor of eight to reduce metal concentrations and adjust for average toxicity of POHCs. Annual cancer incidence from incinerator emissions is estimated to be about five cases.

Uncertainties in the analysis are mostly with regard to metal concentrations in hazardous wastes, the efficiency of air pollution control devices in removing metals from off-gas streams, and the amount and type of PICs.

Waste Oil

Releases of contaminants to air, surface water, and ground water were modelled for model facilities representing nine major oil disposal and reuse methods. Exposures from airborne contaminants resulting from waste oil burning, estimated in an earlier analysis as responsible for approximately 40 percent of total waste oil risks, were examined within a radius of 50 km for the model facilities. Nationwide annual cancer incidence is estimated to be about two cases from this exposure route, after adjusting for overly conservative assumptions in the initial analysis.

Solid Waste Management Units

There are about 9,000 SMUs. They are ill-defined, and the contamination associated with them is undetermined. A unit may consist of buried asphalt, old spills of material, or an industrial landfill contaminated with hazardous materials.

The extent of the human health problem posed by these sites is unknown. Many could be located fairly close to drinking water supplies.

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IEC and ICF, Inc. Regulatory Analysis of Proposed Restrictions on Land Disposal of Hazardous Waste. Prepared for U.S. EPA, Office of Solid Waste, Economic Analysis Branch, December 1985.

Sobotka & Co., Inc. "Comparative Risk Analysis of Sources of Groundwater Contamination." Phase 3 draft report. Prepared for U.S. EPA, Office of Policy Analysis, Sepember 25, 1986.

Temple, Barker and Sloane, Inc. "Background Document: Regulatory Impact Analysis of Proposed Standards for Management of Used Oil." Prepared for U.S. EPA, Office of Solid Waste, November 1985.

U.S. EPA, Office of Solid Waste, Economic Analysis Branch. "Cross-Program Analysis of Land Disposal Regulations."

U.S. EPA. Assessment of Incineration As a Treatment Method for Liquid Organic Hazardous Wastes, "Background Report IV: Comparisons of Risks from Land-Based and Ocean-Based Incineration." March 1985.

U.S. EPA. "Regulatory Analysis for Waste-as-Fuel Technical Standards, Proposed Rule." Draft report. October 1986.

U.S. EPA. "Comparison of Risks from Land-Based and Ocean-Based Incineration of Hazardous Wastes." Supplemental analysis. October 1986.

CATEGORY 2

#14: Non-Hazardous Waste Sites -- Industrial

PROBLEM DEFINITION

This category includes risks posed by the 3,400 industrial landfills and 15,000 industrial surface impoundments throughout the country. The primary route of exposure is through ground water.

POLLUTANTS ADDRESSED IN THIS SUMMARY

Pollutants included for analysis are arsenic, chloroform, benzene, and 1,1,2,2-tetrachloroethane. These are the major carcinogens for the three industries analyzed for this summary (iron and steel, pulp and paper, and organic chemicals).

RISK ASSESSMENT METHODOLOGY

The liner location model was used to simulate individual health risks from unlined surface impoundments containing raw waste from five industries: iron and steel, fabricated metals, pulp and paper, inorganic chemicals, and organic chemicals. For each industry, a prototype surface impoundment containing raw waste was modeled in 64 environmental settings. Ground-water concentrations were simulated over a 400-year time horizon (200 years of release plus 200 years of transport) at wells 60m, 600m, and 1500m hydraulically downgradient from the source. Only individual risks were estimated in this analysis. The model calculates running 70-year (lifetime) average exposures beginning with each year of the 400-year simulation, and estimates risks based on these 70-year average exposures. Average risks are reported by summing the risks for each of the 400 years, and dividing by 400.

Surface impoundments for the fabricated metals and inorganic chemicals industries did not have carcinogenic consitutents of concern. Therefore, the cancer risks from these two industries were not modeled.

The table below shows the range of individual risk associated with each carcinogen for each of the other three industrial sources.

Industries	Arsenic	Chloroform	Benzene
Iron and Steel	$3x10^{-5}$ - $6x10^{-1}$	--	--
Pulp and Paper	--	$2x10^{-4}$ - $1x10^{-8}$	--
Organic Chemicals	--	$1x10^{-6}$ - $1x10^{-5}$	$1x10^{-4}$ - $1x10^{-3}$

The highest individual risk across sources was for arsenic (6×10^{-1}). However, this would drop by about an order of magnitude if a more recent potency estimate were used. Chloroform and benzene also had significant individual risk in some of the environmental settings. The chemical 1,1,2,2-tetrachloroethane was also modeled for pulp and paper, but showed no risk.

When risks were modeled under the assumption that people would stop drinking water as soon as one of the contaminant concentrations exceeded the taste and order threshold, risks for organic chemicals from surface impoundments were considerably lower. Risks from the other two categories were not affected under this assumption.

No attempt has been made to estimate population cancer risk from this problem area.

REFERENCE

Sobotka & Co., Inc. "Comparative Risk Analysis of Sources of Groundwater Contamination." Phase 3 draft report. Prepared for U.S. EPA, Office of Policy Analysis, September 25, 1986.

CATEGORY 2

#15: New Chemicals

PROBLEM DEFINITION

New chemicals are defined as those not listed on the TSCA Inventory of Existing Chemical Substances. New substances typically enter the market as substitutes for existing chemicals. Therefore, risks considered in this environmental problem category cover the range of exposure scenarios presented by existing chemicals.

Approximately 1,800 Premanufacture Notifications of intent to manufacture new chemical substances are submitted to EPA each year. Of these, approximately half are actually manufactured and used commercially.

The term "new" chemicals as used in this summary refers to industrial chemicals. New pesticides are considered elsewhere, and new food additives and drugs are not considered in the overall Comparative Risk Project.

EPA'S RESPONSIBILITY IN THIS AREA

The Toxic Substances Control Act (TSCA) requires that industry notify EPA at least 90 days before manufacturing or importing a new chemical substance. The notification must include exposure-related data and available information on the health effects (including potential carcinogenicity) of the new substance. EPA reviews these data to evaluate the potential risks posed by the new substance. Where concerns are identified, EPA takes action to control exposure to it, pending the development of additional data.

POLLUTANTS ADDRESSED IN THIS SUMMARY

In the great majority of cases, the chemical identity and use of new chemicals are claimed confidential by the submitter. Therefore this analysis does not identify the risks presented by specific new chemicals.

RISK ASSESSMENT METHODOLOGY

EPA's assessment of risk focuses on the third year after market introduction. Therefore, it does not reflect the risks that may be realized in the longer term when use and production increase.

ESTIMATED RISKS

Most new chemicals are manufactured for relatively few customers in response to a limited commercial demand. Although specific data are not available, it is generally believed that most new chemicals only have a limited commercial life and do not significantly penetrate existing markets. In spite of this fact, slowly but inevitably, the current spectrum of existing chemicals will be substantially replaced by new chemical substances that have

been "screened" by EPA review. To the extent that this review identifies potential carcinogens and prohibits their marketing, overall carcinogenic risks of chemicals in commerce will decrease. In this regard, the program has in the past six years taken action to limit exposure or prevent the marketing of over 100 potentially carcinogenic substances. However, given the nature of this environmental problem, it is not possible to present a more quantitative estimate of risk.

In addition, the heightened industry awareness that results from the requirement to undergo independent review of potential risk before marketing has probably resulted in industry's decision to not submit for EPA review chemicals likely to be considered potential carcinogenics.

CATEGORY 3

#16: Non-Hazardous Waste Sites -- Municipal

PROBLEM DEFINITION

This category includes the risk posed by both open and closed municipal landfills, municipal sludge and refuse incinerators, and municipal surface impoundments. The primary route of exposure is ground water for municipal landfills and surface impoundments and air for sludge and refuse incinerators.

POLLUTANTS ADDRESSED IN THIS SUMMARY

The pollutants addressed for each source are as follows.

Municipal Landfills

Carcinogenic constituents included for analysis are considered typical of constituents in leachate generated from the codisposal of municipal solid waste, nonhazardous waste, household waste, and hazardous wastes from small-quantity generators. (It would not be typical of landfills that received substantial quantities of hazardous waste.) The constituents modelled are vinyl chloride, arsenic, 1,1,2,2-tetrachloroethane, dichloromethane, and carbon tetrachloride.

Municipal Sludge Incinerators

The constituents modelled include the following metals: arsenic, beryllium, cadmium, chromium, mercury, and nickel; and the following organics: aldrin, benzo(a)pryrene (BAP), chlordane, DEHP, PCBs, toxaphene, and vinyl chloride.

Municipal Refuse Incinerators

The major constitutents included in the analysis were dioxins, BAP, cadmium, chromium, arsenic, and beryllium. The analysis did not assess formaldehyde and other organics and trace metals.

Municipal Surface Impoundments

No pollutants were analyzed for these sites.

RISK ASSESSMENT METHODOLOGY AND ESTIMATED RISKS

Estimates of risk posed by each source were developed as follows.

Municipal Landfills

Concentrations of contaminants in ground water were modelled at 60m, 600m, and 1,500m for 14 different environmental settings and 11 different hydrogeologic settings using the liner-location ground-water model. The

same leachate, representative of codisposed wastes, was used for each combination of settings. Modelling analyzed a 300-year period, and the risks at each well distance were calculated as the mean average lifetime risk over 300 years. The modelling results were then weighted to account for the relative frequencies of environmental settings, hydrogeological settings, landfill sizes, and well distances observed in the actual populations of subtitle D (nonhazardous waste) facilities.

This resulted in the following distribution of average individual cancer risk at municipal landfills.

Range of Individual Risk	Frequency of Occurence
10^{-4} - 10^{-5}	12%
10^{-5} - 10^{-6}	28%
10^{-6} - 10^{-7}	27.5%
10^{-7} - 10^{-8}	13%
10^{-8} - 10^{-9}	3.5%
10^{-9} - 10^{-10}	2%
$\leq 10^{-10}$	14%

Annual cancer incidence is estimated by: (1) multiplying the weighted average risk from the above distribution (8×10^{-6}) by (2) an estimate of the average population living within one mile of such sites (about 600) by (3) the number of open and closed landfills (9,100 + 30,000 = 39,100), and (4) dividing by 70 (average lifetime). This procedure yields an estimate of about three cancer cases per year resulting from exposure to wastes from municipal landfills.

The major uncertainties in the analysis are the simulated release rates, constituents present in leachate, particularly for closed landfills, and future population exposure.

Municipal Sludge Incinerators

Approximately 309 sludge incinerators operate at 195 facilities. About 80 percent are multiple hearth, 11 percent are fluidized-bed, and the remainder are electric infra-red, co-combustion with refuse, or other.

Three air dispersion models were used to estimate concentrations in a 50-km radius around the model facilities: the ISCLT model for flat terrain, LONG Z for rural settings, and COMPLEX I for urban settings. Model facilities were selected to be representative of the capacity, stack height, stack gas exit velocity, population distribution, terrain, and meteorology of actual incinerators. Actual plants were assigned to each model facility, based on similarity in characteristics. Emissions were then adjusted to account for the actual amount of sludge burned and whether the sludge was a light industrial or heavy industrial sludge.

The resulting estimate of cancer incidence is about 23 cases per year, most of which are caused by emissions of metals. The maximum individual risk was estimated to be as high as one in 1,000 (10^{-3}).

Uncertainties in the analysis arise from uncertainty about metal concentration in sludge and the form of chromium emitted (hexavalent was assumed).

Municipal Refuse Incinerators

Based on very preliminary modelling and national extrapolation, excess cancer incidence is estimated at 3-14 cases per year, and maximum individual risk at about 10^{-3}. Most of the modelled risk is from dioxins. However, the analysis is based on very limited data, and the estimates are speculative.

Municipal Surface Impoundments

There are about 20,000 municipal surface impoundments in the United States. No analysis has yet been performed on the extent to which these sites contaminate ground water.

REFERENCES

U.S. EPA, Office of Solid Waste, Economic Analysis Branch. "Preliminary and Ongoing Municipal D Analysis."

U.S. EPA, Office of Policy Analysis. "Crossmedia Impacts of Utilization and Disposal of Municipal Sludges." May 1986.

CATEGORY 3

#17: Contaminated Sludge

PROBLEM DEFINITION

In 1983-84, in preparing technical regulations for the use/disposal of sewage sludge from publicly owned treatment works (POTWs), EPA considered the following use/disposal options: land application to human food-chain and non-food-chain crops, distribution and marketing of sludge or sludge-derived products for use as soil conditioners and nutrients, landfilling, incineration, and ocean disposal.

It is difficult to determine if sludge is a factor in causing cancer and, if so, the extent of that problem. The initial regulation is being designed to forestall as many of the environmental consequences of sludge disposal as possible.

POLLUTANTS ADDRESSED IN THIS SUMMARY

Forty-one chemicals were selected for the initial regulation based on a survey of sludge from 40 POTWs representative of national sludge quality. Of these, 24 are known or suspected carcinogens according to either EPA's Office of Pesticide Programs or the Carcinogen Assessment Group. A list of these chemicals and the disposal options being considered appears at the end of this summary.

RISK ASSESSMENT METHODOLOGY AND RISK ESTIMATES

The methodologies developed are comprised of environmental models that consist of mathematical expressions or algorithms. The models predict the movement of pollutants from sludge placed on or into the land, emissions from sludge that is incinerated, or dispersion from sludge dumped into the ocean through several environmental compartments or media to reach a receptor or target organism at a calculated concentration. This concentration of pollutant is then compared with either an environmental impact or a human health criterion. This criterion is a concentration of a pollutant, that, when exceeded, will harm the environment or human health.

Several quantitative risk estimates from a previous analysis are presented in the following paragraphs. These estimates were derived in a report developed by EPA's Office of Policy Analysis using methods that have not been formally adopted by EPA (U.S. EPA, 1986). Therefore, in all cases these estimates are preliminary; more detailed analyses are currently under way.

Land Application

Sludge may be applied to land as a soil conditioner and provider of supplemental nutrients. The broad term "land application" includes many specific end uses, such as applying sludge for growing row crops, pasture lands, commercial forests, highway landscape, turf farms, and nurseries. In addition, land application includes "dedicated" land disposal -- the application of sludge to POTW land as an alternative to other methods of disposal.

The risks from three types of land application have been estimated. Application of sludge to pasture land has been associated with appoximately 0.6 cancers annually, while crop land application results in about 0.5 cases a year. Most of these are risks are linked to PCBs, which tend to accumulate in plants. Dedicated use yields an estimate of 0.3 cancers annually, resulting from surface water runoff. Other exposure routes were not considered significant.

Distribution and Marketing

With this is type of land application, the primary exposure route is ingestion of food crops treated with (and contaminanted by) sludge. Distribution and marketing products are expected to be transported and used at far greater distances from the POTWs where the sludge originated than the usual land application practices. In addition, less experienced users (e.g., homeowners' application to home gardens or lawns) are involved. Therefore, distribution and marketing practices pose a potentially greater hazard than land application practices.

Distribution and marketing of sludge has been modeled to currently account for just under two cancer cases annually. Just over half of these projected cases result from toxaphene exposure.

Landfilling

Landfills are a widely used and well-documented sludge disposal option. About 45 percent of all sludge generated ends up in a landfill. The two major routes of sludge landfill disposal are monofill (sludge only) and co-disposal with municipal refuse, with the latter predominating.

For the purposes of this analysis, we assumed that vapor loss does not constitute a real cancer hazard. While benzene, trichloroethylene, and other organics are quite volatile, the concentrations generated are generally very low. We also assumed that suspension of contaminant particles from the work face is insignificant, given that the necessary wind velocities rarely occur. In addition, because of the general practice of covering the work face with fresh soil and digging drainage ditches, surface runoff does not pose serious cancer risks. We therefore assumed that ground-water contamination presents the greatest threat to human health.

Approximately 13 cancer cases per year have been estimated to result from ground water contaminated by landfilled sludge. However, this figure would decrease by about an order of magnitude if this analysis were redone using a revised potency estimate for ingested arsenic.

Incineration

Incineration is a major disposal practice for sludge in the United States. While this risk analysis is continuing, preliminary results are available on the basis of an extrapolation from eight model plants to the entire 309 operating sludge incinerators in the nation. This extrapolation yields an estimate of 23 cases annually from the inhalation of carcinogens emitted by sludge incinerators. However, over 90 percent of this estimate result from exposure to chromium, which was assumed to be in the hexavalent form. If the any of the chromium actually emitted is in the trivalent form, these risks may be overstated.

Ocean Disposal

With ocean disposal, sewage sludge is barged for dispersion at a federally approved dumpsite. In general, state authorities and EPA issue a dumping permit to either one large POTW or several small ones.

Using several conservative assumptions, risks were estimated from the consumption of seafood contaminated by sludge from 27 POTWs that dispose of the sludge off the coast of New Jersey. Based on this analysis, over two cases a year were estimated; no national assessment is available.

OVERALL RISK ESTIMATE

The combination of the results of these preliminary analyses results in a total estimate of approximately 40 cases annually. However, there are considerable uncertainties and assumptions implicit in this estimate. We hope that future analyses will improve the reliability of these estimates.

Known or Suspected Carcinogens Being Evaluated Under Section 405(d) of the Clean Water Act

Pollutant	Disopsal Options
Aldrin	LA, I, OD
Arsenic	LA, LF, I
Benzene	LF, I
Benzidene	OD
Benzo(a)pyrene	LA, LF, I, OD
Beryllium	LA, I
Bis(2-ethylhexyl)phthalate	LA, LF, I, OD
Cadmium	LA, LF, I, OD
Carbon Tetrachloride	I
Chlordane	LA, LF, I, OD
Chloroform	I
Chromium	LA, LF, I
DDT/DDE/DDD	LA, LF, OD
Dieldrin	LA, I, OD
Dimethyl Nitrosamine	LF
Heptachlor	LA, OD
Hexachlorobenzene	LA
Methylene Chloride	LA, I
PCBs	LA, LF, I, OD
Pentachlorophenol	LA
Tetrachloroethylene	I
Toxaphene	LA, LF, I, OD
Trichloroethylene	LA, LF
Vinyl Chloride	I

LA = Land Application and Distribution and Marketing
LF = Landfilling
I = Incineration
OD = Ocean Dumping

REFERENCE

U.S. EPA, Office of Policy Analysis, Regulatory Integration Division. "Cross-media Impacts of Utilization and Disposal of Municipal Sludges." May, 1986.

CATEGORY 3

#18: Mining Waste

PROBLEM DEFINITION

This category includes risks posed by mining operations, smelting and refining wastes, and oil and gas operations. The primary route of exposure is ground water.

POLLUTANTS ADDRESSED IN THIS SUMMARY

A formal risk analysis was only conducted for smelting and refining wastes. The carcinogens addressed were arsenic, cadmium, and nickel.

RISK ASSESSMENT METHODOLOGY AND ESTIMATED RISKS

The risks posed by each source are as follows.

Mining Operations

A risk assessment has not been completed for the mining industry. General information on mining operations suggests that population cancer risks are low. To a large extent, this is because the average population within five km of the 300 or so active mines is about 5 percent of that surrounding RCRA Subtitle C (hazardous waste) facilities. Only 22 percent of mines have public drinking water wells within five km, and about 68 percent of these wells serve fewer than 1,000 people.

Smelting and Refining Waste

The liner location model was used to model risk from 29 landfills and surface impoundments receiving wastes from 29 actual smelting and refining facilities in four categories: aluminum, copper, lead, and zinc. The facilities modeled were selected as a representative sample of the entire population of smelting and refining facilities in the United States. Site-specific information on waste type and volume, environmental setting, and exposed populations was used as input to the model. The individual and population cancer risks from arsenic, cadmium, and nickel were estimated over 100 years.

Arsenic was responsible for almost all of the cancer risk. Of the 29 units modeled, 16 had cancer risk, all due to arsenic. Average individual cancer risk ranged from two cases for every million people exposed (2×10^{-6}) to four for every ten people exposed (4×10^{-1}). Extrapolating the results nationally on the basis of population surrounding smelting and refining sites yields an estimate of about 112 cancer cases per year. However, this would be reduced by about an order of magnitude if a more recent potency factor were used for ingested arsenic.

Oil and Gas Operations

Carcinogenic compounds have been observed in ground water and surface water around oil and gas operations, but no concentration data are available.

REFERENCES

Buc and Associates, Inc. "Location of Mines and Factors Affecting Exposure." Draft report. Prepared for U.S. EPA, Office of Solid Waste. June 30, 1986.

ICF, Inc. "Analysis of Human Health Risks Associated with the Management of Hazardous Waste from the Primary Smelting and Refining Industries." February 1985.

U.S. EPA, Office of Solid Waste. Report to Congress, Wastes from the Extraction and Benefication of Metallic Ores, Phosphate Rock, Asbestos, Overburden from Uranium Mining, and Oil Shale. December 1985.

CATEGORY 3

#19: Releases from Storage Tanks

PROBLEM DEFINITION

This category includes risk posed by releases from storage tanks, primarily underground storage tanks containing gasoline. The primary route of exposure is ingestion of ground water.

POLLUTANTS ADDRESSED IN THIS SUMMARY

The risk analysis evaluates the risk posed only by benzene. Risks from toluene and ethylene dibromide (an additive in leaded gasoline) have been generally assessed and are small relative to risks posed by benzene.

RISK ASSESSMENT METHODOLOGY

The underground storage tank model (a Monte Carlo simulation model) was used to predict the number of leaks and the distribution of plume sizes for a range of scenarios. The scenarios were defined by different combinations of tank design, vadose zones, and ground-water velocities. The results were weighted according to the actual distribution of such factors. Wells were assumed to draw from the uppermost saturated zone, rather than from lower aquifers. No degradation of benzene was assumed to occur, but exposure was assumed to stop after a leak was detected. The number of people exposed was based on the national average density per acre of people using private or public wells times the modelled area of the plume of contaminated ground water.

ESTIMATED RISK

Yearly national cancer incidence was estimated to be less than one, both case where people were assumed to stop drinking contaminated water when the taste and odor threshold was exceeded and where they were assumed to continue drinking contaminated water.

UNCERTAINTIES

Major uncertainties in the analysis involve the rate of tank failures, the actual migration of and extent of contaminated ground water, and the effects of mitigating behavior on exposure. In addition, this analysis are preliminary and ongoing.

CATEGORY 4

#20: Nonpoint Source Discharges to Surface Water

PROBLEM DEFINITION

Chemicals can reach the aquatic environment not only from delineated point sources, such as industrial facilities, but also in the runoff of pesticides and stormwater from the land, infiltration from ground water, and air pollutants settling in water. Currently, because of the legislative framework, nonpoint-source pollution is dealt with through the ambient water quality criteria and standards program.

POLLUTANTS ADDRESSED IN THIS SUMMARY

A great number of environmental pollutants are of potential concern under this environmental problem. With respect to carcinogens, perhaps the greatest risk relates to the runoff of agricultural chemicals into surface water, primarily due to the large volume of substances entering surface waters in this way.

No specific pollutants are adressed in this summary, as cancer risk assessments have not been performed beyond that of the water quality criteria.

RISK ASSESSMENT METHODOLOGY

Cancer potencies developed by the Carcinogen Assessment Group or Office of Pesticide Programs are used when necessary. However, no quantitative cancer risk analysis is available for assessing the riks from this problem.

RISK ESTIMATES

There is no quantitative estimate of cancer risk for this problem. The work group ranked it relatively low due to limited human exposure, but the consensus view was that this was the most serious of the surface water problems.

CATEGORY 3

#21: Other Ground-Water Contamination

PROBLEM DEFINITION

Ground water can be polluted by an abundance of different sources, most of which are listed in a 1984 report prepared by the Office of Technology Assessment (see table). The majority of the listed sources that may release carcinogenic contaminants are conceptually addressed in discussions of other environmental problems in this document, such as hazardous waste sites, underground storage tanks, wastewater, sludge, agricultural pracctices, and landfills.

Table 1—Sources of Groundwater Contamination

Category I—Sources designed to discharge substances
Subsurface percolation (e.g., septic tanks and cesspools)
Injection wells
- Hazardous waste
- Non-hazardous waste (e.g., brine disposal and drainage)
- Non-waste (e.g., enhanced recovery, artificial recharge, solution mining, and in-situ mining)

Land application
- Wastewater (e.g., spray irrigation)
- Wastewater byproducts (e.g., sludge)
- Hazardous waste
- Non-hazardous waste

Category II—Sources designed to store, treat, and/or dispose of substances; discharge through unplanned release
Landfills
- Industrial hazardous waste
- Industrial non-hazardous waste
- Municipal sanitary

Open dumps, including illegal dumping (waste)
Residential (or local) disposal (waste)
Surface impoundments
- Hazardous waste
- Non-hazardous waste

Waste tailings
Waste piles
- Hazardous waste
- Non-hazardous waste

Materials stockpiles (non-waste)
Graveyards
Animal burial
Aboveground storage tanks
- Hazardous waste
- Non-hazardous waste
- Non-waste

Underground storage tanks
- Hazardous waste
- Non-hazardous waste
- Non-waste

Containers
- Hazardous waste
- Non-hazardous waste
- Non-waste

Open burning and detonation sites
Radioactive disposal sites

Category III—Sources designed to retain substances during transport or transmission
Pipelines
- Hazardous waste
- Non-hazardous waste
- Non-waste

Materials transport and transfer operations
- Hazardous waste
- Non-hazardous waste
- Non-waste

Category IV—Sources discharging substances as consequence of other planned activities
Irrigation practices (e.g., return flow)
Pesticide applications
Fertilizer applications
Animal feeding operations
De-icing salts applications
Urban runoff
Percolation of atmospheric pollutants
Mining and mine drainage
- Surface mine-related
- Underground mine-related

Category V—Sources providing conduit or inducing discharge through altered flow patterns
Production wells
- Oil (and gas) wells
- Geothermal and heat recovery wells
- Water supply wells

Other wells (non-waste)
- Monitoring wells
- Exploration wells

Construction excavation

Category VI—Naturally occurring sources whose discharge is created and/or exacerbated by human activity
Groundwater—surface water interactions
Natural leaching
Salt-water intrusion/brackish water upconing (or intrusion of other poor-quality natural water)

SOURCE: Office of Technology Assessment, 1984

For the purposes of this document, "other ground-water pollutants" are defined here as sources of carcinogenic contaminants that are unaddressed elsewhere in the document. Such sources may include residential waste disposal sites (e.g., septic tanks, dumps), stockpiles of materials, graveyards, railroad yards, and naturally occurring carcinogenic substances (e.g., arsenic and selenium), as well as unidentified sources of ground-water contamination.

Under the right hydrogeologic conditions, any substance released on the land surface or subsurface can contaminate ground water. The major problem in estimating cancer risks from ground-water contamination is a lack of information on what the sources are, where they are located, where they are actually contaminating the ground water, how significant the contamination is, and how many people it is affecting. Comprehensive information to answer these necessary questions is not available. Consequently, the extent of ground-water supplies contaminated by carcinogenic substances is unknown.

POLLUTANTS ADDRESSED IN THIS SUMMARY

The criteria for selecting sources were first to determine the sources of ground-water contamination, and then to select the sources that were most likely to contain carcinogens. Carcinogenic sources discussed in other sections were eliminated. For the remaining sources, the only cancer risk estimates available were for methylene chloride, which is used as a septic tank degreaser.

RISK ASSESSMENT METHODOLOGY

Cancer Potencies

Methylene chloride is the only potential carcinogen studied by EPA's Office of Policy, Planning and Evaluation (OPPE) for septic tank activity. Many carcinogens enter septic tanks from normal household use of chemicals (e.g., paint).

Exposure Assessment

Domestic septic tanks are cleaned usually every five years. This analysis assumed the worst case, which was that they are cleaned yearly.

A septic system includes septic tanks, cesspools, and the leachfields. Three categories of density of septic tank systems were analyzed: low density is septic tanks of less than ten units within one square mile, medium density is 0-40 units per square mile, and high density is greater than 40 units per square mile.

The relative distribution of the systems by location was derived from the 1980 Bureau of the Census Report and the 1970 Census of Housing Reports. Heath (1984) combined the map from the latter publication with a map of ground-water regions to arrive at density calculations.

Estimation of Risk

In the past, general procedures for estimating risks from ground-water contaminants have followed the flow chart in the attached figure.

The Liner Location model and the Prickett Random Walk Particle Tracking model produced ground-water concentrations of methylene chloride for over 400 years. These concentrations were then translated into doses. The risk estimate equation multiplies the dose by a dose-response factor:

$$R = 1 - \exp[-H \times (D-t)^k]$$

r = risk
H = potency of constituent
D = dose
K = factor describing shape of dose-response curve = 1 for carcinogens, = 1
t = response threshold, = 0(mg/kg/day)

Potential concentrations of carcinogens reaching ground water were calculated based on 64 hydrogeologic settings over 400 years. In all of the settings that were modeled, individual lifetime cancer risks did not exceed one in 10,000 (10^{-5})(based on the potency of methylene chloride). However, the risk fell between one in a million (10^{-6}) and one in 10 million (10^{-7}) in approximately half of the settings. Based on this, the OPPE study concluded that the risk presented by methylene chloride in septic systems is "not significant." Populations risks were not estimated in this report (U.S. EPA, 1986).

UNCERTAINTIES AND CAVEATS

The OPPE study is the first of its kind in defining carcinogenic and non-carcinogenic ground-water risks. It thus provides a basis from which to expand and improve ground-water risk analyses. However, the study has severe limitations for estimating cancer risk.

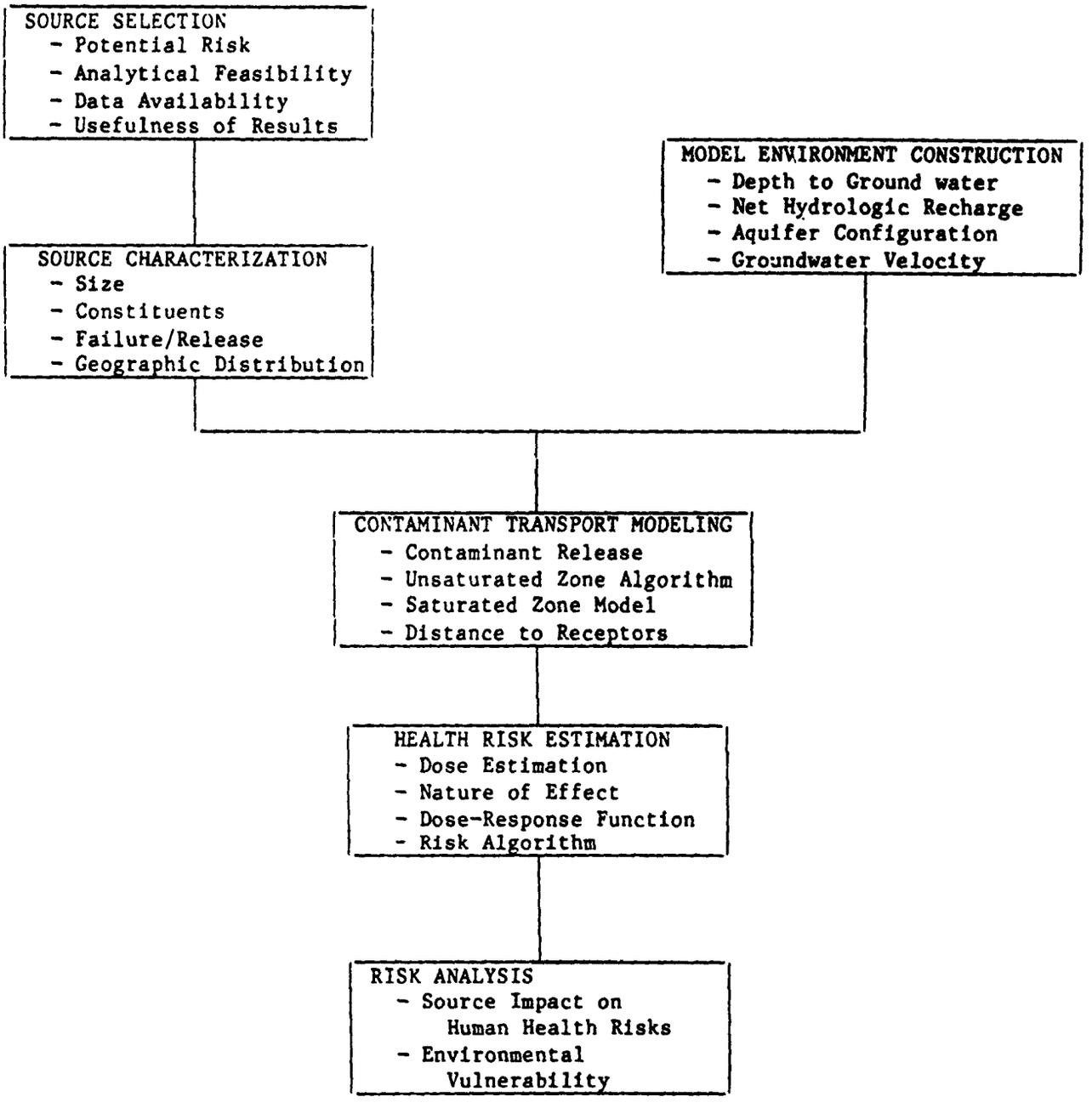
For example, the study calculates individual risk only and does not estimate population risk, which may be significant. However, a rough estimate of the population risk associated with this problem indicates a cancer incidence of less than one case every six years.

In addition, the following assumptions were made to reduce the time involved and simplify the complexity of this ground-water problem to make analysis feasible. The greater the number of assumptions, the less accurate the results are likely to be.

1. The models do not represent multiple sources or multiple chemical constituents. Septic tanks can release several carcinogens into ground water. Interactions between chemicals may greatly influence risks.

2. An average rate of release of the chemical was assumed. Because actual rates of release are not constant, failure and release rates are not characterized well.

METHODS AND ANALYSIS FLOW CHART



3. Important soil and hydrogeologic parameters remained constant across all environments. Actual hydrogeologic environments fluctuate significantly, and are not isotopic and homogeneous, as assumed in both models.

4. With respect to the hydrogeologic settings used for studying risk, calculations are probably underestimated. Once again, this is because septic tanks are placed in the best-drained areas locally, and these areas are the most vulnerable hydrogeologic settings.

5. Drinking water wells were assumed to be located 600 meters (1,800 feet) directly downgradient from the septic tanks. In actual residential settings, rural septic tanks and water well systems are most often less than 600 meters apart.

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CATEGORY 3

#22: Criteria Air Pollutants

PROBLEM DEFINITION

Sections 108 and 109 of the Clean Air Act provide for the establishment of national ambient air quality standards (NAAQS) for air pollutants from "numerous or diverse mobile or stationary sources." Termed "criteria pollutants," the six pollutants listed under section 108 are lead, particulate matter, nitrogen dioxide, ozone, sulfur oxides, and carbon monoxide.

ASSESSMENT OF RISK

Several of the criteria air pollutants and their metabolic or atmospheric by-products have been identified as possible human carcinogens or co-carcinogens. The human cancer risks associated with exposure to these pollutants have not been quantified due to inadequate toxicological, pharmacokinetic and epidemiological data. Therefore, estimating the cancer risks of the criteria pollutants is impossible at this time.

For the purposes of this project, the potential cancer risk associated with exposure to criteria pollutants should be considered to be low. However, to some extent this low ranking is an artifact of the way the work group defined this problem area.

To control ambient ozone concentrations, EPA has focused on preventing the emissions of diverse organic compounds, some of which are carcinogenic. In addition, carcinogenic particles, such as chromium and asbestos, make up a portion of what is controlled to meet standards for particulate matter. Indeed, it is likely that the criteria pollutants program has thus far been more successful in the control of airborne carcinogens than any other activity administered by EPA. However, the work group has elected to consider the risks associated with organics and carcinogenic particles under Hazardous/Toxic Air Pollutants.

A brief discussion of the carcinogenic potential of each criteria pollutant follows.

Lead

At relatively high concentrations, lead displays some evidence of carcinogenicity in experimental animals, such as the rat. Lead may act as either an initiator or a promoter of carcinogenicity. The role that lead may play in the induction of human neoplasia has not been established.

Epidemiological studies of workers exposed to lead provide no definitive findings. However, statistically significant elevations in respiratory tract and digestive system cancer in workers exposed to lead and other agents warrant concern. Also, since lead acetate can produce renal tumors in some experimental animals, it may be prudent to assume that lead compounds may be carcinogenic in humans (U.S. EPA, 1986a).

In light of the recent preliminary determination by the Carcinogen Assessment Group to classify lead as a probable human carcinogen (Class B2), consideration should be given to performing a quantitative cancer risk assessment for lead.

Particulate Matter

Particulate matter represents a broad class of chemically and physically diverse substances that exist as discrete particles (liquid droplets or solids). Levels of polycyclic organics and some inorganic carcinogens associated with particulate matter may have contributed to elevated cancer rates in urban areas during the high particulate pollution of the 1940-60s. Since then levels of some particulate carcinogens have declined, as has, presumably, their carcinogenic risk.

Studies of current U.S. air suggest that the mutagenicity of particle extracts is dominated by organic particle fractions that may not have been of significance in the past. The available evidence does not show that current particle exposures contribute to cancer, nor does it disprove any effect. The presence of mutagens in organic particulate fractions from unidentified sources and the potential interaction between these or other particles and carcinogens from cigarettes or occupational exposures suggest some need for caution and further study (U.S. EPA, 1982a).

Nitrogen Oxides

A few epidemiological studies have attempted to link environmental nitrates, nitrites, and nitroso compounds (derived from various oxides of nitrogen in the atmosphere) with human cancer. The criteria document for nitrogen oxides states that atmospheric nitrogenous compounds have not been shown to contribute significantly to the in vivo formation of nitrosamines in humans or that ambient air levels of nitrosamines represent a significant health hazard. There is no direct evidence that nitrogen oxides contribute to human cancer (U.S. EPA, 1982b).

Ozone

Except for the data on ozone-induced genotoxicity in peripheral blood lymphocytes, the potential genotoxic effects of ozone in humans is unknown. Epidemiological data on the contribution of ozone and other photochemical oxidants to human cancer are inconclusive. Recent studies have suggested that exposure of laboratory animals to ozone may increase the incidence of lung tumors. Due to the limited and equivocal nature of the available data, no conclusive statement can be made at this time regarding the potential carcinogenicity of ozone (Hassett et al., 1985; Last et al., 1986; U.S. EPA, 1986b).

Sulfur Oxides

Sulfur dioxide and bisulfite have been reported to be mutagenic in microbial test systems at acidic pH. Negative results have been reported for mammalian cells and insects. Inconclusive evidence suggests that SO_2 may be a carcinogen or co-carcinogen with benzo[a]pyrene. Available epidemiological studies neither prove nor negate the possibility that SO_2, acting alone or with particulate carcinogens, may contribute to cancer. Because of the

positive results of mutagenicity studies and the results of the cancer studies, the criteria document concludes that "SO_2 must remain suspect as a carcinogen or co-carcinogen" (U.S. EPA, 1982c).

Carbon Monoxide

There is no evidence to suggest that exposure to carbon monoxide poses a cancer risk.

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CATEGORY 4

#23: Direct Point Source Discharges to Surface Water

#24: Indirect Point Source Discharges to Surface Water

PROBLEM DEFINITION

The Criteria and Standards Division handles these problems together by addressing them in terms of ambient water quality criteria. Because of this approach, the Division is concerned with the universe of chemicals that reach surface water (both fresh- and saltwater) from industrial outfalls and treatment plant effluents. The approach also indirectly addresses contaminants from man-made and natural nonpoint sources by establishing maximum safe limits in water, regardless of the source.

POLLUTANTS ADDEESSED IN THIS SUMMARY

The chemicals in surface water originally addressed by EPA were mandated by consent decree: the so-called priority pollutants. Since 1980, little has been done in terms of human health.

Using lists prepared from EPA monitoring data, state information, and lists compiled by a number of offices within EPA, the Office of Water Regulations and Standards (OWRS) has compiled a list of 1,500 chemicals of concern. Of these, we ranked 150 chemicals based on human and aquatic toxicity, potential carcinogenicity, and exposure. This ranking yielded 45 chemicals that will be addressed during fiscal year 1987. Unfortunately, risk estimates are not yet available.

While this approach will eventually address about 10% of the chemicals on our current lists, we expect that it covers a much greater portion of the total problem. Since the chemicals on the list dictated by Section 307(a) of the Clean Water Act have already been looked at, at least half of the total problem will be addressed after we finish assessing the 150 chemicals we are now working on.

RISK ASSESSMENT METHODOLOGY

Cancer Potencies

OWRS generally uses potency estimates developed by the Carcinogen Assessment Group for all chemicals it is concerned with.

Exposure Assessment

The objective of the health assessment portions of the Ambient Water Quality Criteria Documents is to estimate ambient water concentrations of contaminants, which in the case of suspect or proven carcinogens, represent various levels of incremental cancer risk. These health assessments typically discuss four elements: exposure, pharmacokinetics, toxic effects, and criterion formulation. These are described below.

The exposure section summarizes information on exposure routes. Most criteria are based solely on exposure from consumption of water containing a specified concentration of a pollutant and from consumption of seafood that is assumed to have bioconcentrated pollutants from the surrounding water. The relative contribution of a pollutant to cancer risk varies with its propensity to bioconcentrate.

The pharmacokinetics section reviews data on absorbtion, distribution, metabolism, and excretion to assess the biochemical fate of the compounds in mammalian systems.

The toxic effects section reviews information on acute, subacute, and chronic toxicity, as well as specific information on mutagenicity, tetratogenicity, and carcinogenicity of the substance.

The criterion formulation section reviews highlights of the text and specifies a rationale for development and a mathematical derivation of the "criterion number."

Carcinogenic risks are estimated by extrapolation from animal toxicity or human epdemiology studies using the following basic exposure assumptions:

- 70-kg male person as the exposed individual;
- average consumption of fresh- and saltwater shellfish and other seafood products equal to 6.5 grams per day; and
- average ingestion of 2 liters of water per day.

The criteria based on these assumptions should protect an adult male experiencing average exposure conditions. The assessments, as indicated above, do not account for special segments of the population, or the possibility of exposures from other media.

RISK ESTIMATES

At present, risk estimates are not available for these problem areas. The cancer work group ranked them on the basis of their professional judgment, with no backing quantitative analysis.

UNCERTAINTIES AND CAVEATS

Much of the uncertainty associated with the program focuses on determining what pollutants may be present in the aquatic environment. Monitoring efforts have historically been directed toward the priority pollutants, so a fair body of data exists for them. Other substances have not been analyzed, so little information on exposure can be found. It follows that even less is known about their potential carcinogenicity.

There is also concern about the extent of the problem as related to sediments. They can act as a temporary sink for pollutants, which can be released over time to the water column.

In general, given the lack of cancer risk analysis, there is considerable uncertainty abount the magnitdue of these problems. The relatively low probability for large population exposure moved these problems to their low ranking.

Category 4

#25: Accidental Releases - Toxics

No information is available on which to base estimates of potential cancer effects. While such effects probably are minor due to the acute nature of exposure, they are likely to be higher than the effects from oil spills because of the toxic nature of these substances. Acute health effects and ecological effects would be of greater concern.

The cancer work group did not explicitly consider potential chronic exposures related to the ingestion of ground water contaminated by accdental releases.

#26: Accidental Releases - Oil Spills

The likely cancer effects from oil spills are negligible, primarily because exposure is likely to be acute. Of greater concern are welfare and ecological effects.

The cancer work group did not explicitly consider potential chronic exposures related to the ingestion of ground water contaminated by accdental releases.

CATEGORY 5 (no risk identified)

Biotechnology

EPA has little or no information that indicates that biotechnology may lead to increased risk of cancer. As research and analysis increase in this area, the magnitude of the potential environmental risks biotechnology poses will become better understood. However, cancer risks are not expected to be a major concern. For these reasons, the cancer work group could not identify any effect of biotechnology on the cancer incidence in the United States.

CO_2 and Global Warming

The long-term effects associated with a global warming as a result of anthrogenic emissions of carbon dioxide and other "greenhouse" gases are likely to be extremely wide-ranging. As a result, this environmental problem may indirectly affect cancer incidences in the United States. However, the cancer work group could not identify cancer risks associated with this environmental problem. Given the obvious major ecological and welfare effects of CO_2 and global warming, relatively little effort is planned in the near future to further assess the cancer impacts of this issue.

Other Air Pollutants

Under the set of definitions adopted by the cancer work group, outdoor air pollutants (other than criteria pollutants) that affect cancer incidence are being considered under Hazardous/Toxic Air Pollutants. For this reason, no cancer risk is attributed to this problem area.

NOT RANKED

To Estuaries, Coastal Waters, and Oceans from All Sources

To Wetlands from All Sources

These environmental problems focus on the receptors of pollutants, while other environmental problems are related to sources of pollutants. We chose not to rank them because the risks associated with them are discussed elsewhere under such source-related problems as Direct Point, Indirect Point, and Nonpoint Discharges to Surface Waters. While it was impossible to completely avoid accounting for the same risk under different problem areas, we generally tried to minimize double counting.