Health Risk from a Coal Tar Disposal Site

J. G. Bolten, P. F. Morrison,
S. A. Resetar, K. A. Wolf

40 Years
1948-1988

RAND

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PREFACE

This report is the sixth in a series of RAND documents that develop and apply a general methodology for assessing the chronic health risks of toxic pollutant emissions. The first report, *Risk-Cost Assessment Methodology for Toxic Pollutants from Fossil Fuel Power Plants* (R-2993-EPRI), developed the methodological framework and applied it in a case study of arsenic emissions from a hypothetical coal-fired power plant. The second, *Alternative Models for Risk Assessment of Toxic Emissions* (N-2261-EPRI), expanded the description of the methodology in the first report to discuss (1) alternative models for the framework, (2) simplified environmental transport models that may be used for initial analysis, and (3) implications of certain assumptions used in the exposure analysis. It also described the application of the approach, incorporated into a computer program WTRISK, to a case study of selenium emissions from a coal-fired plant. The subsequent three reports discussed the application of WTRISK to a case study of selenium and beryllium emissions from an existing coal-fired power plant in eastern Pennsylvania.

The present study applies the general framework and WTRISK to a study of a coal tar disposal site. The study investigates the potential health risks associated with emissions of five organic chemicals from the site. This effort, sponsored by the Energy Analysis and Environment Division of the Electric Power Research Institute, is directed toward federal, state, and local policymakers, as well as members of industry, concerned with risk assessment and pollution control regulation. The reports are intended to increase understanding of the toxic risk assessment process—an issue of increasing importance in recent years—and its inherent uncertainties.

SUMMARY

Coal processing was a major industry in the United States during the end of the 19th century and the first half of the 20th century. The carbonization processes produced coal tar as a residual waste product. Although some of this coal tar could be sold or processed, much of it ended up in shallow disposal ponds near the processing sites.

These disposal sites are now becoming a matter of concern. Although coal tar itself is not now considered to be a hazardous waste, some of the chemicals found in coal tar have adverse effects on human health and the environment. Many of these chemicals, including benzene, toluene, benzo(a)pyrene, and phenanthrene, have been identified and controlled in legislation such as the Clean Water Act and the Resource Conservation and Recovery Act. Accordingly, both utility personnel and government officials are paying increasing attention to the potential long-term health hazards posed by old coal tar disposal sites.

During 1983, personnel at a major electric utility began investigating past gas production activities in companies acquired through merger and consolidation. This work identified a number of sites that currently contain coal tar deposits. These sites are currently being monitored and studied to determine if any of them constitute a significant threat to the environment or human health.

This situation presented an opportunity for us to study the problem of risk assessment at coal tar disposal facilities. We have previously developed and applied a general methodology and computer program, WTRISK, to calculate the chronic human health risks of toxic emissions from fossil-fuel-fired power plants. The present study describes the application of the general approach (including WTRISK) to the risk assessment of an identified coal tar disposal site. This site is located in a small eastern city on the banks of a minor river.

In the study, using data collected by the utility and information developed for them by contractors, we investigated the potential risk to human health and the environment from this site. The exposure pathways considered include surface water contamination from leaching into local groundwater, atmospheric emissions of volatile pollutants, and ingestion or dermal contact with contaminated soil and dust from the site. The study examined five organic pollutants—benzene, toluene, xylene, naphthalene, and phenanthrene—chosen to be representative of the chemicals found at the site. For each pollutant, we studied environmental transport and fate, possible exposure pathways, the characteristics of the exposed population, and the nature and extent of adverse chronic health effects.

The environmental transport analysis was performed using WTRISK and the CDMQC atmospheric transport model. Pollutant concentrations in the air, surface water, and soil and dust were determined for a number of alternative cases, designed to reflect the basic uncertainty in many assumptions, parameters, and input values. For each alternative case, we determined the exposed population, their exposure rates for each pollutant, and the consequent health risks.

We found that, in general, emissions of the five pollutants from the site pose little risk to the surrounding environment and population. Surface water concentrations of the pollutants should be low, and the affected river is not used for either drinking water or irrigation. The risk associated with current fish consumption and oyster production is not significant. Should the general quality of the river improve sufficiently, so that it could be used as a water source, the potential risk would increase. Under those circumstances a more thorough examination of the risk from all pollutants found at the site would be justified.

If the site is disturbed through excavation or land treatment, benzene and other volatile pollutants may be emitted to the air at higher rates than they are now. It is unlikely that the volatile emissions from the site will pose significant health hazards to the neighboring population. Although there is considerable uncertainty about potential emission rates, even in the worst case, dispersion throughout the environment will cause exposure rates to be below hazardous levels.

The results are less conclusive for exposure through contact with soil and dust particles that may be picked up by the wind from an uncovered tar pond. Conservative calculations show that the volatile pollutants in dust and soil should pose little problem. Unfortunately, these same assumptions, which produce worst-case exposure rates, find that potential exposure from naphthalene and phenanthrene could be important. Although we know that the results are an upper bound, it is not possible to say how much lower the actual exposure rates would be. We can say, however, that sufficient care to minimize fugitive dust and soil emissions (from an uncovered or excavated site) should reduce the potential health risks to negligible levels.

Persons working at the site frequently for long periods should be protected from inhalation and dermal exposure to the coal tar and contaminated soil. The potential health hazards, particularly for benzene and phenanthrene, or other carcinogenic polynuclear aromatic hydrocarbons (PAHs), will be higher for these personnel. The site should continue to be fenced and locked to prevent children from playing near or on the tar pond.

Major uncertainties affect the results and conclusions of this work. Because the analysis was intended to demonstrate an approach as well as investigate the risk, we examined only five pollutants. There may be higher risks associated with other pollutants at the site, although the pollutants examined in the study were chosen in part because of their high concentrations in the coal tar. Moreover, the adverse health effects caused by many of the pollutants have not been examined by researchers. Very little information is available for many of the chemicals present in coal tars. This deficiency makes it difficult to assess the potential health hazards of coal tar deposits in general, and tends to force the analysis toward extremely conservative calculations.

Finally, the health risks determined by this study are based on calculated values of emission rates to the groundwater, surface water, and air. If the tar pond or lake bed deposits are to be disturbed for any reason, additional monitoring of air, soil, and groundwater pollutant concentrations should be instituted to ensure that emission rates do not increase to levels significantly higher than those assumed in the analysis.

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I. INTRODUCTION

Coal carbonization processes were a major means of producing gas in the United States during the years before the end of World War II. Coal tar is a residual waste of these production processes. It was commonly sold for use in producing roofing materials, road tars, industrial fuels, wood preserving chemicals, and paints and enamels. That part of the tar that could not be sold or processed for other chemicals was generally placed in shallow ponds or wells near the gas processing site and subsequently covered with soil and other materials.

Coal tar itself is not currently considered to be a hazardous waste. However, some of the chemicals in coal tar can adversely affect human health and the environment. During the period of coal processing, there was little understanding of the importance or potential risk from the chemicals in coal tar. Consequently, coal tar wastes slowly accumulated at many sites scattered across the country. Because of the processing and disposal practices used in the industry, many sites have significant deposits of coal tar and similar wastes in their soil. These materials can volatilize into the air, leach into groundwater, or reach surface waters through runoff and leaching from the soil.

Only recently have people become aware of the potential problem that these wastes represent. Their presence in soil, water, and air could have adverse effects on the environment and on human health. Many of the chemicals contained in coal tars have been identified as toxic in legislation such as the Clean Water Act (CWA) and the Resource Conservation and Recovery Act (RCRA). Some of these chemicals are benzene, toluene, xylene, naphthalene, anthracene, phenanthrene, and benzo(a)pyrene.

As a consequence of these developments, present and past owners of coal processing facilities and sites, as well as state and federal authorities, are currently investigating the potential health and environmental risks posed by coal tar deposits. The general process involves identifying the site, characterizing its wastes (through geological and hydrological monitoring and knowledge of plant operations), and assessing the potential health and environmental risks.¹

BACKGROUND

Site Investigation

During 1983, personnel at a major utility began investigating past gas production activities in companies acquired through merger and consolidation during previous years. The research identified a number of company-owned sites that currently contain coal tar deposits. Company management subsequently approved the field investigation of these sites, to determine the effects of site emissions on local groundwater quality and to assess any potential risks to human health and the environment.

One of the surveyed coal tar disposal sites was selected for investigation, because it contained relatively large deposits of tar and showed a potential for groundwater contamination. A plan for the study was prepared and submitted to the state Environmental Protection Department (EPD). The investigation sought to determine the (1) size and composition of the

¹In this study we investigate only the potential health hazards associated with coal tar disposal sites. The possible occupational exposure and health risks in industries that use coal tar products (such as roofing tar and road tar) lie outside the scope of this analysis.

deposits, (2) potential for soil, groundwater, and surface water pollution, and (3) potential risks to the environment and human health. In this report we describe the assessment of the potential for human health and environmental risks.

Risk Assessment Framework

The general approach used in this study and its application to other case studies have been described in a series of reports. The initial report (Bolten et al., 1987) describes the formulation of the general methodology for assessing the relative costs and risks associated with the control of toxic substances emitted by coal-fired power plants. The method emphasizes contamination of surface water bodies but includes and follows toxic emissions through all environmental media. Pollutants are tracked through the environment to determine population exposure rates and the consequent chronic health effects on the regional population.² The second report (Bolten et al., 1985) discusses the general types and features of surface water transport and exposure models and briefly reviews some of the specific models available in both areas. The report also describes the development of the relatively simple environmental transport models for atmospheric, overland, and surface water transport. These models have been incorporated into a computer program (WTRISK) that also includes procedures for calculating exposure rates for common pathways and the consequent human health effects.

These first two reports also described the application of the WTRISK computer program to the analysis of arsenic and selenium emissions from hypothetical coal-fired power plants located in different areas of the country. Although both of these case studies were valuable in demonstrating how one would apply the approach to an actual problem, they were of limited interest. Because they dealt with hypothetical power plants and scenarios, the studies could produce results with only very general conclusions.

In contrast to these earlier studies, the subsequent reports applied the models of the WTRISK program to an actual situation. They investigated the chronic health effects associated with the selenium and beryllium emissions to surface waters from the Montour power plant operated by Pennsylvania Power & Light Company (PP&L). These studies focused on population exposure through consumption of drinking water, fish, and several animal and vegetable products produced on land irrigated with the surface waters affected by Montour discharges.

In this study, the general approach and the WTRISK program are applied to the problem of assessing the potential long-term chronic health risk associated with the emission of five organic chemicals from the selected coal tar disposal site. The five chemicals (benzene, toluene, xylene, naphthalene, and phenanthrene) were selected to be representative of the different classes of chemicals found at the site. Although the report discusses the analysis of emission rates, transport, exposure, and health risk for these chemicals, it emphasizes the calculation of potential exposure rates and chronic health risks.

Procedure

The overall investigation was separated into three areas, based on the different types of exposure pathways considered. These consisted of (1) surface water transport and consumption of drinking water and aquatic organisms, (2) atmospheric transport and inhalation of

²Chronic health effects are cumulative effects caused by long-term exposure at relatively low levels, rather than the acute effects associated with short-term or one-time exposure to high pollutant concentrations.

volatile pollutants, and (3) exposure through ingestion and dermal contact with soil and dust particles from the site. The analysis used a surface water transport model in WTRISK to determine how site emissions affect pollutant concentrations in the local surface waters. A separate atmospheric transport model called the Climatological Dispersion Model (CDMQC) was used to estimate dispersion of volatile pollutants from the site. The concentrations calculated with the transport models were used with the exposure model in WTRISK and other procedures to calculate the consequent exposure rates to the affected populations.

In most of the transport and exposure analyses, we used a base case situation and several alternative scenarios. These alternatives were selected to emphasize the importance of alternative assumptions and parameters. The base case situation assumed nominal emission and removal rates, mean flows, and nominal values for all parameters in the exposure calculations. In the alternative cases, parameter values and scenario assumptions were varied in a number of ways to determine how pollutant concentrations and exposure rates changed under different, reasonable assumptions. The results of the exposure calculations for alternative cases were converted into estimates of the future chronic health risks for the regional population.

The health risks for exposure to the different pollutants were calculated using dose-response functions developed from the literature, particularly information reviewed and summarized by the Environmental Protection Agency (EPA) in their water quality criteria documents. The specific health effects and potential toxicity of the pollutants varied widely, as did the availability of suitable data for developing dose-response functions. As we have found before, most of these pollutants have not been adequately studied, and quantitative assessment of their adverse health effects is extremely difficult and uncertain.

Section II of this report describes the general risk assessment framework, including the analysis procedure and potential exposure pathways. Section III discusses the case study and presents the results of the analysis. Appendix A contains the calculations of potential emission rates for volatile pollutants from the tar pond at the site. Appendix B presents discussions of the health effects of all pollutants, including a derivation of our approaches to calculating chronic health risks.

RESULTS AND CONCLUSIONS

In general, the emissions from the coal tar disposal site do not appear to pose a significant hazard for the local environment or the population in the region. This conclusion is somewhat uncertain for several reasons. First, we investigated the potential health risks from only five pollutants. Although these pollutants were selected to be representative of their various classes of organic chemicals at the site, they do not include all potentially hazardous pollutants that could be found there. Second, there is commonly very limited information (qualitative or quantitative) about the adverse health effects of the organic chemicals typically found in coal tar sites. As a consequence, although it may be possible to estimate exposure rates for various environmental pathways, it can be extremely difficult to translate these exposure rates into meaningful measures of health risk. We have tried to use the best information available in this analysis, but that information is sadly limited for many of the pollutants.

The analysis calculated exposure rates and health risks associated with exposure through three types of pathways: surface water, atmosphere, and soil or dust. The results of the surface water transport and exposure calculations indicate that there is very little risk from future site emissions, given the current state of the adjacent river and its uses. The only meaningful current exposure pathways involve consumption of fish and oysters from the downstream

harbor area. Numerous worst-case assumptions were required to make these pathways potential sources of exposure for a significant population. The health risks of site emissions for these pathways are not important. If the river were to be used as a source of drinking water, it would be necessary to evaluate the potential for exposure and consequent health risk in more detail for all of the important pollutants.

The other pathways considered were exposure through inhalation of volatile organic chemicals and dermal contact with, or ingestion of, airborne dust and soil particles from the site. For both of these pathways, the calculated health risks seem to be small for the population of the surrounding town as a whole and the people living within a kilometer of the site. Exposure rates are somewhat higher for personnel located directly at the tar pond, i.e., either workers on the site or children playing in the area. Because the site is fenced to keep out unauthorized personnel, it is unlikely that these higher, but not necessarily dangerous, potential exposure rates constitute a significant hazard for the population.

We compared predicted levels of the pollutants in the river with current EPA water quality criteria for protection of aquatic life. Although the data are again extremely limited, the potential concentrations of pollutants in the river are typically 1,000 times lower than the concentrations that have been found to be hazardous to aquatic life. For that reason, we feel that it is unlikely that the emissions from the coal tar site will constitute a hazard to the environment.

The primary purpose of this work has been to demonstrate an approach to risk assessment of coal tar disposal sites and to form general conclusions about their potential for adverse health effects and environmental damage. The conclusions drawn from our results are conditional on the limitations of the information used in the analysis. They could be changed if revised data show the inputs to, or the assumptions behind, the analysis to be questionable. Such additional information could involve the existing concentrations of the pollutants at the site, their mobility and fate in the environment, or the type and extent of adverse health effects associated with any of the pollutants.

II. RISK ASSESSMENT FRAMEWORK AND PROCEDURE

Coal tar disposal sites can release toxic materials into the air, soil, surface water, and groundwater. Once emitted, these pollutants may be dispersed and transported throughout the area, while undergoing degradation and decay, adsorption, transformation, and biological uptake processes. Pollutants may also be transferred between media, such as when organic chemicals released to groundwaters reach a nearby lake or river. To determine the ultimate chronic health risk associated with such emissions, one must be able to follow them through the environment and to calculate the long-term concentrations in all media of the area surrounding the emission source.

Pollutants in the environment reach the population through numerous exposure pathways that can be grouped into three general intake modes: inhalation, ingestion, and dermal absorption. Not all of these intake modes will be important for any given pollutant. Risk assessment procedures must be able to convert pollutant concentrations in the environment into exposure rates for each pathway. In addition to direct pathways, such as inhalation or consumption of drinking water, exposure may occur through consumption of animal and vegetable products at various positions in the food chain.

To assess whether the calculated exposure rates constitute a significant hazard to the population, the dose of each pollutant over time should be evaluated for each geographically and biologically distinct portion of the population. These doses are used with dose-response models to estimate the increase in health risks for a particular response (or set of responses). Because we are concerned with chronic (long-term) health risks, we are particularly interested in dose-response models of chemical carcinogenesis.

The framework for this risk assessment process consists of five distinct stages: source generation, environmental transport, exposure calculation, toxicology and pharmacokinetics, and dose-response modeling. To assess the risk associated with emission of a toxic pollutant from a specific source, one must use models, measured data, or a combination of both at each stage of the analysis.

The general risk assessment method is shown schematically in Figure 2.1. A pollutant enters the environment through source emissions. The emission rates serve as the inputs for environmental transport models that calculate the distribution of pollutant concentrations in the different media (air, soil, groundwater, and surface water). As shown in the figure and explained below, the transport models may be either relatively simple and aggregated (Level I) or more detailed and complex (Level II). The pollutant concentrations determined by the transport models are used in the exposure analysis to calculate exposure rates as a function of location and type of exposure (e.g., inhalation or consumption of drinking water, animal products, or fish). These exposure rates can then be combined with local population data to determine a distribution of exposure rates for the region. Knowledge of the toxicology and pharmacokinetics of the pollutant are used with available data describing its health effects to develop appropriate dose-response models. Using the distribution of exposure rates as an input into one or more of these models, we calculate the expected chronic health risk associated with the pollutant emissions from the tar source.

Fig. 2.1—Risk assessment approach

Figures 2.2 and 2.3, modified from Bolten et al. (1987), present a schematic outline of the detailed risk assessment process.¹ Figure 2.2 traces the toxic pollutant from the tar source through the environmental transport stages. The source may emit organic compounds into the air, surface water, and soil or groundwater. Discharges into the different media are dispersed, transformed, and transferred between media. The figure indicates these general processes and the interactions between media that must be considered in determining the distribution of pollutant concentrations in the region surrounding the source. The models for atmospheric, overland, groundwater, and surface water transport are shown as darker rectangles in the figure.

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

The method has been designed to determine chronic health risks for the regional population. By limiting the assessment to chronic risks, we can restrict the study to long-term average emission and exposure rates. This permits the use of steady-state models in the environmental transport and exposure calculations, considerably simplifying the calculation procedure.

Complex phenomena are involved in pollutant transport and transformation processes. These phenomena make it difficult to calculate the distribution of pollutant concentrations.

¹In these figures, rectangles indicate processes or activities. Plant emissions or wastes are shown by slanted rectangles, and hexagons indicate pollutant concentrations in either the environmental media or parts of the exposure network. The arrows indicate the general flow of pollutants through the environment and the risk assessment process.

Fig. 2.2—Risk assessment framework — part I

Fig. 2.3—Risk assessment framework — part II

Frequently, complex environmental transport models are used for these calculations. Such models attempt to incorporate all relevant phenomena and should be used in the detailed analysis of pollutant emissions problems. However, these models are expensive to use and require large amounts of time and data collection and preparation. Accordingly, before beginning the extensive data collection and analysis effort needed to apply these models, it may be appropriate for the user to perform a first-order Level I (or screening) analysis.

This Level I analysis uses simplified calculations of pollutant concentrations to determine the relative importance of different media and exposure pathways and to calculate upper and lower bounds on concentrations and exposure rates. With this information, the analyst can develop a better understanding of the problem, direct study effort and resources to the most important areas, improve selection of more complex models for subsequent Level II analysis, and reduce the need for subsequent iterations between complex models and other revisions of the study.

The Level I models used in this type of preliminary analysis, like the more detailed and complex Level II transport models, can be used to calculate pollutant concentrations and transfer rates between media. They are simplified models that use limited, aggregated data and can be run quickly and cheaply. Accordingly, they are appropriate for extensive sensitivity analysis and the development of approximate results and upper or lower bounds on concentrations, transfer rates, and subsequent exposure rates through the various pathways. Requiring limited amounts of data and other resources, these models can quickly produce results that permit the user to better understand and define the problem and solution approach, to isolate important aspects of a problem, and to determine where further detailed analysis is necessary.

The Level I models have been incorporated into a computer program that also contains routines for calculating exposure rates and chronic health effects using a variety of alternative dose-response models. This program, called WTRISK, is described in Bolten et al. (1985), which includes a discussion of the primary program variables, inputs and input formats, the types of output generated by the program, an example of the inputs and outputs associated with a typical analysis, and the listing of the computer code.

In the current analysis, we used the WTRISK program and other models to investigate the chronic health risk associated with emissions of five selected organic compounds (benzene, toluene, naphthalene, xylene, and phenanthrene) from an existing coal tar site. The source term (emissions) included leaching into groundwaters with subsequent transfer into surface waters, soil contamination, and gaseous and particulate emissions to the air. These emissions were used as inputs to the Level I surface water transport model, the CDMQC air model, and a simplified calculation of dust transport to determine pollutant concentrations in the area. Exposure routes that were considered included direct ingestion of treated drinking water, consumption of aquatic organisms from affected waterways, inhalation of volatile organic emissions from the tar, and ingestion and dermal exposure to dust from the primary tar deposit. The exposure rates calculated for these routes were combined with data describing the regional population and the potential health effects of the pollutants to estimate the incremental health risk associated with source emissions.

III. ANALYSIS OF THE CASE STUDY

BACKGROUND AND DESCRIPTION OF STUDY

In this case study we investigate the risks associated with potential emissions from a coal tar disposal pond located on the former site of a coal processing plant in an eastern state. The pond is situated adjacent to a fairly small river, which ultimately discharges into a harbor on the Atlantic Ocean. This analysis assesses the potential human health risk associated with exposure to five organic pollutants found in the source and present in the air, soil, groundwater, and surface waters.

In the analysis, emission rates are based on calculated concentrations and intermedia transfer rates. These are used as the source terms in the environmental transport calculations but are compared to measured concentrations and transfer rates whenever possible. The transport calculations are made using the simplified procedures of the WTRISK program, the CDMQC model, and empirical equations developed from the literature. These calculations determine pollutant concentrations in the air, dust, and surface waters of the region surrounding the plant. The concentrations are the basis for calculating exposure rates and health effects in the exposed regional population. The potential exposure routes for the analysis include ingestion of drinking water and aquatic organisms, inhalation of gases and dust, and ingestion of, and dermal contact with, dust and soil particles from the source.

The subsections that follow briefly describe the disposal pond and surrounding region, the properties of coal tar, and the chemicals selected for the risk analysis.

Description of the Area

Disposal Site. This risk assessment investigates the potential chronic health risk to humans from coal tar deposits at a former gas-production facility. This facility, located in a small town (Town A) in an eastern state, is currently owned by a major electric utility company. It began operation during the late 19th century as a water gas plant. After a merger during the 1930s, the plant was subsequently converted to produce carburetted water gas. It later ceased operations entirely and was closed down in the mid 1950s.

During its operation, the plant produced about 1.5 million cubic meters of gas per year, resulting in approximately 11,300 cubic meters of tar. Much of this tar may have been sold, but large amounts remain in the pond. During recent explorations, the utility found two primary tar deposits: (1) a tar pond approximately six meters thick with six meters of tar-saturated soil below it, and (2) a lake-bed deposit about 1.5 meters thick with three meters of tar-saturated soil below it. The two deposits cover approximately one hectare with a volume of 42,000 cubic meters. The saturated soils below the deposits have a volume of 50,000 cubic meters.

Figure 3.1 shows the location of the source. It is bounded on the west by a flat area that slopes down to the floodplain of the adjacent river (the JK River). This floodplain was at one time a lake on the river. The property and land adjacent to the north and south are zoned for residential development, whereas the land to the east is industrial. Current land use is not expected to change in the foreseeable future. Figure 3.1 also shows the location of the two tar deposits located on the general site.

Fig. 3.1—Site map with disposal areas

Surface Water Network. The JK River is located about 200 meters west of the tar pond. The river originates about 25 km north of the area and flows generally southward through a number of towns until it reaches the harbor at City B. The last several kilometers of the river south of the area and before the City B Harbor form an estuary that contains oyster seeding beds. At its southern end, the JK River empties into the City B Harbor and the Atlantic Ocean, as shown in Figure 3.2. The river drains 430 square kilometers and has a mean flow rate of 8.47 cubic meters per second. The seven-day ten-year low flow rate is 1.25 cubic meters per second. In the estuary area, both tidal flow and water diversions provide additional dilution of pollutants carried downstream by the river.

The river generally has poor water quality. In the most recent survey by the state EPD, the river was classified in the area and downstream as SC/SB. This means that the river is "suitable for fish, shellfish and wildlife habitat; suitable for recreational boating and industrial cooling; good aesthetic value." The river is not suitable for bathing, swimming, or drinking water supply, even after treatment. Harvesting of shellfish for direct consumption is prohibited in the harbor and in the estuary. All shellfish must be transferred to unpolluted waters for at least one month for depuration of pathological organisms before sale or consumption. In spite of the pollution, the estuary and harbor areas support a large and valuable oyster seeding industry.

The water quality of the river has been adversely affected by a number of different pollutant sources. The basin was settled early in colonial times and is one of the most densely developed areas of the state. Industries along the river divert water for use in plant operations, but almost all of it is returned to the river. The river currently receives discharges from 21 industrial sites, five municipal and two private sewage treatment plants, landfill leachate, and combined sewer overflows from the City B sewer system. Because the river is so small compared with the number of wastewater dischargers, both municipal and industrial dischargers have to use a higher degree of treatment than is normally required. Although water quality has improved in recent years with the reduction or elimination of some point sources, the river will likely not be acceptable for swimming or fishing in the foreseeable future.

The City B Harbor, which receives the flow of the JK River, is also adversely affected by the discharge of inadequately treated sewage from two primary treatment plants. Although this problem is being corrected, it is unlikely that the quality of the harbor water will improve significantly for a number of years until the combined sewer system in City B is replaced.

Recreational use of the river is currently limited to boating. Although the state has recently stocked some of the upstream area with fish (as an experiment), there is no fishing downstream before the water reaches the harbor area. In the harbor there is limited fishing for anadromous and marine species. Currently Town A has been excavating sand and gravel from the bed of the former lake to use as cover in the local landfill. There have been discussions of redeveloping a recreational lake (called the City Lake) at the site of the former lake, although no definite plans have been made for this action.

Groundwater and Drinking Water Resources. Drinking water in the JK River Basin is obtained primarily from groundwater wells. Town A itself is supplied primarily by two local water departments. The groundwater quality in the local area has been rated as GB by the EPD. This means the water "may not be suitable for public or private use as drinking water without treatment . . . groundwaters specified as GB are known or presumed to be degraded." This classification generally means that drinking water is obtained from municipal supplies that use other sources. New discharges to the groundwater are unlikely under current EPD water policy, and private or municipal supply wells are also not expected to be developed.

Fig. 3.2—Case study river basin

In general the groundwater quality in the JK River Basin has suffered from significant degradation. Of the approximately 70 large public supply wells, 21 are known to be contaminated or believed to be threatened by nearby land use, waste disposal practices, or other sources of potential contamination. Sixteen of these wells are located along the JK River corridor, and four towns, including Town A, have been forced to shut down major wells, primarily because of contamination by organic solvents (not coal tar contaminants).

There are no municipal or private surface or groundwater supply sources within one mile of the disposal area. Exploratory wells in the 1960s produced large quantities of water, but municipal wells were never developed because of uncertainty about the location, extent, and effects of the potential tar deposits and other pollutant sources.

A hydrologic study of the area was conducted to determine the direction and rate of groundwater movement in the area. In the study, the electric utility drilled 20 wells at 11 locations to monitor up-gradient and down-gradient hydrologic and chemical conditions (both vertical and lateral) at varying depths. Water level measurements were taken at least once a month to determine seasonal changes in levels and general flow conditions.

The study found that groundwater movement in the area is unconfined and generally toward the JK River. Vertical gradients indicate that the entire flood plain and lake bed is a discharge zone for the area. The river is the principal discharge point for the local groundwater, although there is also minor discharge at a nearby pond. The water table level fluctuates seasonally, but the general flow direction remains unchanged through the year. The monitoring study also provided evidence that dredging along the river banks has caused the flow to be directed more sharply toward the downstream section of the river. Other geological, meteorological, hydrological, and geographic data for this location were obtained from a variety of sources, including topographic maps.

Characteristics of Coal Tar

Coal tar does not have a standard composition. It generally consists of a mixture of (1) polynuclear aromatic hydrocarbons (PAHs) such as benzo(a)pyrene, naphthalene, anthracene, acenaphthene, and phenanthrene; (2) phenolic compounds including phenol and methylphenols; (3) light aromatic compounds such as benzene, toluene, and the xylenes; (4) miscellaneous organics such as dibenzofuran; and (5) small quantities of inorganic chemicals such as iron, lead, copper, zinc, various sulfides, cyanides, and nitrates. The physical characteristics and chemical composition of the tar depend on the kind of coal utilized, the type of process, and the temperatures used in coal processing (Anastos et al., 1983).

Coal tar deposits in soil can divide into four fractions. The first consists of solid or semi-solid tars that tend to remain where deposited. The second is a liquid fraction heavier than water, which can sink until it reaches an impermeable clay or bedrock layer and then move horizontally. The third fraction is also liquid, but lighter than water, which floats and moves on the groundwater surface. The last fraction is soluble and dissolves in the groundwater.

Many characteristics influence how far the liquid fractions can move in the groundwater. When chemicals are leached from a source, the extent of the subsequent pollution depends on (1) the size of the source, (2) the porosity, permeability, and chemistry of the soil, (3) the local groundwater gradient, and (4) the solubility of coal tar chemicals. Under some conditions, groundwater contamination can move considerable distances, and large deposits can act as contaminant sources for many years.

Pollutants

As discussed above, coal tar does not have a standard composition. Although samples from various areas may contain similar chemicals, the relative concentrations will be affected by many different influences. Because tar deposits contain so many different organic and inorganic compounds, it is impossible to consider all of them in risk assessment activities. Detailed analysis must be limited to a few important chemicals selected to be as representative as possible of the majority of constituents.

In this study, we limited our consideration to five chemicals. These chemicals were chosen on the basis of (1) their presence in the coal tar deposits in significant concentrations, (2) their potential to cause human or environmental risk, and (3) their ability to travel through the groundwater to reach the river at different rates. An initial analysis supported by the utility considered the physical, chemical, and biological properties that might affect transport of the chemicals through the groundwater. Each chemical was ranked in terms of its adsorption characteristics, being grouped into very high, high, moderate, and low adsorption categories.

For the analysis, we selected a chemical from each adsorption group. We chose a chemical that was present in relatively high concentrations in the tar pond deposits and tried to vary the type of health effects that would have to be considered in the analysis. Accordingly, we structured our investigation around (1) benzene (low adsorption), (2) toluene (medium), (3) the xylenes (high), (4) naphthalene (high to very high), and (5) phenanthrene (very high). We discuss each chemical in more detail below.

Benzene. Benzene is a volatile, colorless, liquid hydrocarbon commonly produced from coal tar distillation, coal processing, and coal coking (EPA, 1980d). It is generally used as an intermediate in synthesis processes by the chemical and pharmaceutical industries. It is also used as a lacquer thinner, a degreasing and cleaning agent, and a solvent. Benzene has the molecular formula C_6H_6 and is less dense than water. It is highly soluble in water and volatile and thus may be mobile in the environment. It is possible that benzene could be washed out of the atmosphere with rainfall and subsequently be evaporated back into the atmosphere in a continuous cycle. It has been detected at various concentrations in the atmosphere, lakes and streams, and in treated drinking water (EPA, 1980d). The measured concentration in drinking water supplies ranges between 0.1 and $10\mu g/l$. Benzene should be photo-oxidized in air and also biodegraded in the environment. Unfortunately, very few data about benzene concentrations in food and water are available.

Toluene. Toluene is a clear, colorless, noncorrosive organic liquid. It is primarily used in the production of benzene and other chemicals and as a paint solvent and gasoline additive (EPA, 1980a). Toluene is a volatile and flammable aromatic hydrocarbon. It is similar to benzene in structure, except that a methyl group is substituted for one hydrogen atom. Toluene has the molecular formula C_7H_8 and is less dense than, and only slightly soluble in, water.

The transport and persistence of toluene in the environment is not well understood (EPA, 1980a). Of the estimated 700,000 tons discharged annually by industry, over 99 percent is as atmospheric emissions. In the atmosphere it can be degraded photochemically and can also reenter the hydrosphere in rain. Because it is volatile, toluene can evaporate easily from water to the atmosphere.

Toluene has been found both in raw water and treated drinking water supplies, in concentrations ranging from 0.1 to $19\mu g/l$ (EPA, 1980a). Very few data about toluene levels in foods are available. Two primary metabolites of toluene—benzaldehyde and benzoic acid—either occur naturally in some foods or are used as food additives.

Naphthalene. Naphthalene, the most abundant constituent of coal tar, is a bicyclic aromatic hydrocarbon produced from coal tar or petroleum (EPA, 1980b). It is used as an intermediate in the production of dyes, in the formulation of solvents, lubricants, and fuels,

and also in the production of moth balls. Naphthalene has the molecular formula $C_{10}H_8$ and is slightly soluble in water. It can be oxidized in the presence of light and air. Naphthalene has been detected in ambient water and drinking water supplies at concentrations up to $2.0 \mu\text{g/l}$ (EPA, 1980b). Very little is known about potential concentrations in food products.

Xylene. Xylene is a colorless liquid aromatic hydrocarbon. It is similar in structure to benzene, except that methyl groups are substituted for hydrogen atoms in two locations. There are three isomers of xylene (ortho, meta, and para), depending on the relative locations of the substituted methyl groups. The molecular formula for any of the xylenes is C_8H_{10} . They have historically been obtained from the byproducts of conversion of coal to coke, but in recent years xylenes are more commonly produced by other methods. Xylenes are used in fuels, solvents, and as chemical intermediates in the production of phthalic acids and derivatives.

Studies show that the mixture of xylenes and the individual isomers are only moderately toxic in acute, subacute, subchronic, and chronic tests (Jori et al., 1986). The three isomers tend to have the same biological activity. For this reason, we shall consider total xylenes in the risk analysis and will not follow the isomers individually.

Xylenes are highly volatile and can undergo significant photo-oxidation in the presence of the hydroxyl radical, but their biodegradability is not well understood and probably not important (Jori et al., 1986). They have been found in concentrations ranging between 3.0 and $8.0 \mu\text{g/l}$ in drinking water (Merian and Zander, 1982).

Phenanthrene. Phenanthrene is a PAH, and like all PAHs it is formed as a result of incomplete combustion of organic compounds with insufficient oxygen. The individual members of the group of PAHs have not been studied extensively, except for benzo(a)pyrene. Phenanthrene is found in the source in high concentrations and is probably representative of the group in terms of its transport properties in groundwater. For these reasons, we have chosen to use it as a typical PAH in our risk analysis.

It is likely that phenanthrene, like most of the PAH chemicals, will adsorb strongly only suspended particulates and biota in the environment. Moreover, when dissolved in water, it can undergo rapid direct photolysis and may subsequently be biodegraded and transformed by benthic organisms (EPA, 1979).

PAHs have been found in groundwater and surface water supplies worldwide. They have also been found in treated drinking waters in varying concentrations, generally well below the World Health Organization recommended limit of 200 ng/l (EPA, 1980c). PAHs are also found at low levels in most food products, primarily entering foods through surface adsorption and biological accumulation. Food additives and packaging materials can also contain these chemicals. These sources contribute to PAH levels in processed foods, as do smoking processes and cooking. Investigators have measured concentrations of phenanthrene as high as 58 ng/g in smoked or barbecued meats (EPA, 1980c). In other foods the concentrations of PAHs are generally found to be much lower. PAHs can also be found in the air. The EPA (1980c) has calculated the present ambient PAH concentration in U.S. urban areas to be 9.3 ng/m^3 .

Because of the lack of specific information about the health effects of phenanthrene, we will use the health effects data for benzo(a)pyrene (BaP) instead. BaP has been widely studied and its health effects are much better known and understood than those of any other PAH. Moreover, it has also been found to be the most carcinogenic of the PAHs that have been studied, and it has been used to derive the water quality criterion for all PAHs (EPA, 1980c). Accordingly, using the health effects for BaP should not be an unreasonable assumption in our analysis and may provide a somewhat pessimistic result.

EMISSION RATES

The composition and extent of the coal tar deposits were determined from exploratory wells. Soil, tar, and groundwater samples were collected from a number of wells in and around the tar pond and lake-bed deposits. These samples were analyzed for 28 inorganic compounds, 32 volatile organic chemicals, and 58 semivolatile organic compounds using a variety of techniques. Most of the organic chemicals studied are listed as priority pollutants under the CWA and as potential toxic chemicals under the RCRA. In the discussion below, we refer to emissions from the tar pond only.

Airborne Emissions

An air monitoring survey was performed at the site to measure levels of organic contaminants during the drilling operations. The monitoring consisted of both instantaneous samples and eight-hour samples, both of which were analyzed for volatile organics. The eight-hour samples were analyzed using gas chromatographic and mass spectrometric methods that were much more accurate than the instantaneous sampling analysis. Most samples did not contain contaminants, and none of the eight-hour analyses found measurable levels of benzene, toluene, or xylene in the area of the wells.¹

These measurements indicate that atmospheric emissions from the coal tar source may not be significant if the source were left in its current state. In the future, however, the area may be disturbed for one reason or another. One scenario would involve uncovering the tar pond and using biological treatment to decompose the organic chemicals. Under these circumstances, atmospheric emissions would increase while the deposits were uncovered.

To assess the potential health risks of this and other alternatives, we attempted to estimate the potential emission rates to the atmosphere for two different assumptions about soil cover—no cover and one meter of soil. Emission rates were calculated for the four volatile chemicals considered in the analysis: benzene, toluene, xylene, and naphthalene. Phenanthrene was not considered in these calculations because it is not sufficiently volatile to produce significant emissions. The details of these calculations are presented in Appendix A.

Appendix A bases its calculations on two measurements of pollutant concentration in the tar pond. The average concentration for each pollutant in the pond is not known and is, in fact, highly uncertain. Although the tar pond occupies an area of roughly 6,100 m², its composition over this area is far from uniform. Sand, gravel, soil, and other materials may be mixed with the tar. Although some measurements have been made, they do not cover the area and give no reliable indication of the average concentrations for the pollutants over the entire volume of the pond. Accordingly, the emission calculations have been performed for two different pollutant concentrations in the soil: (1) the measured concentration in samples from the one drilling site within the tar pond and (2) the maximum concentration found for any soil or tar sample taken at the site. The results for these two sets of data, summarized in Table 3.1, thus represent possible emission rates and a set of upper bounds on the emission rates for the pollutants.

Atmospheric emissions can also include dusts and soil particles picked up by winds and distributed throughout the local area. The surrounding population can be exposed to the pollutants in these soil particles through inhalation, ingestion, and dermal contact with the material. The resuspension of particulate emissions from the ground has been studied by investigators in a number of different areas. This problem is important to researchers con-

¹The detection limits of the analysis procedures were 0.05 and 0.1 ppm for benzene and 0.08 and 1.0 ppm for both toluene and xylene.

Table 3.1
 POLLUTANT FLUX TO THE ATMOSPHERE
 (ng/cm²/s)

Chemical	Measured Concentration		Maximum Concentration	
	Day 10	Day 50	Day 10	Day 50
Benzene	5.3	2.4	5.3	2.4
Toluene	1.6	0.71	5.0	2.3
Xylene	0.31	0.14	3.8	1.7
Naphthalene	0.77	0.34	53.7	23.8

cerned with resuspension of settled substances (including radioactive emissions and fallout), fugitive dust emissions (from storage and disposal areas), and wind erosion of soils.

Unfortunately, resuspension rates cannot generally be predicted for a particular situation, because of the complexity of the problem. Resuspension will depend on many factors, including (1) soil moisture, (2) the distribution of particle sizes, (3) soil contours, (4) erosion history, (5) vegetative cover and character, (6) wind velocities, and (7) soil texture (Gillette and Cowherd, 1982; Sehmel, 1982). For pollutants distributed within the soil, emission rates will also depend, in part, on the physical and chemical properties of the pollutant. These properties include volatility and solubility, in addition to particle size. Contaminants may be resuspended both as discrete particles and attached to soil material.

The tar pond deposits are currently covered with several feet of soil and light vegetation. The concentrations of tar pollutants in the cover soil are not known, but particulate emissions of tar constituents should be below measurable levels. However, if the deposits are subjected to land treatment or other disturbance in the future, this soil will either be removed or the upper layers turned over and mixed with the tar to expose the pollutants for aerobic decomposition. Under these circumstances, some of the deposits will be available for wind erosion.

One would not expect coal tar to be easily eroded by winds, but some contamination of the mixed soil would occur. Moreover, benzene, toluene, and xylene are extremely volatile, with half-lives of about five hours (EPA, 1981). Accordingly, as much as 99.9 percent of these chemicals could disappear from soil particles in two days of open exposure. Because of the uncertainties associated with this situation, we can not rigorously predict probable emission rates for the pollutants in particulate form from the tar pond. Instead, as we describe below, we have chosen to use the approach developed by Hawley (1985), in conjunction with assumptions about the pollutant concentrations on the distributed soil.

Surface Soil Concentrations

Soil and tar samples collected from the source were analyzed for the primary inorganic and organic pollutants. The results for the five pollutants of interest are presented in Table 3.2. Note that these samples did not include the surface soil layers above the tar deposits. For that reason, we have chosen to use the same assumption for the surface soil concentrations that we use for the atmospheric emissions. When necessary, we calculate emission rates related to soil concentrations for a situation where there is no soil cover. Accordingly, the surface soil is assumed to have pollutant concentrations equal to that in the tar deposit itself.

Table 3.2

CHEMICAL CONCENTRATIONS IN SOIL AND TAR DEPOSITS

Item	Sample Location		
	Well 5 Shallow	Well 5 Shallow	Well 5 Deep
Depth (m)	3.5	3.5	11.5
Material	Tar and soil	Tar only	Saturated soil
Concentration ($\mu\text{g/gm}$)			
Benzene	370	N.A.	N.A.
Toluene	116	N.A.	N.A.
Xylene	44	N.A.	N.A.
Naphthalene	0.11	430	0.05
Phenanthrene	0.32	690	0.13

NOTE: N.A.—no analysis performed for this chemical.

Groundwater

As discussed above, it should be unlikely that any wells will be affected by potential groundwater emissions from the disposal pond. Current groundwater quality falls below drinking water standards without treatment, Town A is served by municipal wells from other areas, and the groundwater flow is generally toward the JK River. Contaminant concentrations in the groundwater decrease downgradient because of dilution and adsorption. Moreover, no detectable concentrations of coal tar constituents have been found in wells upgradient of the area. All contaminants move along the flow paths toward the river and should discharge to the river. Accordingly, we do not directly consider groundwater concentrations of pollutants in this analysis.

Surface Water

Groundwater contamination and transport does enter the analysis, however, because the river could be contaminated by pollutants leached from the tar pond. Analysis of river water samples taken opposite the location has found no evidence of coal tar contamination of the surface waters. However, because of delays in transport through the groundwater and soil, the coal tar pollutants might not reach the river in significant levels for a number of years. Accordingly, it was necessary to use groundwater transport analysis for the tar pond deposits to calculate when the pollutants of concern could reach the river and what might be the equilibrium loading rates on the river.

General groundwater movement and pollutant transport near the source have been investigated in separate studies. Information about groundwater flow fields and the location and characteristics of the coal tar deposits was used to calculate pollutant concentrations and movement in the contaminant plume from the tar pond. These calculations, for the five organic chemicals considered in the risk analysis, included dilution and dispersion, as well as adsorption onto soil along the flow path. The results of the analysis, presented in Table 3.3, include both the expected steady-state transfer rates to the JK River and the number of years required to reach 95 percent breakthrough, or 95 percent of the ultimate steady-state loading rate. These concentrations and travel times are conservatively estimated assuming no chemical transformations during groundwater transport.

Table 3.3

POLLUTANT TRANSFER TO THE JK RIVER

Chemical	Transfer Rate (mg/sec)	Breakthrough Time (yr)
Benzene	0.1754	70
Toluene	0.1401	150
Xylene	0.1327	700
Naphthalene	0.2990	800
Phenanthrene	0.0201	9,500

These loading rates can be used as the source terms for the surface water transport analysis of the river. Because of the uncertainty regarding the future status and configuration of the City Lake, we assume that the loading rates with the lake in place would remain the same.²

DESCRIPTION OF ANALYSIS

In general, the source may emit pollutants to the air and groundwater. Atmospheric emissions consist of volatilization and wind-carried dusts from the tar pond. Groundwater emissions include leaching from the tar pond and lake bed source. This study investigates the risk associated with surface water, air, and soil pollution resulting from these emissions. The dominant source of surface water pollution is interaction with the groundwater contaminated with leachate from the primary tar pond.

The analysis was divided into three distinct parts, corresponding to the different types of exposure, for two primary reasons. First, the population affected by the different exposure pathways will not necessarily be the same. Although inhalation and dermal contact pathways affect only the people living in the immediate vicinity, a much larger group of people may someday consume fish or oysters raised in the affected waterways or may use the JK River as a drinking water source. Second, in some cases, different exposure pathways may lead to different types of health effects. Although we have generally combined exposure rates for inhalation and ingestion, this is not always an acceptable approach. Moreover, dermal contact exposure frequently leads to more local adverse effects than the other intake modes. In this study, we use an alternative approach to calculate the potential health risk of dermal contact with phenanthrene.

Dividing the analysis in this manner also makes it possible to perform a sensitivity analysis on each part of the problem separately. In general, this simplifies the sensitivity analysis and makes it easier to understand how specific parameter variations affect the final results.

It is also a logical method of addressing the problem, because various actions can affect potential exposure through one pathway more than others. As an example, if dermal contact

²It is unlikely that loading rates would increase significantly. First, the lake would not be reconstructed until the lake bed tar deposits were removed. Second, the tar pond rests on well-defined sand, silt, and clay deposits. There is no evidence of fractures or other features that could cause the loading rates to change dramatically with the lake in place.

were particularly important, relative to the inhalation and surface water pathways, greater care could be taken to preserve the deposits and prevent either accidental or deliberate disturbance and subsequent contact. If future surface water pollution proved to be significant, the deposits could be treated or other measures taken to minimize this hazard. Thus it is important to know the potential for exposure and health risk through each pathway independently.

Accordingly, we will consider each of the three types of exposure pathways separately in the subsequent description of the analysis.

SUBREGION DESCRIPTION AND POPULATION DISTRIBUTION

As the first step in calculating pollution concentrations in the environment and population exposure rates, one must describe the region around the source. This process includes dividing the region into subregions and describing the distribution of population in these subregions. It may also include determining land use patterns in the subregions and describing agricultural and industrial uses of affected surface or groundwaters. This determination will be necessary when agricultural production or industrial water use could be potential pathways for exposure to the contaminants of concern in the analysis.

This subsection discusses the definition of subregions and distribution of population. The remaining characteristics of the region will be considered as needed in the description of each part of the analysis below.

The total exposed population must include all people affected by one or more of the exposure pathways considered. In this analysis, the various exposed population groups differed widely with exposure pathway. As a consequence, no universally applicable group of subregions could be defined. Instead, subregions and their corresponding populations were determined for each primary type of exposure. The exposure rates and subsequent health risk for each population group could then be determined by adding the risks for appropriate pathways under the different scenarios.

We determined that the population that could consume drinking water or fish from the river is much larger than the population exposed through other routes. Accordingly, we assumed that the total exposed population consisted of all people in the region who could consume contaminated drinking water. This population also ate aquatic organisms caught in the affected waterways. In the total population, we identified those people who could also be exposed through inhalation of volatile pollutants or ingestion and dermal contact with wind-blown soil and dust from the source.

In all subregions of the area, the population was partitioned into age groups for the exposure analysis. Each age group will differ in terms of how much drinking water and fish it consumes, breathing rate, and activity patterns. In this case, we considered four such groups, specifically:

- Infants—younger than one year.
- Children—one to ten years.
- Teenagers—11 to 16 years.
- Adults—17 and older.

Given the specific exposure pathways and pollutants considered in this analysis both exposure levels and health effects are more likely to vary among age groups than between the sexes. For those pollutants with teratogenic health effects, we can deal with the sex influence by using reasonable assumptions about population size. As a result, the complications involved with separating the population by sex, in addition to age group, were not warranted.

The particular subregion definition depends on the specific exposure pathways considered in the analysis. Accordingly, three types of subregion definitions were considered, corresponding to the distribution of population exposed to the contaminated surface waters, volatile air pollutants, and deposited soil and dust.

Surface Water

For the surface water exposure pathways (consumption of drinking water and aquatic organisms), the population included all persons living downstream of the discharge point within reasonable distance of the JK River. Although the current river water quality is not suitable for drinking, and all drinking water is obtained from other sources, this pathway was included as a worst-case situation.

Using maps and census data, we determined the population distribution in the City B Standard Metropolitan Statistical Area (SMSA). This population, which includes Town A and all towns south of it, was used as the base population for consumption of drinking water and fish. The total, partitioned into age groups, was also divided into two subregions. The first subregion consisted only of Town A, and the second subregion included all of the remaining population in the SMSA. In the analysis, these subregions have different (hypothetical) locations for their drinking water intakes. The people in both subregions were assumed to consume potentially contaminated fish at equal rates, as will be described in more detail below. Table 3.4 shows the population distribution by group for the surface water exposure analysis.

Air and Soil

For inhalation of volatile air pollutants from the source, we considered the exposed population to consist of all persons living within a 5 km radius. This radius was reduced to 1 km to define the population potentially exposed to airborne soil particles and dust.

For these exposure pathways, particularly the inhalation of volatile organics, it was necessary to partition the local area into geographic regions. This partitioning process, using a combination of angular sectors and radial segments, is commonly used in safety and environmental impact analyses performed for nuclear power plants, and has been generally incorporated into the WTRISK program. In this situation, because the atmospheric emissions are coming from a low-concentration surface source, a radius of 5 km around the coal tar pond was considered sufficient. Therefore radii every 22.5 degrees were extended 5 km from the coal tar pond to

Table 3.4

POPULATION DISTRIBUTION FOR SURFACE WATER EXPOSURE

Subregion	Infant	Child	Teenager	Adult	Total
Town A	433	5,018	3,847	27,977	37,275
City B	4,619	47,194	37,201	291,305	380,319
Total	5,052	52,212	41,048	319,282	417,594

define a total of eight subregions, as shown in Figure 3.3. For chronic soil and dust exposure, we assumed a smaller radius of 1 km. The eight subregions defined above with a 1 km radius were used to determine the population exposed in the soil and dust analysis.

For the subregions defined and identified in Figure 3.3, the population was approximated using 1980 census data. Census tract information for the 10 tracts in Town A was apportioned among the eight 5 km subregions. Only two census tracts and a portion of a third contained population within the 1 km radius. The age distribution for each subregion was calculated using the distribution for the town. Table 3.5 presents the subregion population and age group distribution for both atmospheric and soil and dust transport. Because some of the census tracts extend outside the 5 km range from the source, the population for inhalation exposure is somewhat less than the total population for Town A. Total exposed populations for the three types of exposure are summarized in Table 3.6.

Fig. 3.3—Subregions for atmospheric transport

Table 3.5

POPULATION DISTRIBUTION FOR ATMOSPHERIC AND SOIL EXPOSURE

Subregion Location	1 NNE	2 NE	3 SE	4 SSE	5 SSW	6 SW	7 NW	8 NNW
Atmospheric (5 km radius)								
Infant	34	49	77	78	55	35	70	59
Child	383	547	858	862	608	392	779	651
Teen	273	390	612	615	434	280	555	464
Adult	2,069	2,952	4,633	4,656	3,285	2,118	4,207	3,517
Total	2,759	3,938	6,180	6,211	4,382	2,825	5,611	4,691
Soil and Dust (1 km radius)								
Infant	3	21	21	18	11	0	0	0
Child	39	229	229	200	124	0	0	0
Teen	28	164	164	143	89	0	0	0
Adult	209	1,239	1,239	1,081	672	0	0	0
Total	279	1,653	1,653	1,442	896	0	0	0

Table 3.6

SUMMARY OF EXPOSED POPULATION

Type of Exposure	Total Population
Surface water	417,592
Atmospheric (5 km radius)	36,596
Soil and dust (1 km radius)	5,923

Land and Surface Water Use

Land use in the area and downstream is primarily residential and industrial. Because of this and other reasons, no surface water irrigation occurs downstream of Town A. Accordingly, exposure through consumption of animal or vegetable products need not be considered in this case. Similarly, industrial withdrawals from the JK River are generally nonconsumptive and do not constitute a potential exposure pathway for the regional population.

ENVIRONMENTAL TRANSPORT**Atmospheric Transport**

The simplified atmospheric transport model in WTRISK is designed to be used for stack emissions in fairly open areas. Accordingly, the transport of volatile air pollutants from the source was investigated using another model that is more suitable for ground-level area sources. This model, the CDMQC, is one of the group of models provided by the EPA for atmospheric transport analysis (Busse and Zimmerman, 1973; Brubaker et al., 1977).

The CDMQC determines long-term (seasonal or annual) quasi-stable pollutant concentrations at ground-level receptors using average emission rates from both point and area sources.

The model uses a joint frequency distribution of wind direction, wind speed, and stability class for the period of interest. This information, commonly called STAR data, can be obtained for most areas from the National Oceanic and Atmospheric Administration. Other information required by the model, including mixing heights and temperatures, was determined from the literature such as USNWS (1970) and Holzworth (1972).

As described above, atmospheric transport calculations were made for the four volatile pollutants—benzene, toluene, xylene, and naphthalene. Phenanthrene is not sufficiently volatile to produce significant concentrations in the air. Background concentrations of these pollutants in the air have not been determined for the region. Some limited information about the ranges of background concentrations nationwide can be found in the EPA water quality criteria documents (EPA, 1980a, 1980b, 1980c, and 1980d). Unfortunately, these concentrations are generally below the minimum detection levels of the measurements made by the utility during the monitoring and drilling program. Accordingly, we chose not to use background concentrations but rather to calculate only incremental pollutant concentrations in the air for the four pollutants.

Ignoring background concentrations in the analysis does not affect the results. We are concerned with the net health risk associated with emissions from the source, and all health effect calculations are based on linear equations. As a result, the health risks for all five chemicals are independent of background pollutant concentrations (or exposure rates) and depend only on the incremental exposure associated with source emissions.

Using both the nominal and maximum concentration emission rates described above, we calculated the pollutant concentrations in the area of the source and at representative locations in each of the angular subregions. We divided each subregion into two equal areas and selected the approximate centroid of each area as a receptor. These results, as incremental pollutant concentrations, are presented in Table 3.7. The table also includes, for comparison, typical background concentrations and either proposed or existing occupational exposure limits (EPA, 1980a, 1980b, 1980c, and 1980d; Jori et al., 1986). Although the pollutant concentrations appear much higher at the site and for the maximum emission cases, they fall either within or below the normal range of background concentrations and orders of magnitude below the regulatory limits. Only naphthalene has a possible air concentration above the normally measured background rates. The analysis was performed using integration grid sizes of 1 km and 200 m. The peak concentration above the source increases as the grid size is reduced, changing by a factor of 13 between the two calculations. A smaller grid size would probably produce a still higher and narrower peak concentration directly over the tar pond. For risk assessment, however, the peak concentration may not be the best measure. Over extended periods the average concentration in the area of exposure is more appropriate. Accordingly, we have based our exposure calculations for site workers on the 200 m grid results.

Surface Water Transport

The groundwater flow and transport analysis was performed by a consultant to the electric utility. This consultant calculated that contaminants leaching into the groundwater will ultimately reach the river in measurable concentrations. Even if the City Lake has been reestablished, these contaminants will be distributed throughout the lake, subsequently carried downstream, and ultimately discharged into City B Harbor and the Atlantic Ocean.

As discussed above, the current and near-term future water quality of the river below Town A does not permit it to be used for drinking, fishing, or swimming. Similarly, there is no

Table 3.7
CALCULATED VOLATILE POLLUTANT CONCENTRATIONS IN AIR
($\mu\text{g}/\text{m}^3$)

Subregion	Benzene		Toluene		Xylene		Naphthalene	
	Nominal	Maximum	Nominal	Maximum	Nominal	Maximum	Nominal	Maximum
SC1I	0.081	0.081	0.024	0.076	0.0048	0.059	0.0116	0.808
SC1O	0.056	0.056	0.016	0.052	0.0033	0.040	0.0079	0.554
SC2I	0.019	0.019	0.006	0.017	0.0011	0.014	0.0027	0.185
SC2O	0.013	0.013	0.004	0.012	0.0008	0.009	0.0018	0.127
SC3I	0.025	0.025	0.007	0.024	0.0015	0.018	0.0036	0.251
SC3O	0.018	0.018	0.005	0.017	0.0011	0.013	0.0025	0.176
SC4I	0.043	0.043	0.013	0.040	0.0025	0.031	0.0061	0.423
SC4O	0.030	0.030	0.009	0.028	0.0018	0.022	0.0043	0.297
SC5I	0.074	0.074	0.022	0.069	0.0044	0.054	0.0105	0.736
SC5O	0.000	0.000	0.000	0.000	0.0000	0.000	0.0000	0.000
SC6I	0.011	0.011	0.003	0.011	0.0007	0.008	0.0016	0.112
SC6O	0.000	0.000	0.000	0.000	0.0000	0.000	0.0000	0.000
SC7I	0.015	0.015	0.004	0.014	0.0009	0.011	0.0021	0.150
SC7O	0.010	0.010	0.003	0.010	0.0006	0.008	0.0015	0.104
SC8I	0.085	0.085	0.025	0.080	0.0050	0.062	0.0121	0.845
SC8O	0.060	0.060	0.018	0.056	0.0035	0.043	0.0085	0.593
Area mean	0.034	0.034	0.010	0.032	0.0020	0.025	0.0048	0.335
Site	6.80	6.80	2.01	6.38	0.404	4.94	0.969	67.67
Background	0.05-47.9		113.0		3.0-380.0		0.00006-0.00035	
Limit	31,940		376,664		434,200		52,413	

NOTE: Subregions are numbered as shown in Fig. 3.3 with I indicating the inner half and O the outer half.

potential exposure through consumption of agricultural products or goods contaminated in industrial processes. However, there is a potential for population exposure through consumption of fish caught in the harbor and oysters raised in the harbor and JK River estuary area. Moreover, as a worst case, we consider the possible exposure rates and health risks associated with future use of the river as a drinking water source.

For these reasons, we conducted a surface water transport analysis for the river from Town A to the City B Harbor. This analysis, using the simplified surface water transport model in WTRISK, was performed for a base-case situation and a number of alternative scenarios or sensitivity cases. The base case was defined to be a situation having nominal emission rates, the long-term mean flow rate for the river, no City Lake, and normal or expected values for other parameters in the analysis.³ The alternative cases studied in the sensitivity analysis were designed to investigate the effects of varying certain assumptions and parameter values that were highly uncertain.

In all cases, the model calculated concentrations for the five organic pollutants at a number of locations downstream from the area. The calculated concentrations were then used to calculate corresponding exposure rates for the affected population, those people who consume drinking water or fish and oysters from the river or harbor.

³The base case is considered to be the starting point for the analysis. It generally corresponds to an existing situation and includes the best estimates for parameter values and inputs.

We defined four reaches for the JK River downstream from the emissions. These reaches were determined by our division of the exposed population, the potential exposure pathways, and hydrological conditions. Although we attempted to keep conditions within each reach as homogeneous as possible, this was not a primary requirement. The characteristics of the reaches are shown in Table 3.8. Only the first two reaches are properly defined. These correspond to the section of the river that extends from Town A to the beginning of the estuary. The third reach includes the estuary area and the fourth reach represents the City B Harbor. Because these two areas are not clearly defined stream segments, stream velocities have very little meaning and the lengths are arbitrary. Instead, the most important consideration is the effective dilution that occurs within each. These dilution factors, which were estimated from information provided by a state study of metal and organic accumulation in aquatic organisms and sediments in the lower reaches of the river, can be incorporated into the model by increasing the river flow rates appropriately in the affected reaches. The model then calculates the average pollutant concentrations over each reach, or in this case, for the estuary and immediate harbor areas.

The emission source is located at the beginning of the first reach. We assume that the groundwater from the area will not affect surface water quality upstream and consider the emissions as a point source discharging the five pollutants of interest at a constant rate. The breakthrough times shown in Table 3.3 differ greatly, and xylene, naphthalene, and phenanthrene should not appear at the river in the calculated concentrations for hundreds of years. However, to define a worst-case situation, we have ignored these times and assumed all chemicals to be present simultaneously.

For cases involving the restored City Lake, the first reach defined in Table 3.8 is replaced with a shallow, unstratified and well-mixed lake that receives the groundwater discharges. As discussed above, in these cases we assume that the emission rates remain constant when the lake is in place. The water discharged from the lake becomes the source for the flow in the remaining reaches.

Potential key parameters in the surface water transport calculations are (1) pollutant background level, (2) pollutant removal rate, and (3) receiving water flow rate. Because we are concerned with chronic health risks arising from continued exposure, long-term mean flow rates are most appropriate for the analysis. However, we did incorporate a future restoration of the City Lake to determine how that might affect pollutant concentrations and exposure rates. Because of considerable uncertainty about both the background levels and removal rates for some of the pollutants, in the sensitivity analysis we investigated a range of values for these parameters and calculated the corresponding pollutant concentrations in the river. Exposure

Table 3.8
DESCRIPTION OF SURFACE WATER REACHES

Reach	Downstream Distance (km)	Length (km)	Velocity (m/s)	Dilution Factor
1	0.0	1.0	0.18	—
2	1.0	13.9	0.18	—
3	14.9	1.0	—	0.45
4	—	1.0	—	0.21

rates for these cases were then calculated using the nominal values of the exposure analysis parameters. As a consequence, the results should indicate how variations in the transport calculations affect the nominal health risks.

The behavior of the five pollutants in natural waters is not well understood. Benzene, toluene, and xylene can be removed through volatilization quickly in well-mixed, rapidly flowing streams (EPA, 1980a and 1980d; Jori et al., 1986). However, their removal from lakes or slowly flowing rivers is uncertain. Similarly, very little is known about the chemistry of naphthalene or phenanthrene in water. Consequently, there are no accepted values for the surface water decay or removal coefficients for any of these pollutants. Accordingly, we used an intermediate value for the removal coefficient for each pollutant, to reflect removal through volatilization, sedimentation processes, and conversion to relatively nontoxic states. This parameter was reduced to virtually zero in the sensitivity analysis, corresponding to the worst-case assumption of complete conservation.

The EPA (1980a and 1980d) also describes a range of background concentrations for both benzene and toluene in natural waters. Accordingly, we chose the upper and lower ends of each range as the two values used in different cases of the calculations. The values of the removal coefficients, background concentrations, and other parameters used in the analysis are given in Table 3.9.

Using the procedure described above and the parameter values and inputs presented in the earlier tables, we calculated pollutant concentrations in the river and estuary, City Lake, and City B Harbor area for a number of alternative cases. These cases are defined in Table 3.10, and the results of the transport calculations are shown in Table 3.11. The table shows the background concentration and the incremental concentration associated with source emissions for each pollutant.

For all five pollutants, the results indicate that the background levels, which are typical values for U.S. waters, dominate the total concentrations. The increment caused by leaching from the source is generally small in comparison, particularly for xylene and naphthalene. This may not be significant, however, because we do not know the background concentrations of these pollutants in the river. The monitoring program conducted by the utility found no measurable amounts of any of the pollutants in the river but had detection limits that were generally above the assumed background levels, except for xylene.

Table 3.9

PARAMETERS FOR SURFACE WATER ANALYSIS

Parameter/Variable	Value
River flow rate (m ³ /s)	8.46
Lake volume (m ³)	1.10 × 10 ⁹
Removal coefficient (s ⁻¹)	
Nominal	8.02 × 10 ⁻⁶
Minimum	8.02 × 10 ⁻¹⁵
Background concentrations (μg/l)	
Benzene	0.10/0.30
Toluene	0.10/1.00
Xylene	4.0
Naphthalene	1.4
Phenanthrene	0.013

Table 3.10

CASES FOR SURFACE WATER TRANSPORT ANALYSIS

Case Number	City Lake	Removal Rate	Background Level
C01	No	Nominal	Low
C02	No	Minimum	Low
C03	Yes	Nominal	Low
C04	Yes	Minimum	Low

Table 3.11

BACKGROUND AND INCREMENTAL CONCENTRATIONS
IN SURFACE WATER ANALYSIS
($\mu\text{g/l}$)

Case	Background	Reach			
		1	2	3	4
Benzene					
C01	0.10	0.0203	0.0148	0.0047	0.0021
C02	0.10	0.0207	0.0207	0.0093	0.0043
C03	0.10	0.0101	0.0076	0.0024	0.0011
C04	0.10	0.0207	0.0207	0.0093	0.0043
Toluene					
C01	0.10	0.0162	0.0119	0.0037	0.0017
C02	0.10	0.0166	0.0166	0.0074	0.0034
C03	0.10	0.0081	0.0060	0.0019	0.0008
C04	0.10	0.0166	0.0166	0.0074	0.0034
Xylene					
C01	4.00	0.0153	0.0112	0.0035	0.0016
C02	4.00	0.0157	0.0157	0.0070	0.0032
C03	4.00	0.0076	0.0057	0.0018	0.0008
C04	4.00	0.0157	0.0157	0.0070	0.0032
Naphthalene					
C01	1.40	0.0346	0.0253	0.0079	0.0035
C02	1.40	0.0353	0.0353	0.0158	0.0073
C03	1.40	0.0172	0.0129	0.0040	0.0018
C04	1.40	0.0353	0.0353	0.0158	0.0073
Phenanthrene					
C01	0.013	0.0023	0.0017	0.0005	0.0002
C02	0.013	0.0024	0.0024	0.0011	0.0005
C03	0.013	0.0012	0.0009	0.0003	0.0001
C04	0.013	0.0024	0.0024	0.0011	0.0005

NOTE: Increasing the background concentration of any pollutant increases the total concentration shown for all reaches by the same amount.

We can also compare these calculated concentrations (and the increments associated with source emissions) with the water quality criteria established for these pollutants by the EPA. Again, we are comparing the incremental concentrations because we do not know the background levels in the river. For benzene, the incremental values are between 10 and 20 ng/l, even under assumptions of no removal from the river. The EPA criterion for benzene (at a risk level of 10^{-7}) is 66 ng/l, at least a factor of three higher. Note that this criterion is based on consumption of drinking water and fish. If we consider only consumption of fish, the corresponding criterion concentration is 4,000 ng/l or 4 μ g/l.

For toluene, the EPA criterion is less well defined, because of insufficient health data. The criterion concentration, not associated with a particular risk level, is 14.3 mg/l (EPA, 1980a). This is more than a factor of 10,000 higher than our calculated concentrations.

Similarly, because of insufficient data there is no current water quality criterion for phenanthrene. In Table 3.11, we see that source emissions could contribute as much as 2.4 ng/l of phenanthrene to the concentration existing in the river. Currently, it is not possible to say if this constitutes a hazardous level should the river ever be used as a drinking water source. However, this incremental concentration falls below the EPA water quality criterion for benzo(a)pyrene (our proxy for phenanthrene health effects) established for the consumption of fish and shellfish. This situation must be investigated more thoroughly if the river should ever be used for drinking water. At that time, more information about the health effects of phenanthrene and the other PAHs will be necessary before conclusions can be drawn.

Unfortunately, the EPA has not established water quality criteria for either xylene or naphthalene. Xylene is not included in the list of toxic chemicals specified under the Clean Water Act, and naphthalene has not been studied adequately to determine quantitative adverse health effects (EPA, 1980b).

Note that the effect of the City Lake depends on the assumed removal coefficient. By simple mass balance arguments, the amount of pollutant entering the lake water must equal the amount removed (through sedimentation, decay, volatilization, and discharge downstream) plus the change in concentration within the lake. If we assume a conservative pollutant (no decay, sedimentation, or volatilization) and no net change in lake volume (inflow equals outflow), it makes no difference to downstream concentrations whether or not the lake is in place. On the other hand, with the nominal decay coefficient, the presence of the lake generally slows travel of the pollutant, allowing more removal, and consequently results in lower downstream concentrations.

Soil and Dust Transport

Contaminated soil can reach the nearby population in two different ways. It may be picked up by winds and carried throughout the local area, appearing as both outdoor and indoor air particulates, dust, and soil. It may also be picked up by adults working near the source or children playing in the area. As a result, exposure to contaminated soil may occur through several pathways. The specific pathways considered in this analysis are ingestion, dermal absorption, and inhalation of soil and dust. Using the soil exposure procedures described by Hawley (1985), we calculated exposure rates and health risk for lifetime exposure to contaminated soil from the coal tar pond for these pathways.

Unfortunately, as described above, it would be extremely difficult to calculate the suspension and transport of contaminated soil from the area. As a consequence, the model used in this investigation assumes that only soil contaminant concentration measurements are

available. Once these concentrations are known, partitioning factors can be applied to determine the pollutant concentrations in the other media—dust and air. These partitioning factors and other parameters have been chosen to reflect conservative case assumptions and therefore, to some extent, should more likely reflect maximum exposure levels. We discuss the specific procedures and exposure calculations below.

EXPOSURE CALCULATIONS

General Information

Humans could be exposed to the chemicals in coal tar wastes through a large number of potential pathways.⁴ Volatile aromatic compounds such as benzene may be present in the air around a disposal location. Standards for air concentrations of these chemicals in the workplace have been developed by the Occupational Safety and Health Administration (OSHA). Chemicals that reach the groundwater or surface water may subsequently be consumed in drinking water supplies or accumulate in fish and shellfish. If contaminated water supplies are used for irrigation or animal raising, these pollutants may also reach the public through consumption of animal and vegetable products. Finally, the population may be exposed either through dermal contact or ingestion of dust or soil from the source.

In the discussion of the regional description and population specification, we explained that the exposure calculations for this study included inhalation of volatile pollutants, ingestion and dermal contact with dusts and soil, and ingestion of drinking water, fish, and oysters from the JK River and City B Harbor area. The populations exposed through these pathways have been described above. In this subsection we discuss the additional information required by the exposure analysis.

Surface Water Pathways

Although the JK River is not suitable for consumption, because of its generally poor water quality, we have included this pathway as a potential worst-case situation. We assume that all people living in the City B SMSA may consume drinking water from the river. The amount of pollutant consumed by an individual in this manner will depend on the subregion in which the individual lives. Each subregion collects its drinking water from a different section of the river and thus uses water which may have different pollutant concentrations.

For the consumption of fish, however, the situation is different. Here we assume that the total catch is distributed uniformly throughout the entire exposed population. Thus each member of a population age group will consume the same amount of contaminated fish, although the amount consumed by the different population age groups will vary. If the supply is less than normal consumption, the difference is made up from sources outside the area. As a consequence of these assumptions, the exposure rate for ingestion of fish will be constant across subregions for each population group.

This procedure means that the entire exposed population will consume fish having the same pollutant concentrations, although these will vary by species. As a consequence, the exposure rates for surface water pathways will vary across subregions only because of the variation in drinking water characteristics. These assumptions should not dramatically affect

⁴Again we are concerned in this study with exposure only to the population surrounding a waste site. Possible occupational exposure to workers falls outside the boundaries of our analysis.

exposure rates or the calculated health risks. For that part of the population consuming drinking water but not actually eating fish or oysters, total exposure will be overestimated. On the other hand, there will be a similar underestimation of exposure rates for the population whose consumption rates of fish and oysters are higher than the mean values. Because all of the health effects calculations are linear, however, the net risk will be the same. Moreover, the analysis should be conservative for virtually all of the population, because of the assumption that all locally produced fish and oysters are consumed locally.

Note also that these calculations assume that all pollutants emitted from the source reach their maximum concentrations simultaneously and maintain these emission rates at constant levels. As Table 3.3 shows, most of the pollutants, particularly the PAHs such as phenanthrene, will not reach significant emission levels for hundreds or thousands of years. Under these circumstances, one must understand that uncertainties in the situation and conditions that are not currently predicted or recognized may invalidate the conclusions of the analysis. Accordingly, these results should be considered as indicators of the potential for health risk to the regional population under current conditions and the assumptions made in the study.

Consumption of Drinking Water. For consumption of drinking water, we have divided the population into two subregions. Town A is assumed to take its entire drinking water supply from the JK River at the end of Reach 1 (or the dam of the City Lake when it is present). The remaining population obtains its drinking water at the end of Reach 2, the beginning of the estuary area.

The pollutant concentrations in the river would normally be reduced by drinking water treatment processes used in the various communities. However, for the pollutants considered in this study, very little is known about the removal efficiency of such treatment processes. Consequently, we have made a worst-case assumption that no removal occurs and that the concentration found in the river is consumed by the population.

Drinking water consumption will vary by age group. The exposure calculations assume the following consumption rates: (1) infants—0.55 l/day, (2) children—0.71 l/day, (3) teenagers—0.71 l/day, and (4) adults—1.01 l/day. These consumption rates, which are used by the Nuclear Regulatory Commission in evaluating releases from nuclear power plants (NRC, 1977), are somewhat lower than the standard 2.0 l/day assumed by the EPA. However, they are probably more realistic in that recent studies have shown that liquid consumption is dominated by soft drinks, milk, and alcoholic beverages rather than tap water.

Consumption of Aquatic Organisms. The aquatic organisms investigated in this study are the primary game fish caught in the City B Harbor area and the seed oysters grown in the harbor and JK River estuary. Data on fishing activities from shore and boats in the harbor and nearby shore areas have been obtained from creel surveys conducted by the EPD. These data are summarized in Table 3.12. The table represents fishing activity from early spring through late fall. Because of weather constraints, we have assumed that fishing during the remainder of the year would be insignificant in comparison.

Bioconcentration factors for the five pollutants have not been studied. However, the EPA (1980b, 1980c, 1980d, 1980e) has calculated expected bioconcentration factors for benzene, toluene, naphthalene, and phenanthrene, based on work with similar chemicals. For xylene, Fishbein (1985) reported values of the bioconcentration factor ranging from 6 to 20. Because there is no information about specific bioconcentration factors, all species were combined, and total fish production was used in the analysis.

Table 3.12
FISHING INFORMATION FOR THE CITY B AREA

Species	Number Caught	Mean Weight (kg)	Total Catch (kg)
Blackfish	4,488	1.30	5,822
Bluefish	62,336	1.60	99,529
Porgy	109,491	0.40	43,705
Snapper bluefish	35,681	0.10	3,561
Flounder	22,401	0.41	9,132
Total	234,397		161,749

The assessment procedure assumes that all locally caught fish are consumed first by the fishermen (licensed and unlicensed) and their families. If fishermen give away or sell part of their catch, the consuming population will also include other families. Unfortunately, we could not find the data necessary to determine the population in this way. Accordingly, in the analysis we assumed that the exposed population consisted of the total City B SMSA population. If the production of fish is insufficient to meet the consumption requirements, additional fish from outside the region are used to supplement the local catch. These fish have background concentrations of the pollutants determined from the literature (EPA, 1980b, 1980c, 1980d, 1980e; Jori et al., 1986). Unfortunately, as with the bioconcentration factors, this type of information is extremely limited, at best, and default values are usually required.

The estuary and harbor are used for oyster seeding operations. Because of poor water quality, these oysters cannot be eaten without first being moved to another area for at least one month of depuration. However, the depuration process may not remove organic chemicals effectively. Studies reported by Nunes and Benville (1979) found removal efficiencies for toluene and xylene that ranged from 67 percent to 91 percent over 14 days. There are few data for other pollutants, and we do not feel that these results should be generalized. Accordingly, we have assumed that no depuration of the five organic chemicals will occur.

Production rates for oysters were calculated using information from the literature and local data. We assumed 80 acres of the harbor were used with 48,000 bushels of seed oysters per year. If a seed oyster has a volume of 29 cm^3 , this represents 58.4 million oysters per year. If the adult weight of an oyster is assumed to be 42.5 gm, total production of oysters would be 2.48 million kg per year. Bioconcentration factors for oysters were assumed to be the same as for the fish, based on the data found in Nunes and Benville (1979). The bioconcentration factors and background concentrations for the five pollutants are shown in Table 3.13. In the exposure calculations, we assumed that all fish were produced in the harbor area (Reach 4) and oysters in the estuary area (Reach 3).

Note that the values used for the background pollutant concentrations in fish from outside the region are not critical parameters. Because the health effects calculations use linear relationships and we are only concerned with the net risk associated with the emissions, the risk from background pollutant concentrations can be subtracted out.

Consumption rates for fish were estimated from national averages by population group

Table 3.13

CHARACTERISTICS OF AQUATIC ORGANISMS

Item	Species	
	Fish	Oysters
Production rate (kg/day)	443.0	6790.0
Bioconcentration factors		
Benzene	5.2	
Toluene	10.7	
Xylene	10.5	
Naphthalene	20.0	
Phenanthrene	30.0	
Background levels ($\mu\text{g/g}$)		
Benzene	0.01	
Toluene	0.01	
Xylene	0.04	
Naphthalene	0.03	
Phenanthrene	0.005	

(NRC, 1977).⁵ We assumed that each population group would consume locally caught fish if they were available and then would consume fish from outside the area, if needed. The specific species of fish used to supplement local production does not matter. Consumption rates for oysters were calculated from data in NOAA (1986), assuming that the distribution by population group would be the same as for fish. Table 3.14 presents consumption rates by population group for fish and oysters. Exposure rates calculated using these assumptions should be very conservative, particularly for oysters. It is highly unlikely that all fish and oysters raised in the harbor area are consumed by the local population. Oysters, in particular, would likely be distributed over a wide area after harvesting.

Inhalation of Volatile Organics

Inhalation exposure can be calculated by multiplying mean air pollutant concentrations by inhalation rates. These inhalation rates, calculated using data from Hawley (1985), are: (1) infants—6.12 m³/day, (2) children—7.85 m³/day, (3) teenagers—16.63 m³/day, and (4) adults—19.94 m³/day. In addition, to account for the fraction of pollutant immediately

Table 3.14

CONSUMPTION RATES FOR AQUATIC ORGANISMS
(kg/day)

Item	Infant	Child	Teenager	Adult
Fish	0.0	0.006027	0.014247	0.018904
Oysters	0.0	0.000465	0.00110	0.00146

⁵These 1977 consumption rates may underestimate current and future fish consumption somewhat because of the recent trend away from red meat consumption.

exhaled and not absorbed, we assumed an absorption fraction of 50 percent for inhalation. We described above how the exposed population was determined and partitioned into subregions. For each subregion we calculated the expected long-term concentration in the air for benzene, toluene, xylene, and naphthalene, assuming that the current soil cover was removed or disturbed. Using this information, we could then calculate long-term inhalation exposure rates by population group for the area.

Contaminated Soil and Dust Exposure

Exposure to contaminated soil may occur through several pathways, including ingestion, dermal absorption, and inhalation of both soil and dust. Using the soil exposure model described by Hawley (1985), we calculated chronic human health risk for lifetime exposure to contaminated soil from the coal tar pond for these specific pathways: (1) inhalation of dust, (2) dermal contact with indoor dust and outdoor soil, (3) and ingestion of indoor dust and outdoor soil. In performing this analysis, we assumed that the contaminated soil, dust, and air particulates are uniformly distributed radially around the source for 1 km.

Three types of assumptions are required when calculating total contaminated soil intake by residents. The first category includes assumptions about the total population at risk and includes parameters such as (1) body surface area, (2) respiratory minute volumes, (3) air particulate levels, (4) surface dust levels, (5) soil or dust density accumulated on the skin, and (6) indoor or outdoor exposure.

The second category of assumptions concerns exposure times, the time spent engaged in particular activities during which an individual, or population group, will be exposed to contaminated soil or dust in each of the specific pathways. The time spent engaged in particular activities will depend on the season (summer or winter) and whether an individual remains at home. Time away from the home depends somewhat on age. For example, a school age child will not play outdoors as much as a preschool age child.

The third category of assumptions relates to absorption rates. These depend both on the exposure medium and the pollutant of interest. The following paragraphs describe the detailed analysis and assumptions used. We do not attempt to justify the parameter values here. Most of this information can be found in Hawley (1985).

Population at Risk. For this analysis, the population at risk was assumed to be those who lived within 1 km of the source. Because many of the exposure rates depend on body surface area and time spent engaged in particular activities, it was necessary to further divide the population into age groups. The groups are basically the same as those used in the rest of the analysis except that children are divided into preschool and school age groups. Definitions of each category and the numbers of persons in each category who live within 1 km are shown in Table 3.15.

Population Characteristics. To reduce the number of calculations required, the average age for the population in each group was used in the analysis. These averages were used to estimate the appropriate body surface areas, respiratory minute volumes, and total body weights needed in the model. Body surface areas were used to calculate dermal, and, in some cases, oral exposure to contaminated soil. For children playing outdoors exposed areas may include hands, arms, legs, and feet, as opposed to only the hand areas for adults working indoors. Respiratory minute volumes were used to determine exposure to air particulates outdoors, indoors, and in dusty attics. Tables 3.16 and 3.17 summarize these characteristics for this analysis.

Other Assumptions. Other assumptions that affect exposure levels for the oral and dermal pathways and inhalation pathway were also taken from Hawley. These data, shown in

Table 3.15

SPECIFICATION OF POPULATION AT RISK

Age Group	Definition	Number
Infant	Younger than 1 year	74
Child		
Preschool	1-4 years	279
School	5-10 years	543
Teenager	11-16 years	586
Adult	17 years and older	4,439

Table 3.16

BODY SURFACE AREAS
(m²)

Age Group	Total Area	Upper Body			Lower Body			
		Total	Hands	Arms	Total	Thighs	Legs	Feet
Infant	0.49	0.08	0.06	0.02	0.13	0.07	0.04	0.02
Child								
Preschool	0.63	0.10	0.08	0.03	0.18	0.09	0.06	0.03
School	0.96	0.15	0.11	0.04	0.33	0.16	0.11	0.06
Teenager	1.52	0.24	0.18	0.06	0.59	0.30	0.20	0.10
Adult	1.78	0.34	0.25	0.08	0.69	0.35	0.23	0.12

SOURCES: Berkow (1924); Gehan and George (n.d.); Finch and Hayflick (1977).

Table 3.17

PARAMETERS FOR SOIL EXPOSURE ANALYSIS

Age Group	Average Age (yr)	Average Weight (kg)	Respiratory Minute Volumes	
			At Rest (l/min)	Light Activity (l/min)
Infant	<1	10.1	2.3	6.2
Child				
Preschool	2	14.1	2.9	8.0
School	8	25.4	4.5	12.2
Teenager	14	51.1	6.3	16.8
Adult	18	64.4	7.6	20.1

SOURCE: Hawley (1985).

Table 3.18, should be self-explanatory and represent conservative choices based on past studies discussed in detail by Hawley.

Time engaged in a particular activity affects exposure levels. For example, if individuals play outdoor sports frequently, they inhale greater amounts of soil particulates in the air. They will therefore be exposed to more of the contaminant than individuals who enjoy resting

Table 3.18
SOIL EXPOSURE ASSUMPTIONS

Item	Value
Dermal and Oral Pathways	
Fraction of soil in dust	80.0 percent
Weight of soil per unit area—children	0.51 mg/cm ²
Weight of soil per unit area—adults	3.75 mg/cm ²
Weight of dust per unit area	0.056 mg/cm ²
Weight of attic dust per unit area	1.8 mg/cm ²
Inhalation Pathway	
Fraction of soil in outdoor particulates	50.0 percent
Suspended particulate concentration	
Outdoors	70.0 µg/m ²
Indoors	53.0 µg/m ²

SOURCE: Hawley (1985).

indoors. Time spent outdoors means an individual is exposed to soil whereas indoors an individual is exposed to dust.

Town A is in the northeastern United States and experiences a pronounced change of season between winter and summer. We assume that summer lasts effectively from May to October. Infants and preschool children may play outdoors eight hours a day, five days a week, during that period, once illnesses and vacation are taken into consideration. School age children may play outdoors eight hours a day from May to September, when school begins. Winter extends from November to April. It is assumed that while at school or work residents are not exposed to the contaminated soil.

For the inhalation calculations it is necessary to determine when individuals are at rest or are engaged in light activity. During the summer months, while indoors, residents are assumed to be at rest, with the exception of teens and adults who engage in four hours of light activity indoors. During the winter months, children are engaged in eight hours of light activity indoors, whereas school age children, teens, and adults are engaged in four hours of light activity indoors. While outdoors, all groups are engaged in light activity. Table 3.19 presents this group-activity time information.

Absorption Rates. Total intake of a pollutant through each pathway depends on both the level of exposure and the absorption rate. Very little information is available about specific absorption rates for the pollutants considered in this study. Accordingly, we used as default values the absorption rates assumed by Hawley. The absorption fraction for ingestion exposure is 1.0 and the inhalation absorption fraction is 0.75 for all age groups. Dermal exposure absorption rates differ by group and exposure medium.⁶ Children are assumed to have twice the dermal absorption rate of adults. Their skin is more permeable, and soil and dust are assumed to remain on a child longer than they would on an adult. The medium affects the absorption rate because (1) the contaminated soil is not necessarily in direct contact with the skin, and (2) the contaminants and the medium, soil or dust, may chemically react. The dermal absorption fractions are summarized in Table 3.20.

⁶This procedure is not correct for those pollutants that have different health effects when absorbed through the skin than when ingested or inhaled. Benzo(a)pyrene, for example, when absorbed through the skin, is not metabolized and subsequently causes local skin cancer rather than the effect considered in our analysis. Accordingly, the health effects of exposure to phenanthrene in this study should be treated in this manner. We have not done this here because of time and resource restrictions.

Table 3.19
ACTIVITY DURATION PERIODS

Age Group	Outdoors		Indoors	
	Fraction of Week	Hours of the Day	Fraction of Week	Hours of the Day
Summer months				
Infant	5/7	8	1	16
Child				
Preschool	5/7	8	1	16
School	1	5 ^a	1	16
Teenager	1	5 ^a	1	16
Adult	2/7	8	1	16
Winter months				
Infant	0	0	1	24
Child				
Preschool	0	0	1	24
School	0	0	1	16
Teenager	0	0	1	16
Adult	0	0	1	16

SOURCE: Hawley (1985).

^aFor school aged residents, five hours of activity outdoors represents the average time spent outdoors while at school and while on summer recess. The missing three hours are spent at school.

Table 3.20
DERMAL ABSORPTION FRACTIONS

Age Group	Soil	Dust
Infant	0.018	0.018
Child		
Preschool	0.018	0.018
School	0.018	0.006
Teenager	0.009	0.003
Adult	0.006	0.009
Adult—attic		0.003

Definition of Cases. There is no reasonable way to measure or calculate the distribution of soil and dust from the source for the conditions assumed in the analysis. The source is currently covered with 1 m of soil and the physical characteristics of the coal tar and soil in the pond are not known. Given no further activity at the tar pond, one would not expect any significant uptake or transport of contaminated soil. However, there is some possibility that the tar deposits will be uncovered and treated on site. Under these conditions some dust and soil may escape from the source.

Even under these circumstances, it is still not possible to calculate the probable distribution of dust and soil in the region. The theory of dust suspension by winds has not been well developed, and the data needed to make the calculations are not available. Consequently, the analysis must be based on assumed soil and dust emissions from the source and distribution throughout the nearby neighborhood.

Accordingly, we defined three cases for the analysis. These cases differ with respect to either (1) the population at risk, (2) the media contaminated, (3) the exposure pathways, or (4) pollutant concentration levels. The cases should be conservative and produce maximum expected exposure levels. The case descriptions are summarized in Table 3.21.

Case I assumes that (1) all soil within 1 km of the source has pollutant concentrations equal to a small fraction of the soil concentration in the tar pond, and (2) people are exposed through all pathways. Case II assumes that contaminated soil is not transported in large amounts, as in Case I, but is present only in air particulates and household dust. Case III assumes that exposure occurs only if one is at the tar pond. This case, which is probably the most realistic, implies that only those adults working at the site or children playing there would be exposed through dermal contact, inhalation, and ingestion of dust and soil particles.

We can define relative pollutant concentrations in the soil from the geometry of the situation and some basic conservative assumptions. The area of the tar pond is taken to be 6,100 m². We assume that the soil cover has been removed and that the top 5 cm of tar is picked up by the winds and uniformly distributed over the surrounding circular area (1 km radius). Soil from other areas will also be picked up and mixed with that from the tar pond. Because of this and other processes, we assume finally that the dispersed tar pond soil is uniformly mixed with the top 1 cm of soil in the area. Under these conditions, the average pollutant concentration in the top 1 cm of soil in the exposed area will be 0.0097 times the concentration in the tar pond, assuming that none of the transported pollutant is lost through degradation or volatilization.

Benzene, toluene, and the xylenes are quite volatile and have evaporative half-lives of between 30 minutes and 5 hours, depending on environmental conditions (EPA, 1980a, 1980d, and 1981; Jori et al., 1986). These measurements were not made for evaporation from contaminated soil, however, and it is likely that the corresponding half-lives would be longer. If we assume a half-life of ten hours, volatilization will reduce pollutant concentrations by a factor of 1,000 in approximately four days (99.7 hours). Similarly, for a half-life of 24 hours, this reduction would be achieved in ten days of exposure to the air.

The time available for volatilization in the overall deposition process includes the period the soil remains at the source before being picked up by the wind, transit time, and the time after deposition before the soil comes into contact with a human. Given the short distances and a mean wind velocity of 3.5 m/s (6.75 kn), transit time is likely to be only an hour or two.

Table 3.21

CASE DESCRIPTIONS

Case	Exposed Population	Exposure Pathways	Media Contaminated	Pollutant Concentration
I	1 km radius	Inhalation Ingestion Dermal	Soil, Dust Particulates	A fraction of pond levels
II	1 km radius	Inhalation Ingestion Dermal	Particulates Dust	A fraction of pond levels
III	Children and adults at site	Inhalation Ingestion Dermal	Particulates Dust, Soil	Pond levels

However, the other two components should be much greater, considering that for chronic exposure, we must consider long-term average conditions and processes. Accordingly, we assume for the three extremely volatile pollutants that mean concentrations in the soil are reduced by an additional factor of 1,000 from the dispersion factor estimated above. Based on these rough calculations, this assumption should be quite conservative, considering (1) that this must be a long-term condition, (2) the amount of soil that must be moved through the air, and (3) the probable residence times at the source and in the neighborhood.

The three cases for soil and dust exposure incorporate these results in different ways. In Case I, exposure levels are calculated assuming that soil contamination levels are 10^{-5} times tar pond levels (both nominal and maximum) for the volatile pollutants and 0.01 or 0.001 times the concentrations for nonvolatiles. For reference, the assumed tar pond concentrations are shown in Table 3.22. Given the relatively low wind velocities in the area, Case II assumes that only minor amounts of soil and dust from the tar pond become airborne and that no significant soil contamination occurs in the area. The airborne dust may settle inside houses. Exposure levels are calculated for the ingestion and dermal absorption pathways for household dust only, and for inhalation of both soil and dust. Soil contamination concentrations are again taken to be 10^{-5} times the tar pond levels for volatiles and 0.01 or 0.001 times tar pond concentration for nonvolatiles. In Case III exposure occurs only if an individual is directly at the pond. Such individuals may include small children illegally playing on the tar pond or workers excavating it. The only exposure pathways will be inhalation, dermal absorption, and ingestion of soil. In this case full concentrations are assumed for one calculation and 10^{-5} (for volatiles) and 0.01 (nonvolatiles) for the second alternative.

Exposure Results

Surface Water Pathways. Table 3.23 presents the results of the exposure analysis for surface water pathways. Rates are shown as mean values (weighted averages over the exposed population groups) for each pathway separately and combined. The results, for the low background concentration cases, are presented as background exposure levels and the increments associated with source emissions. If the high background levels were assumed, the background exposure rates would increase, but the incremental exposure rates would remain constant. The results have been presented in this manner because the net health effects depend only on the incremental values.

Actual exposure rates for the region are highly uncertain for all pollutants, and background levels may differ significantly from those shown in the table. Nevertheless, the variations between cases and the increments above background levels should be significant.

Table 3.22

COAL TAR POND CONTAMINATION LEVELS
(mg/kg)

Pollutant	Concentration	
	Nominal	Maximum
Benzene	370	370
Toluene	116	370
Xylene	44	560
Naphthalene	430	30,000
Phenanthrene	690	18,000

Table 3.23
EXPOSURE RATES FOR SURFACE WATER PATHWAYS
($\mu\text{g}/\text{day}$)

Chemical/ Case	Pathway					
	Drinking Water		Fish/Oysters		Combined	
	Background	Site	Background	Site	Background	Site
Benzene						
C01	0.094	0.014	0.157	0.000042	0.250	0.014
C02	0.094	0.019	0.157	0.000085	0.250	0.019
C03	0.094	0.007	0.157	0.000021	0.250	0.007
C04	0.094	0.019	0.157	0.000085	0.250	0.019
Toluene						
C01	0.094	0.011	0.158	0.000069	0.252	0.012
C02	0.094	0.016	0.158	0.000140	0.252	0.016
C03	0.094	0.006	0.158	0.000036	0.252	0.006
C04	0.094	0.016	0.158	0.000140	0.252	0.016
Xylene						
C01	3.75	0.011	0.72	0.000065	4.47	0.011
C02	3.75	0.015	0.72	0.000130	4.47	0.015
C03	3.75	0.006	0.72	0.000033	4.47	0.006
C04	3.75	0.015	0.72	0.000130	4.47	0.015
Naphthalene						
C01	1.31	0.024	0.53	0.00028	1.84	0.025
C02	1.31	0.033	0.53	0.00056	1.84	0.033
C03	1.31	0.012	0.53	0.00014	1.84	0.013
C04	1.31	0.033	0.53	0.00056	1.84	0.033
Phenanthrene						
C01	0.012	0.0017	0.079	0.000028	0.091	0.0017
C02	0.012	0.0022	0.079	0.000056	0.091	0.0023
C03	0.012	0.0008	0.079	0.000014	0.091	0.0009
C04	0.012	0.0022	0.079	0.000056	0.091	0.0023

Because all of these values have been calculated with the same set of inputs and parameters, the differences among exposure rates are far less uncertain than the absolute rates themselves. These differences should reflect real variations between cases and background levels.

Several conclusions can be drawn from these results. First, the incremental exposure rates for all pollutants are small, ranging from about 6 to 34 ng/day. Second, if we consider only fish and oyster consumption, these incremental exposure rates decrease dramatically to less than 1 ng/day. The pollutant doses are dominated by drinking water consumption, which is included only as a worst-case situation in the future. Third, when the City Lake is included (cases C03 and C04), exposure rates decrease by roughly a factor of two, assuming nominal decay rates. For conservative pollutants there is no effect. Fourth, no one pollutant has significantly larger incremental exposure rates than the others, although phenanthrene is about a factor of ten lower than the rest. Finally, making the assumption that the pollutants are conservative (cases C02 and C04) only increases exposure levels by roughly a third over the nominal situation.

These results do not include additional exposure that might occur as a result of recreating the City Lake. We have assumed that this lake would be used primarily for recreational boating and swimming, although the water quality may not be adequate to allow swimming. If the overall water quality does not improve, no swimming or fishing would be permitted. As a result, there would be no additional exposure associated with the presence of the lake.

If upstream discharges are controlled sufficiently to permit swimming and fishing, the incremental exposure from these activities should still be small. The number of people swimming in the lake would be relatively small, and their exposure time would be limited. The primary exposure pathway should be accidental ingestion of lake water. Dermal exposure would be relatively unimportant, because pollutant concentrations in the lake water are much lower than in the dust and soil exposure analysis. Similarly, ingestion of lake water should not be significant compared with the other ingestion pathways, because the analysis assumes that the entire population of the city already uses the lake as its sole source of drinking water. The quality of the untreated lake water will be the same as that of the drinking water, because we have assumed that no pollutant is removed during water treatment.

Inhalation Exposure. Table 3.24 presents the results of the exposure calculations for inhalation of volatile emissions from the tar pond. Because of the similarity in population to

Table 3.24
EXPOSURE RATES FOR VOLATILE INHALATION PATHWAY
($\mu\text{g}/\text{day}$)

Chemical	Infant	Preschool	School	Teenager	Adult
Nominal Emission Rates					
Benzene					
Site	20.82	26.70	40.91	56.58	67.85
Area	0.100	0.128	0.197	0.272	0.326
Toluene					
Site	6.15	7.89	12.08	16.72	20.04
Area	0.030	0.038	0.058	0.080	0.096
Xylene					
Site	1.24	1.58	2.43	3.36	4.02
Area	0.0059	0.0076	0.0117	0.0161	0.0193
Naphthalene					
Site	2.97	3.80	5.83	8.06	9.67
Area	0.0143	0.0183	0.0280	0.0387	0.0465
Maximum Emission Rates					
Benzene					
Site	20.82	26.70	40.91	56.58	67.85
Area	0.100	0.128	0.197	0.272	0.326
Toluene					
Site	19.54	25.05	38.39	53.10	63.67
Area	0.094	0.120	0.185	0.255	0.306
Xylene					
Site	15.12	19.38	29.70	41.08	49.26
Area	0.073	0.093	0.143	0.197	0.237
Naphthalene					
Site	207.06	265.53	406.81	562.72	674.78
Area	1.00	1.28	1.96	2.71	3.24

that in the soil analysis, the results separate the preschool and school age children within their combined age group. The rates shown are divided into exposure at the site and mean exposure for the population within 5 km. The rates are shown as incremental exposure associated with source emissions assuming either nominal or maximum pollutant concentrations. No background exposure rates have been included because no pollutant concentration data in the air are available and only incremental exposure rates are relevant to the health risks.

The incremental exposure rates for the area population are comparable in size to potential exposure from consumption of drinking water and fish, assuming that the nominal emission rates represent the source. If the maximum emission rates are more representative, the corresponding exposure rates, particularly for naphthalene, become larger than the ingestion exposure rates. However, it is important to remember that even the nominal emission rates used in these calculations are conservative upper bounds, given the pollutant concentrations in the soil, and do not represent either measured or expected values of pollutant flux.

Potential exposure to workers and children who visit the tar pond regularly for long periods will be much higher than the exposure to the population of Town A. The presence of soil cover should initially reduce the concentration of volatile pollutants (and thus exposure rates) in the area. The soil should only retard the release of the pollutants, however, and emission rates will eventually reach their equilibrium levels.

Soil and Dust Exposure. The cases of the soil and dust analysis were designed to cover the range of potential exposure pathways and rates. Case I is a worst-case extreme upper bound on exposure rates for the area population. It assumes that large amounts of contaminated soil and dust can be suspended from the source and transported to a 1 km region surrounding it. Given the characteristics of coal tar, the local winds, and the area, this situation has negligible probability. The results for Case I are shown in Table 3.25. It includes exposure through all pathways: ingestion, dermal absorption, and inhalation of both soil and household dust. Pollutant concentrations in the soil surrounding the area are assumed to be 10^{-5} of nominal or maximum source levels for volatiles and either 0.001 or 0.01 for nonvolatiles. Note that these tables do not include background exposure rates. There is virtually no information available for making even rough estimates of these exposure rates, and the nature of our risk calculations make it unnecessary. Case I exposure levels are dominated by oral ingestion. Since young children ingest large amounts of soil and dust they are at greatest risk. Teenagers have minimal risk, since they do not ingest as much soil and dust as young children, are away at school, and do not work in heavily soiled areas (gardens and attics) as much as adults do. For both nominal and maximum concentrations of the three volatile pollutants, these exposure rates are far below those of consuming drinking water and fish. For naphthalene and phenanthrene they are much higher than either surface water or inhalation exposure pathways. Under these conditions, the worst-case assumptions used in the analysis do not result in useful information. The exposure rates are too high to ignore, but they are not truly believable because of the assumptions. In this situation, the only alternatives are either to measure contamination of nearby soils or to perform a much more complex and detailed analysis of the problem.

Case II represents a somewhat more likely situation in which small soil and dust particles from the tar pond are present in the air in the region surrounding the tar pond. These particulates may settle as household dust, but there is no significant contamination of soil in the area. Exposure levels in this case are therefore calculated only for ingestion and dermal absorption pathways for household dust and inhalation of soil and dust. Mean soil contamination concentrations are again taken to be 10^{-5} of the tar pond for volatiles and either 0.01 or 0.001 of tar

Table 3.25
EXPOSURE RATES FOR DUST AND SOIL—CASE I
($\mu\text{g/day}$)

Pollutant	Infant	Preschool	School	Teenager	Adult
Nominal Concentrations					
Benzene	0.00104	0.00126	0.000274	0.000221	0.000644
Toluene	0.00033	0.00040	0.000086	0.000069	0.000202
Xylene	0.00012	0.00015	0.000033	0.000026	0.000077
Naphthalene					
- 0.01	1.21	1.47	0.318	0.256	0.748
- 0.001	0.121	0.147	0.0318	0.0256	0.0748
Phenanthrene					
- 0.01	1.94	2.36	0.511	0.412	1.20
- 0.001	0.194	0.236	0.0511	0.0412	0.120
Maximum Concentrations					
Benzene	0.00104	0.00126	0.000274	0.000221	0.000644
Toluene	0.00104	0.00126	0.000274	0.000221	0.000644
Xylene	0.00152	0.00185	0.000400	0.000322	0.000940
Naphthalene					
- 0.01	84.3	103.0	22.2	17.9	52.2
- 0.001	8.43	10.3	2.22	1.79	5.22
Phenanthrene					
- 0.01	50.6	61.5	13.3	10.7	31.3
- 0.001	5.06	6.15	1.33	1.07	3.13

pond concentrations for nonvolatiles. Case II results are shown in Table 3.26. The exposure rates for Case II are much lower than those for Case I for all pollutants, as would be expected. Again, the younger children are exposed at higher levels to all pollutants, because of their inclination to play outside, wash less often, and put their hands in their mouths. These exposure rates for the volatile pollutants are generally much less than those from surface water ingestion pathways. For naphthalene and phenanthrene they are lower than in Case I, but still relatively high, again reflecting the extremely conservative nature of the analysis.

The results for Case III are shown in Table 3.27. In this special case, we calculate exposure rates only for those individuals who are present at the tar pond regularly for long periods. This may include small children who climb the fence and play on the tar pond and workers who are excavating the area. The only exposure pathways will be inhalation, dermal absorption, and ingestion of soil. In this case both volatile and nonvolatile pollutants are assumed to have their full tar pond concentrations in addition to the fractions assumed in the previous cases. Note that these are concentrations in soil and dust that are airborne and exposed on the surface. For that reason, assuming full volatile pollutant concentrations should be a worst case, because these pollutants would evaporate from the surface soil and airborne dust very quickly, as we have already discussed.

These exposure rates are much higher than the rates calculated in Cases I and II, as they should be. At full concentration levels, the exposure to benzene in the soil is comparable to the levels calculated for inhalation exposure. For both nominal and maximum pollutant concentrations in the coal tar, the potential exposure rates for naphthalene and phenanthrene are quite high (several milligrams per day) at full concentration. In this case, however, only

Table 3.26
EXPOSURE RATES FOR DUST AND SOIL—CASE II
($\mu\text{g}/\text{day}$)

Pollutant	Infant	Preschool	School	Teen	Adult
Nominal Concentrations					
Benzene	0.000224	0.000225	0.0000286	0.0000038	0.000041
Toluene	0.000070	0.000071	0.0000090	0.0000012	0.000013
Xylene	0.000027	0.000027	0.0000034	0.00000045	0.0000049
Naphthalene					
- 0.01	0.261	0.261	0.0332	0.00437	0.0474
- 0.001	0.0261	0.0261	0.00332	0.000437	0.00474
Phenanthrene					
- 0.01	0.418	0.419	0.0533	0.00701	0.0760
- 0.001	0.0418	0.0419	0.00533	0.000701	0.00760
Maximum Concentrations					
Benzene	0.000224	0.000225	0.0000286	0.0000038	0.000041
Toluene	0.000224	0.000225	0.0000286	0.0000038	0.000041
Xylene	0.000327	0.000328	0.0000417	0.0000055	0.000060
Naphthalene					
- 0.01	18.2	18.2	2.32	0.305	3.30
- 0.001	1.82	1.82	0.232	0.0305	0.330
Phenanthrene					
- 0.01	10.9	10.9	1.39	0.183	1.98
- 0.001	1.09	1.09	0.139	0.0183	0.198

persons who work or play near or on the *uncovered* tar pond consistently over long periods would be exposed to these levels. If the area is fenced to prevent children from using it, and workers are protected with masks and gloves, no one would receive exposure at a level anywhere near these calculated values.

HEALTH EFFECTS OF THE POLLUTANTS

In this subsection we use the results of the discussion in Appendix B to calculate the chronic health risks associated with the exposure results presented above. Because the health data and health effects of the five pollutants differ, we must use a different approach for each one. In general, these pollutants have many toxic endpoints that depend on the route of administration, dose rate, and total time of exposure. Only two of them, benzene and phenanthrene, can be classified as carcinogens. The remaining pollutants—toluene, xylene, and naphthalene—must be considered noncarcinogens. As we discussed above, because there are no data relating to the specific health effects of phenanthrene, we have chosen to use the health data for benzo(a)pyrene as a substitute. In this discussion and in Appendix B we refer to benzo(a)pyrene instead of phenanthrene for this reason.

Table 3.27
EXPOSURE RATES FOR DUST AND SOIL AT THE SITE
($\mu\text{g}/\text{day}$)

Pollutant	Infant	Preschool	School	Teen	Adult
Nominal Concentrations					
Benzene					
- 1.00	81.5	104.0	24.6	21.7	60.3
- 0.00001	0.000815	0.00104	0.000246	0.000217	0.000603
Toluene					
- 1.00	25.6	32.6	7.70	6.81	18.9
- 0.00001	0.000256	0.000326	0.0000770	0.0000681	0.000189
Xylene					
- 1.00	9.70	12.4	2.92	2.58	7.17
- 0.00001	0.000097	0.000124	0.0000292	0.0000258	0.000072
Naphthalene					
- 1.00	94.7	121.0	28.5	25.2	70.1
- 0.01	0.947	1.21	0.285	0.252	0.701
Phenanthrene					
- 1.00	152.0	194.0	45.8	40.5	113.0
- 0.01	1.52	1.94	0.458	0.405	1.13
Maximum Concentrations					
Benzene					
- 1.00	81.5	104.0	24.6	21.7	60.3
- 0.00001	0.000815	0.00104	0.000246	0.000217	0.000603
Toluene					
- 1.00	81.5	104.0	24.6	21.7	60.3
- 0.00001	0.000815	0.00104	0.000246	0.000217	0.000603
Xylene					
- 1.00	119.0	152.0	35.8	31.7	88.0
- 0.00001	0.00119	0.00152	0.000358	0.000317	0.000880
Naphthalene					
- 1.00	6610.0	8430.0	1990.0	1760.0	4890.0
- 0.01	66.1	84.3	19.9	17.6	48.9
Phenanthrene					
- 1.00	3970.0	5060.0	1190.0	1060.0	2930.0
- 0.01	39.7	50.6	11.9	10.6	29.3

Approach to Toxicity Calculations

We will discuss each of the pollutants individually and briefly summarize the approaches developed in Appendix B for calculating their adverse chronic health effects. More detailed discussion of health effects and the derivation of these equations can be found in that appendix.

Benzene. Based on numerous epidemiological and animal studies, there is ample evidence that exposure to benzene causes leukemia in humans. From Appendix B, the incidence of leukemia in the exposed population can be found from

$$R_{t,a} = (0.052/70.0) \sum_i (d_i P_i / W_i)$$

where $R_{t,a}$ = number of leukemias in exposed population per year,
 0.052 = potency of benzene ($\text{mg}/\text{kg}/\text{day}$)⁻¹,

d_i = exposure rate for population group i (mg/day),

P_i = number of people in population group i , and

W_i = mean weight for population group i (kg).

The summation over population groups is necessary because the members of different groups have different exposure rates and weights, hence different effective doses.

Toluene. For toluene, we must calculate two separate health effects. The first is the no-effect endpoint, used by the EPA in developing its 1980 water quality criterion for toluene. This calculation, based on the maximum no-effect level observed in subchronic tests conducted by Wolf et al. (1956) has general toxicity as its endpoint. The corresponding equation for this estimate is

$$R_{t,b} = (0.0233/70.0) \sum_i (d_i P_i / W_i)$$

where $R_{t,b}$ = number of deaths in exposed population per year, and

0.0233 = potency of toluene (mg/kg/day)⁻¹.

The second endpoint for toluene is a teratogenic effect—cleft palate. To calculate this risk, we assume that the exposed population is in steady state. Using an average lifespan of 70 years, this implies 0.01429 births per person per year. Accordingly, the incidence of cleft palate in newborns can be found from

$$R_{t,c} = (0.0001562)(0.0149)d_a P_t / 55.0$$

where $R_{t,c}$ = number of cleft palates born to exposed population per year,

0.0001562 = potency of toluene (mg/kg/day)⁻¹,

0.01429 = number of births per person per year,

55.0 = average weight of a pregnant woman (kg),

d_a = exposure rate for adults in the population (mg/day), and

P_t = total exposed population.

There is no summation over population groups, because only the exposure to pregnant women is important. However, the total population must be used in the equation to determine the total expected number of pregnant women being exposed.

Xylene. We consider the adverse health effect of xylene exposure also to be teratogenicity, or general malformation of the fetus. As with toluene, the incremental chronic health risk of exposure is given by

$$R_{t,d} = (0.0002748)(0.0149)d_a P_t / 55.0$$

where $R_{t,d}$ = number of malformed fetuses born in the exposed population per year, and

0.0002748 = potency of xylene (mg/kg/day)⁻¹.

Naphthalene. Toxicity data for naphthalene are scarce. In its 1980 water quality criteria document, the EPA (1980b) stated that there were no chronic animal toxicity studies or epidemiologic studies adequate for use in developing water quality criteria. Although there have been other studies performed more recently, adequate data still do not exist for organ-specific toxicity. As a result, we again use subchronic general toxicity as an endpoint. The equation for this calculation is

$$R_{t,e} = (0.0323/70.0) \sum_i (d_i P_i / W_i)$$

where $R_{t,e}$ = number of deaths in the exposed population per year, and
 0.0323 = potency of naphthalene($\text{mg}/\text{kg}/\text{day}$) $^{-1}$.

Benzo(a)pyrene. Benzo(a)pyrene is an extremely carcinogenic chemical that has been studied in some detail. We use it as a proxy for the health effects of phenanthrene, also a PAH, because the health effects of chronic exposure to phenanthrene have not been examined adequately. The endpoint for benzo(a)pyrene to be considered in this situation, related to ingestion and inhalation, is gastric tumors. Although this would not be the health effect caused by dermal exposure, in this initial analysis we will use it for all exposure pathways. From Appendix B, the equation for calculating the incidence of gastric tumors is

$$R_{t,f} = (11.50/70.0) \sum_i (d_i P_i / W_i)$$

where $R_{t,f}$ = number of gastric tumors in the exposed population per year, and
 11.50 = potency of benzo(a)pyrene ($\text{mg}/\text{kg}/\text{day}$) $^{-1}$.

Note that benzo(a)pyrene has been found to be a cocarcinogen with some heavy metals—nickel and chromium, for example. This could be a potential source of additional risk in this case, although these two particular metals are not found in excessive levels in the groundwater near the source. Understanding of the relationship between the pollutants is limited, however, and the time and resources available for this study have not permitted us to examine it in more detail.

Use of Log-Linear Dose-Response Model

Dose-response estimates were obtained for all substances in this report from a log-linear model or log-linear limit of a multistage model. Other models are available for use, including multihit, probit, and logit models as well as no-observed-effect or lowest-observed-effect levels. For the case of those compounds known to be carcinogenic (benzene and benzo(a)pyrene), general agreement exists that these compounds transform individual cells at the genetic level through a sequence of one or more stochastic events. Hit or stage type models account for these cellular events and are therefore appropriate descriptors of the carcinogenic process. At low dose these models often limit in the mean to the log-linear form employed in this work, and, if confidence limits are taken into account, nearly always limit to such a form (Guess and Crump, 1977). Hence our use of log-linear models with benzene and benzo(a)pyrene is an attempt to capture confidence limit behavior and to force bias in our results toward a conservative overestimation of risk. This is also the favored approach followed by agencies regulating carcinogens and, thus, carcinogenic risk estimates obtained from it are representative of the standards that must be considered by those seeking compliance with present or expected regulations.

Use of hit or stage models for noncarcinogenic endpoints is more controversial. Acute lethal dose data, considered by us for both toluene and naphthalene, are generally not described by hit or stage models but rather by a probit model, with its underlying assumptions that each individual has a threshold for response and that these thresholds are distributed log-normally over a population. Likewise, there is a school of thought that teratogenicity (which we consider for toluene and xylene) is by and large a threshold phenomenon (Brent, 1986; Beckman and Brent, 1984). Gene changes or chromosome abnormalities generated in eggs or sperm (presumably by mutational events described by hit or stage models) can still lead to embryopathology in this school of thought, but since these types of changes occur before the exposure period employed in most teratologic studies (the period of organogenesis), most

teratologic outcomes arise from nonmutational mechanisms (Wilson, 1973). If one considers these mechanisms (such as death of embryonic cells) to become operative at a threshold level of toxic agent, then individual variability might be responsible for distributing these thresholds over an exposed population and, in analogy to whole animal acute lethality occurrence, teratogenic frequency might also be described by a probit model.

However, the limited xylene and toluene data make parameterization of the three parameter (one background and two added-risk) probit model somewhat difficult. Furthermore, the specific choice of a log-normal distribution of teratogenic thresholds as the most exact functional form is not firmly established. Hence, both for ease of computation and to cover any uncertainty related to the choice of threshold distribution, we elected to use the log-linear dose-response model in preliminary analysis of both the acute lethality and teratogenic data. Ease of computation follows from the occurrence of one less parameter in the log-linear model. Conservative risk estimation follows from the observation that probit-derived responses drop off faster than any power of dose in the low dose limit. Thus, if a risk estimated from a log-linear model is at an acceptable level, then so should be the probit model result (or most other model results). Risks computed in this manner for this report should therefore only be considered upper-bound estimates. Should these bounds later be judged in excess of acceptable risk (we have tentatively assumed that our risks are acceptable), then risk estimation will become more model dependent, and recomputation of upper-bound risk from the widely accepted (although not proven) probit description should be performed for comparison.

No-observed-effect levels or lowest-observed-effect levels have not been employed in our analyses at all. They have been excluded because their use introduces the concept of an infinitely sharp threshold of response *for a population*. There is little or no biological evidence for making this assumption and substantial indication from the characteristics of other responses that thresholds are distributed. Hence, threshold distributions (such as the probit), which may be parameterized to any degree of sharpness, are a preferable approach.

In the special case that sample sizes of exposed populations are too small to detect any toxic response, the data are not sufficient to calibrate a probit model. However, it is inappropriate to impose a threshold at the lowest dose level examined when small numbers of exposed subjects have been used (e.g., ten or fewer), since it leads to possibly severe underestimation of toxic response when large populations (e.g., 100,000) are considered. If the true risk of toxic response at a given dose is, say, 1/100, exposure of ten subjects may very likely reveal no toxic response; yet exposure of 100,000 subjects would reveal nearly 1,000 toxic responses. Had a threshold approach been taken on the ten-subjects data, none of these 1,000 responses would have been predicted. Because of severe difficulties such as this, we have chosen to err on the side of overestimating risk with the log-linear model rather than underestimating it with threshold approaches.

Danger to Aquatic Environment

There is very little information about the chronic adverse effects of these pollutants on the aquatic environment. Most of the research performed in the past has been directed toward measuring the effect of acute doses on aquatic organisms. Unfortunately, the results of these studies— LD_{50} information—do not provide useful insight into the problem of how long-term, low-level exposure to the chemicals affect aquatic species. We will try to draw some limited conclusions for the studied pollutants, based on the available information and our calculations of potential surface water concentrations.

Benzene. The EPA (1980d) has stated that acute toxicity to freshwater organisms occurs at benzene concentrations of 5.3 mg/l. Although no chronic toxicity data have been published, one experiment found that some adverse effects occurred to a saltwater fish species at concentrations as low as 0.7 mg/l. These limited tests on a few species clearly indicate that chronic adverse effects could occur at lower concentrations in more sensitive species of organisms. In spite of this possibility, it is difficult to conclude that there is significant risk from the benzene emissions at Town A. The maximum benzene concentration calculated in the analysis was 0.1 $\mu\text{g/l}$, more than 1,000 times smaller than these levels, and probably far below the concentration where adverse effects would occur.

Toluene. Only slightly more information is available for the chronic toxicity of toluene to aquatic organisms. The EPA (1980a) cites studies indicating adverse effects on some species at concentrations as low as 5.0 mg/l, with the possibility of lower concentrations for more sensitive species. These results are similar to those described above for benzene. From Table 3.11, we see that the maximum calculated toluene concentrations in the river are less than 0.12 $\mu\text{g/l}$, again a factor of more than 1,000 below the levels observed to be hazardous.

Xylene. Studies of the acute toxicity of marine organisms to xylene exposure have found LC_{50} levels ranging from 0.17 to more than 100 mg/l (Jori et al., 1986). Studies of algae growth found inhibition at similar concentrations, between 10 and 100 mg/l. These results, which are consistent with those for benzene and toluene, again compare favorably with the calculated xylene concentrations of 4 $\mu\text{g/l}$. Note also that we should be considering the *incremental* concentration of these pollutants associated with emissions from the source. Because we have assumed typical background concentrations in the river, the fraction of the total caused by source emissions is far smaller than the concentrations we have just been discussing.

Naphthalene. Acute and chronic toxicity studies for naphthalene indicate that adverse effects can occur in tested species at concentrations above 620 $\mu\text{g/l}$ (EPA, 1980b), and that these effects could occur at lower levels in more sensitive species. Our results indicate that source emissions may be responsible for incremental concentrations of 0.03 $\mu\text{g/l}$ on top of an assumed background level of 1.4 $\mu\text{g/l}$. As before, these concentrations are far below those indicated as being potentially hazardous to aquatic life.

Phenanthrene. There is virtually no information about the acute or chronic toxicity to aquatic organisms from PAH exposure. The very limited data, representing tests for only two chemicals in the group, found acute toxicity to saltwater aquatic life at concentrations as low as 300 $\mu\text{g/l}$ (EPA, 1980c). These results are far too limited for the EPA to draw conclusions about criteria for protection of aquatic life. We can only note that the 300 $\mu\text{g/l}$ is more than 20,000 times higher than our calculated concentrations for phenanthrene.

In general, the concentrations (even including background) of the five pollutants studied in this analysis should be much lower than the levels associated with adverse impacts on aquatic life. We must conclude that, on the basis of existing data, the emissions from the disposal site should not constitute a hazard to the local aquatic environment.

Health Risk Results

Using the equations presented above and the exposure rates shown for the various pathways and cases, we calculated the expected chronic health risk for each pollutant. These results—the incidence of adverse health effects in the exposed population (which varies by exposure pathway)—are shown separately by pollutant and pathway. For clarity, adverse effects on pregnant women have been separated from the other health effects in the tables. The background and net health risks for surface water exposure are presented in Table 3.28. This table shows the incidence of adverse health effects (number of cases per year) in the

exposed population. The per capita lifetime health risks are shown in Table 3.29. For the two birth defects, this represents the number of children born with the birth defect per person in the exposed population over an average human lifetime. Note that multipliers have been used in these tables to express the numbers in more easily readable format. The total risks in Table 3.28 should be multiplied by 0.0001 to obtain true risk results. Per capita lifetime risks shown in Table 3.29 should be multiplied by 10^{-6} . In other words, the per capita risks shown in Table 3.29 are in units of one part in a million.

Both total and per capita health risks from the surface water pathways are extremely small for all pollutants. The health risks associated with phenanthrene exposure (using

Table 3.28
ANNUAL HEALTH RISKS FOR SURFACE WATER PATHWAYS

Chemical/ Case	Pathway					
	Drinking Water		Fish/Oysters		Combined	
	Background	Site	Background	Site	Background	Site
General Toxicity						
Benzene						
C01	5.34	0.819	7.75	0.0021	13.1	0.821
C02	5.34	1.11	7.75	0.0042	13.1	1.11
C03	5.34	0.416	7.75	0.0011	13.1	0.417
C04	5.34	1.11	7.75	0.0042	13.1	1.11
Toluene—Toxicity						
C01	2.40	0.294	3.51	0.0015	5.91	0.295
C02	2.40	0.397	3.51	0.0031	5.91	0.400
C03	2.40	0.149	3.51	0.0008	5.91	0.150
C04	2.40	0.397	3.51	0.0031	5.91	0.400
Naphthalene						
C01	46.5	0.869	16.4	0.0085	62.9	0.877
C02	46.5	1.17	16.4	0.0172	62.9	1.19
C03	46.5	0.442	16.4	0.0043	62.9	0.446
C04	46.5	1.17	16.4	0.0172	62.9	1.19
Phenanthrene						
C01	153.7	20.8	860.4	0.308	1014.0	21.1
C02	153.7	28.1	860.4	0.618	1014.0	28.7
C03	153.7	10.6	860.4	0.157	1014.0	10.7
C04	153.7	28.1	860.4	0.618	1014.0	28.7
Birth Defects						
Toluene—Cleft Palate						
C01	0.0171	0.00210	0.0305	0.000013	0.0476	0.00211
C02	0.0171	0.00284	0.0305	0.000027	0.0476	0.00286
C03	0.0171	0.00107	0.0305	0.000007	0.0476	0.00107
C04	0.0171	0.00284	0.0305	0.000027	0.0476	0.00286
Xylene						
C01	1.20	0.00348	0.244	0.000022	1.45	0.00350
C02	1.20	0.00473	0.244	0.000044	1.45	0.00477
C03	1.20	0.00177	0.244	0.000011	1.45	0.00178
C04	1.20	0.00473	0.244	0.000044	1.45	0.00477

NOTE: Risks are expressed as total incidence of the health effect per year in the population caused by background exposure and the net increase from site emissions. All numbers in the table should be multiplied by 0.0001 to obtain true risks. Total population at risk in these calculations is 417,594.

benzo(a)pyrene data) are the largest by more than a factor of 20. Yet the per capita lifetime risk of consuming fish and oysters from the river and harbor is only about 1×10^{-8} . Even in the worst case, when we assume that the entire population consumes drinking water from the river and that there is no pollutant removal either in the stream or drinking water treatment processes, this risk remains below 5×10^{-7} . The combined risk of the other health effects in this worst-case situation is less than 5×10^{-8} , or one-tenth of the phenanthrene risk. The risk of birth defects from toluene or xylene exposure is less than 1×10^{-10} . These numbers should be compared with the often-used reference values of 1×10^{-5} or 1×10^{-6} for lifetime per capita health risk.

Table 3.29
LIFETIME PER CAPITA HEALTH RISKS FOR SURFACE WATER PATHWAYS

Chemical/ Case	Pathway					
	Drinking Water		Fish/Oysters		Combined	
	Background	Site	Background	Site	Background	Site
General Toxicity						
Benzene						
C01	0.0896	0.0137	0.130	0.000035	0.220	0.0138
C02	0.0896	0.0186	0.130	0.000071	0.220	0.0186
C03	0.0896	0.0070	0.130	0.000018	0.220	0.0070
C04	0.0896	0.0186	0.130	0.000071	0.220	0.0186
Toluene—Toxicity						
C01	0.0402	0.00492	0.0588	0.000026	0.0990	0.00495
C02	0.0402	0.00666	0.0588	0.000052	0.0990	0.00671
C03	0.0402	0.00250	0.0588	0.000013	0.0990	0.00252
C04	0.0402	0.00666	0.0588	0.000052	0.0990	0.00671
Naphthalene						
C01	0.780	0.0146	0.274	0.000143	1.05	0.0147
C02	0.780	0.0197	0.274	0.000289	1.05	0.0200
C03	0.780	0.0074	0.274	0.000072	1.05	0.0075
C04	0.780	0.0197	0.274	0.000289	1.05	0.0200
Phenanthrene						
C01	2.58	0.348	14.4	0.0052	17.0	0.353
C02	2.58	0.471	14.4	0.0104	17.0	0.481
C03	2.58	0.177	14.4	0.0026	17.0	0.179
C04	2.58	0.471	14.4	0.0104	17.0	0.481
Birth Defects						
Toluene—Cleft Palate						
C01	0.00029	0.00004	0.00051	<0.000001	0.00080	0.00004
C02	0.00029	0.00005	0.00051	<0.000001	0.00080	0.00005
C03	0.00029	0.00002	0.00051	<0.000001	0.00080	0.00002
C04	0.00029	0.00005	0.00051	<0.000001	0.00080	0.00005
Xylene						
C01	0.0202	0.00006	0.0041	<0.000001	0.0243	0.00006
C02	0.0202	0.00008	0.0041	<0.000001	0.0243	0.00008
C03	0.0202	0.00003	0.0041	<0.000001	0.0243	0.00003
C04	0.0202	0.00008	0.0041	<0.000001	0.0243	0.00008

NOTE: Risks are expressed as total lifetime probability of the health effect for a member of the population, caused by background exposure and the net increase from site emissions. All numbers in the table should be multiplied by 10^{-6} to obtain true risks.

The health risks associated with inhalation of volatile pollutants from the source are presented in Table 3.30. This table shows the incremental risk associated with both nominal and maximum emission rates for the tar pond emissions in the local area and at the pond itself. The table contains only per capita risk estimates for the latter, because no exposed population can be clearly defined.

As the table shows, for people in the area near the tar pond, the total and per capita lifetime risks associated with inhalation of volatiles are small. The highest risks, associated with benzene and naphthalene exposure (at the maximum emission rates) are on the order of one in a million. As we discussed above, the maximum emission rates are conservative upper bounds. Similarly, in Appendix B, we describe how the risk calculations for naphthalene are also conservative upper bounds and most likely are far smaller. These are thus conservative estimates of the risks for lifetime exposure from a tar pond with no soil cover and maximum pollutant concentrations throughout.

The risks associated with on-site activities are much higher, particularly for the maximum emission rates. Although these risk values would apply only to people working or playing continually at an uncovered site for long periods of time, they are still up to 3.7×10^{-4} for individual pollutants. These could be significant risks, if the calculations are not extremely conservative. Only better data on pollutant concentrations, soil characteristics, and pollutant health effects can resolve the uncertainties in these results. Note that we have not calculated a site-related risk for either of the two birth defects. It is not reasonable that pregnant women would be exposed to these concentrations for any significant time.

Table 3.30
HEALTH RISKS OF INHALATION PATHWAY

Chemical	Nominal Emissions		Maximum Emissions	
	Area	Site	Area	Site
Annual Risk				
General Toxicity				
Benzene	0.000143		0.000143	
Toluene—Toxicity	0.0000189		0.0000601	
Naphthalene	0.0000126		0.000884	
Birth Defects				
Toluene—Cleft Palate	1.43×10^{-7}		4.54×10^{-7}	
Xylene	5.05×10^{-8}		6.19×10^{-7}	
Lifetime per Capita Risk ($\times 10^6$)				
General Toxicity				
Benzene	0.274	59.2	0.274	59.2
Toluene—Toxicity	0.0362	7.83	0.115	24.9
Naphthalene	0.0242	5.24	1.69	365.5
Birth Defects				
Toluene—Cleft Palate	0.000274		0.000869	
Xylene	0.000097		0.00118	

NOTE: Total population at risk in these calculations is 36,597. Actual per capita risks have been multiplied by 10^6 to make the numbers more easily displayed.

The health risks associated with exposure to airborne soil and dust from the source are presented in Tables 3.31 and 3.32. These tables show the risk associated with tar pond emissions in the local area for each of the three cases. Case I is a worst case that assumes large dust and soil transport throughout a range of 1 km from the source. Case II considers only exposure to airborne soil and dust within that radius. Case III represents soil and dust exposure to personnel working or playing at the source. Table 3.31 shows total risk for the exposed population, and Table 3.32 summarizes the per capita lifetime risk for exposed individuals. The per capita lifetime risks shown in the table have been multiplied by one million, so that a value of 1.0 represents a lifetime risk of one in a million. There is no background risk calculated for this type of exposure, because there is no information about background concentrations in soil and dust. Because we are concerned with net risk, there was no reason to estimate background levels.

The health risks are shown for only one of the alternatives in each case. For Cases I and II, we have included the risks related to the higher fraction of naphthalene and phenanthrene (0.01) in the soil and dust. The risks associated with the lower pollutant concentrations are just one-tenth of those shown in the table for the two pollutants in those cases. For Case III, we have included the alternative having full concentrations of naphthalene and phenanthrene, but 10^{-5} of soil concentrations for benzene, toluene, and xylene. Because these three pollutants are so volatile, their concentrations should be reduced significantly in surface soil layers and suspended dusts.

It is clear from the table that the lifetime per capita risks of exposure to the volatile pollutants in airborne soil and dust from the tar pond is very small. The risk is below 10^{-9} for benzene, toluene, and xylene. For naphthalene and phenanthrene, however, the risks are much higher, particularly for Case III and the maximum concentrations of these pollutants. These

Table 3.31

ANNUAL HEALTH RISKS OF SOIL AND DUST EXPOSURE

Chemical	Case I	Case II	Case III
Nominal Concentrations			
General Toxicity			
Benzene	6.24×10^{-8}	7.39×10^{-9}	2.29×10^{-10}
Toluene—Toxicity	8.79×10^{-9}	1.04×10^{-9}	3.21×10^{-11}
Naphthalene	4.51×10^{-5}	5.33×10^{-6}	1.65×10^{-5}
Phenanthrene	2.58×10^{-2}	3.04×10^{-3}	9.45×10^{-3}
Birth Defects			
Toluene—Cleft Palate	7.84×10^{-11}	1.69×10^{-11}	0.00
Xylene	5.24×10^{-11}	1.13×10^{-11}	0.00
Maximum Concentrations			
General Toxicity			
Benzene	6.24×10^{-8}	7.39×10^{-9}	2.29×10^{-10}
Toluene—Toxicity	2.80×10^{-8}	3.31×10^{-9}	1.02×10^{-10}
Naphthalene	3.15×10^{-3}	3.72×10^{-4}	1.15×10^{-3}
Phenanthrene	6.72×10^{-1}	7.93×10^{-2}	2.45×10^{-1}
Birth Defects			
Toluene—Cleft Palate	2.50×10^{-10}	5.38×10^{-11}	0.00
Xylene	6.43×10^{-10}	1.38×10^{-10}	0.00

Table 3.32

PER CAPITA HEALTH RISKS OF SOIL AND DUST EXPOSURE

Chemical	Case I	Case II	Case III
Nominal Concentrations			
General Toxicity			
Benzene	0.000738	0.0000875	0.000457
Toluene—Toxicity	0.000104	0.0000118	0.000064
Naphthalene	0.533	0.0630	33.0
Phenanthrene	304.5	36.0	18910.0
Birth Defects			
Toluene—Cleft Palate	0.000000926	0.000000200	—
Xylene	0.000000620	0.000000133	—
Maximum Concentrations			
General Toxicity			
Benzene	0.000738	0.0000875	0.000457
Toluene—Toxicity	0.000331	0.0000390	0.000205
Naphthalene	37.3	4.40	2303.0
Phenanthrene	7938.0	936.7	490600.0
Birth Defects			
Toluene—Cleft Palate	0.00000236	0.000000636	—
Xylene	0.00000709	0.00000163	—

NOTE: All risks have been multiplied by 10^6 to simplify the display.

results for phenanthrene are somewhat misleading, because of the way in which we dealt with the health effects of that pollutant. As discussed above, we are using the health data for benzo(a)pyrene as a proxy. Moreover, much of this exposure is associated with dermal contact. When benzo(a)pyrene is applied to the skin, it does not cause gastric tumors but rather localized skin cancer. As a result, the risks shown should be taken as only indicative of the relative risk associated with exposure to the various pollutants from this source. The PAHs are generally more toxic than the other chemicals considered, but the actual magnitude will be different from that shown.

The size of these risks at the tar pond, although they may be high upper bounds, indicates that children should be prevented from playing on the site and that workers should be adequately protected when excavating or otherwise handling the tar and soil. We have noted that the tar pond is currently covered with a one meter soil layer. This and the nature of the tar itself should prevent any significant amount of contaminated soil or dust from escaping the area. However, if the soil cover is removed and the tar excavated for treatment or removal, care should be taken to control fugitive dust emissions from the material.

UNCERTAINTY

Major uncertainties in the calculations should be recognized. First, the adverse health effects of the selected pollutants are not well known. Only benzene and benzo(a)pyrene (as a proxy for phenanthrene) are recognized carcinogens with adequate data bases for quantitative risk assessment. The remaining three pollutants—toluene, xylene, and naphthalene—have not been studied extensively and do not have well-documented health risks. The PAHs, in general,

have not been examined individually in either animal tests or epidemiologic analyses. Their health effects must be determined more quantitatively before definitive conclusions can be drawn.

Second, we have assessed the exposure and risks associated with five selected pollutants. These pollutants were chosen because they are present at high concentrations in the source and they should represent all of the chemicals in terms of transport characteristics and health risks. However, we have not investigated all of the chemicals present or the possible synergistic interactions among them. Benzo(a)pyrene, for example, is a cocarcinogen with many heavy metals, including nickel and chromium. Although there are sufficient data to recognize this behavior, there is not enough information available to deal adequately with it. There may be other instances of this type of relationship that are currently unknown and that we have not been able to consider in this limited analysis. No one will be able to estimate the total health risks accurately until more complete health data are compiled and a more thorough study can be undertaken.

Third, our results are based on calculated emission rates into the groundwater, surface water, and air. Should conditions change or the tar pond and lake bed deposits be disturbed, these emission rates should be monitored or reinvestigated to determine that they have not significantly increased. The current risks from the five pollutants investigated are generally small, even under conservative assumptions, but these risks could become important if the emission rates assumed in the study are substantially underestimated.

CONCLUSIONS

These results indicate that there should be very little potential health risk (for the five pollutants considered) to the people of Town A or the population downstream along the river. The incremental exposure rates and health effects associated with potential surface water contamination are small, even assuming that the river could be otherwise cleaned up and made suitable for drinking and fishing. Similarly, the risks to area residents from exposure (either through inhalation, ingestion, or dermal contact) to the volatile pollutants in the tar deposits also seem to be small, even under conservative assumptions.

The potential risks associated with exposure to dusts and soil containing naphthalene and phenanthrene are more difficult to characterize. Although these risks seem high in some cases, particularly for personnel frequently working at the tar pond for long periods, they are based on extremely conservative assumptions and calculation procedures that should produce upper bounds. In particular, they assume (1) that the site is not covered by dirt over a period of many years; (2) that the maximum concentrations measured at the site are characteristic of the entire tar pond; and (3) that large amounts of dust and soil can be picked up from the tar deposits by the wind. As a result, it is likely that the real risks to the area population from these exposure pathways are not important under existing conditions. They should remain unimportant if fugitive dust emissions are controlled whenever the tar deposits are uncovered.

Finally, the calculations indicate some potential for risk (again under conservative, upper-bound assumptions) to workers at the site. Accordingly, workers who visit the site regularly and remain there for long periods (particularly if the tar pond is excavated for treatment) should be adequately protected from exposure to the pollutants.

We have been able to describe and apply in a limited manner an approach to risk assessment for coal tar disposal locations. This assessment has been based on reasonable (but far from complete) data about the concentrations of pollutants in the source. For most current

coal tar disposal sites, very little information about the contents and history of the site is available. As a result, this analysis has probably provided better estimates of potential risks to the neighboring population than are available for most locations.

We have tried to illustrate the types of exposure pathways that should be considered and methods for evaluating the risk to the regional population. Although the results indicate that the potential risks are generally small for selected pollutants, they are not definitive. Most of the calculations are highly uncertain, and we believe they represent conservative upper bounds on exposure rates and health effects. This analysis has demonstrated that coal tar risk assessment requires extensive data about the contaminant sources and local environment. It has also shown that the state of the art must be advanced before risk analysis can provide more confident estimates of the real health risks. This advancement will come from research into how pollutants behave in the environment and how they affect the chronically exposed population.

Appendix A

ATMOSPHERIC EMISSION FLUX FROM THE TAR POND

This analysis will provide reasonable upper-limit estimates of the fluxes of volatile hydrocarbons (benzene, toluene, naphthalene, and xylene) from the tar disposal pond at the site. These hydrocarbons have been detected at concentrations in the range of 44 to 30,000 $\mu\text{g/gm}$, at a depth of approximately 3 m. The concentration profiles of the chemicals within the tar pond have not been determined by the monitoring program.

Because of the lack of data on the vertical distribution of the tar in the pond, the degree of soil water content, soil porosity, temperature history, and sorption isotherms for the volatile organics in contact with the tar solids, several simplifying assumptions must be made in this analysis. These simplifying assumptions should make the calculated emission rates, and the time required to reach equilibrium, conservative to very conservative estimates.

PHYSICAL DESCRIPTION OF THE TAR ZONE

The tar deposited soil is divided into four horizontal layers of different depths (Figure A.1). The top layer is a soil cover of depth y_1 . It is kept variable to provide insight into the effectiveness of placing a soil cover over the tar zone. The second layer is an unsaturated tar zone 3 m deep. The third layer is a soil zone saturated with tar. Hence, the third layer has zero soil-air and soil-water content. Its depth is also 3 m, and we will assume that it is at a constant contaminant concentration, C_o , at the unsaturated tar zone/soil saturated tar interface. Finally, the fourth horizontal layer consists of a water-saturated soil (groundwater) that extends 50 m to the impermeable bedrock.

THEORETICAL FRAMEWORK AND TRANSPORT PARAMETERS

The transport of the organic chemicals is predicted based on a simple diffusion model, assuming that chemical equilibrium exists among air, water, and tar solid phases. This simple diffusion model is given by

$$\frac{\partial C}{\partial t} = D \frac{\partial^2 C}{\partial z^2} \quad (\text{A.1})$$

where D = equilibrium soil diffusion coefficient (cm^2/s),

C = composite chemical concentration in the soil matrix ($\mu\text{g}/\text{cm}^3$),

z = depth (cm), and

t = time (sec).

In this equation, the value for D is based on the following equation (Cohen and Ryan, 1985),

$$D = \frac{(D_a/\tau_a)H_{wa}\theta_a + (D_w/\tau_w)\theta_w + (D_s/\tau_s)(1 - \theta_w - \theta_a)H_{ws}}{\theta_w + \theta_a H_{wa} + (1 - \theta_w - \theta_a)H_{ws}} \quad (\text{A.2})$$

where D_i = molecular diffusion coefficient in phase i (cm^2/s),

τ_i = tortuosity factor in phase i ,

Fig. A.1—Schematic diagram of the tar deposits

θ_i = volumetric phase content of phase i , and

H_{ij} = partition coefficient for phases i and j .

The final term in the numerator of Eq. (A.2) can be ignored because the molecular diffusion coefficient for soil should be negligibly small. In the absence of porosity data for the individual horizontal soil layers, the porosity of the soil cover and the unsaturated tar zone is set to a constant value of 0.5. Finally, the partition coefficient H_{ij} is calculated based on experimental data and semi-empirical estimation procedures.

Physicochemical properties used for estimating the transport of benzene, naphthalene, toluene, and xylene from the source are listed in Table A.1. The parameter values reported in the table are for an environmental temperature of 15°C, which is an average daytime temperature. Unfortunately, the sorption of organics by the solid phase of the tar has not been measured, and no data have been located in the literature. Consequently, the soil-water/soil-solids partition coefficient is calculated assuming 2 percent organic carbon content and a bulk soil density of 1.5 g/cc.

The adsorption of organic chemicals in the soil increases with carbon content. Accordingly, it is likely that the partition coefficients between tar-saturated soil and air or water would be considerably different from those between "normal" soil and air or water, because

tar-saturated soil should have a carbon content greater than 2 percent. Under such conditions, Eq. (A.2) indicates that the effective diffusion for tar-saturated soil would be much less than for "normal" soil. Consequently, the assumed 2 percent organic carbon content will lead to a conservative estimate of the diffusion rates.

The soil diffusion coefficient is calculated from Eq. (A.2) using the data in Table A.1 at air phase volume fractions of 0.00, 0.25, and 0.50, as shown in Table A.2. For the volatile organics investigated, naphthalene was determined to have the smallest soil diffusion coefficient at all air phase volume fractions. Benzene, toluene, and xylene were found to have similar diffusion coefficient values.

The resistance to chemical volatilization from the soil surface to the atmosphere can be determined as follows. The atmospheric-side mass transfer coefficient, k_g , above the soil surface, modeled as gravel, is calculated based on the paper by Brutsaert (1975). Table A.3 lists the dependence of this mass transfer coefficient based on wind speed at a height of 10 m. The corresponding atmospheric mass transfer resistance to pollutant transport from the soil is calculated at the ratio of the air diffusivity of the chemical divided by the air-side mass transfer coefficient, D_a/k_g . For the range of k_g listed in Table A.3 and the air phase diffusivities given in Table A.1, the atmospheric-side resistance to mass transfer is at the most 0.1 cm. This latter value is insignificant relative to the resistance of the top soil layer. Also, the volatile organic concentrations in the soil for this study are expected to be much greater than their corresponding air concentrations. Thus, the air concentration for the chemicals is assumed zero (for computational and presentation convenience). Consequently, the contaminant concentration at the top of the soil surface is set equal to zero.

Table A.1

PHYSICOCHEMICAL PROPERTIES FOR ORGANICS AT 15°C

Organic Chemical	Molecular Diffusivity (cm ² /s)		Partition Coefficient	
	D_a (Vapor)	$D_w \times 10^5$ (Liquid)	H_{wa}	H_{ws}
Benzene	0.084 (1)	0.88 (1)	0.146 (2)	2.44
Toluene	0.076 (1)	0.73 (1)	0.174 (2)	3.00
Xylene	0.069 (1)	0.68 (1)	0.169 (3)	10.6
Naphthalene	0.066 (1)	0.64 (1)	0.031 (3)	30.7

SOURCES: (1) Reid et al. (1977); (2) Leighton et al. (1981); (3) MacKay and Patterson (1981).

Table A.2

DIFFUSION COEFFICIENTS FOR VOLATILE ORGANICS
SOIL DIFFUSIVITY (cm²/s) $\times 10^{-3}$

Organic Chemical	Air Phase Volume Fraction (θ_a)		
	0.0	0.25	0.50
Benzene	0.0024	0.546	4.42
Toluene	0.0017	0.494	3.88
Xylene	0.00054	0.140	1.01
Naphthalene	0.00018	0.0088	0.062

Table A.3

GAS SIDE MASS TRANSFER COEFFICIENT

Wind Speed (m/s)	k_g (cm/s)
5	32
1	6.5
0.1	0.66

ANALYSIS AND RESULTS

Two cases are considered to elucidate the expected transport behavior of the chemicals in the tar pond. In Case A the pollutant flux to the atmosphere, in the absence of a soil cover, can be calculated by assuming typical concentrations (in the tar-saturated soil zone) of the chemicals reported for tar. In Case B the role of the soil cover is investigated by calculating the pollutant flux to the atmosphere at two soil cover depths of 1 m and 4 m. For both cases the diffusivity values calculated for the soil cover and unsaturated tar zone are for an air content of 0.25.

Case A

The contaminant mass flux to the atmosphere, N_a , is calculated (as a function of time) based on an initially polluted layer of unsaturated tar deposit in contact with the atmosphere. The saturated tar deposit is modeled as a contaminant source at a constant concentration, C_o , at the bottom boundary of the unsaturated tar zone (see Figure A.1). The corresponding flux, N_a , to the atmosphere is given by Kreyszig (1972) as:

$$N_a(z = 0, t) = (C_o D / L) [1 + 2 \sum_{n=1}^{\infty} \exp(-Dt(\pi n / L)^2)] \quad (\text{A.3})$$

in which L (equal to 3 m) is the height of the initially polluted unsaturated tar zone. The dimensionless pollutant flux expressed in terms of the Sherwood number ($Sh = N_a L / DC_o$) is given in Table A.4. The dimensionless pollutant flux changes appreciably, by about 100 percent, between the tenth day and the fiftieth day for all chemicals. For longer periods of time the exponential term in Eq. (A.3) becomes insignificant, and the dimensionless pollutant flux approaches unity asymptotically.

Finally, sample results of the dimensional pollutant fluxes computed for two different concentrations are given in Table A.5. The two concentrations are the measured concentration reported for the source and the maximum concentration reported for tar at the site.

As shown in the table, the nominal pollutant flux for all four chemicals is on the order of $1 \text{ ng/cm}^2/\text{s}$. The flux for naphthalene varies significantly between the nominal and maximum values, because the measured naphthalene concentrations in the soil fall within a wide range. Accordingly, the expected (upper-bound) flux for naphthalene could be similar to that of the other pollutants or up to an order of magnitude greater, depending on the actual average concentration in the tar deposits at the site. To resolve this uncertainty, it would be necessary to make additional measurements of naphthalene concentrations at different locations in the tar deposit.

Table A.4

POLLUTANT FLUX (SHERWOODS) TO THE ATMOSPHERE—NO SOIL COVER

Organic Chemical	Soil Diffusivity $\times 10^4$ (cm ² /s)	Pollutant Flux (Sh)				
		Day 10	Day 20	Day 30	Day 40	Day 50
Benzene	5.46	7.8	5.5	4.5	3.9	3.5
Toluene	4.94	8.2	5.8	4.7	4.1	3.7
Xylene	1.40	15	11	8.9	7.7	6.9
Naphthalene	0.088	61	43	35	31	27

Table A.5

POLLUTANT FLUX TO THE ATMOSPHERE—NO SOIL COVER
(ng/cm²/s)

Organic Chemical	Measured C _o		Maximum Coal Tar C _o	
	Day 10	Day 50	Day 10	Day 50
Benzene	5.3	2.4	5.3	2.4
Toluene	1.6	0.71	5.0	2.3
Xylene	0.31	0.14	3.8	1.7
Naphthalene	0.77	0.34	53.7	23.8

For the single-layer model described by Eq. (A.3), both the pollutant flux, N_a , and the characteristic time for reaching steady state, L^2/D , depend on the diffusion coefficient, D . If D were much smaller (because of a higher organic carbon content in the tar-saturated soil), the flux would be correspondingly smaller and the time to reach steady state much longer.

Case B

The importance of a soil cover in changing the pollutant flux to the atmosphere can be evaluated by examining two soil cover depths, 1 m and 4 m. As in Case A, the initial concentration of the volatile organics is assumed to be uniform for the unsaturated tar depth of 3 m, but initially zero within the soil cover. The initial and boundary conditions are:

$$C = 0 \quad \text{at } t = 0 \quad z < y_1 \quad (\text{A.4a})$$

$$C = C_o \quad \text{at } t = 0 \quad y < z < L \quad (= 3 + y_1) \quad (\text{A.4b})$$

$$C = C_o \quad \text{at } t > 0 \quad z = L \quad (\text{A.4c})$$

The solution of Eq. (A.1) subject to the above initial and boundary conditions is given by Carslaw and Jaeger (1959):

$$C(z,t) = C_o(z/L) + 2/\pi \sum_{n=1}^{\infty} \exp(-a_n) \cos(b_n) \sin\{(C_n)(Z/L)\} / n] \quad (\text{A.5})$$

where the variables a_n , b_n , and c_n are defined by

$$a_n = Dn^2\pi^2t/L^2 \quad (\text{A.6a})$$

$$b_n = n\pi y_1/L \quad (\text{A.6b})$$

$$c_n = n\pi \quad (\text{A.6c})$$

The corresponding atmospheric flux for the chemicals to the atmosphere is given by

$$Sh = 1 + 2 \sum_{n=1}^{\infty} \exp(-a_n) \cos(b_n) \quad (\text{A.7})$$

Ideally, the partition and diffusion coefficients should have different values in the soil cover and the unsaturated tar layer. This could not be done because the required data were unavailable. Consequently, the addition of the soil layer only changes the initial conditions and increases the time to steady state when compared with Case A.

For the 1 m soil cover the pollutant fluxes expressed in terms of the Sherwood number ($Sh = N_a L / DC_o$) are given in Table A.6. The dimensionless pollutant flux does not change appreciably—by less than 20 percent between the fiftieth and hundredth day for benzene and toluene. The pollutant fluxes for benzene and toluene attain a maximum at about 100 days (see Table A.6). For xylene, the dimensionless pollutant flux still increases after 150 days. Finally, the naphthalene Sherwood numbers at 50 and 150 days are insignificant. Consequently, naphthalene pollutant fluxes were calculated for 500, 1,000, and 1,500 days. For naphthalene, the pollutant flux keeps increasing, even four and one-half years from time zero. For longer periods of time, identical to Case A, the exponential term in Eq. (A.7) will become insignificant, and the dimensionless pollutant flux will asymptotically approach unity.

Finally, the actual pollutant fluxes computed from the dimensionless flux values given in Table A.6 are calculated for two concentrations. The two concentrations are the measured concentration reported for the source and the maximum concentration measured at the site. The pollutant fluxes are reported at 10 and 50 days in Table A.7, except for naphthalene for which the pollutant fluxes are reported at 500 and 1,500 days.

A final set of sample results for a 4 m soil cover is presented in terms of the dimensionless pollutant flux. The results are given in Table A.8. The extra 3 m of soil cover significantly affects the pollutant flux. A much longer time is needed for the dimensionless pollutant flux to approach unity when compared with a 1 m soil cover (see Tables A.4, A.6, and A.8).

Table A.6

POLLUTANT FLUX (SHERWOODS) TO THE ATMOSPHERE—1 M SOIL COVER

Organic Chemical	Soil Diffusivity $\times 10^4$ (cm ² /s)	Pollutant Flux (Sh)		
		Day 50	Day 100	Day 150
Benzene	5.46	1.61	1.93	1.88
Toluene	4.94	1.51	1.92	1.91
Xylene	1.40	0.147	0.82	1.33
Naphthalene	0.088	Day 500	Day 1000	Day 1500
		0.016	0.30	0.75

Table A.7

POLLUTANT FLUX TO THE ATMOSPHERE—1 M SOIL COVER
(ng/cm²/s)

Organic Chemical	Measured C _o		Maximum Coal Tar C _o	
	Day 50	Day 150	Day 50	Day 150
Benzene	0.81	0.95	0.81	0.95
Toluene	0.22	0.27	0.69	0.87
Xylene	0.0023	0.021	0.028	0.26
	Day 500	Day 1500	Day 500	Day 1500
Naphthalene	0.00015	0.0071	0.011	0.50

Also, in this case no local maximum in the pollutant flux will be observed with time. Finally, similar to results for the 1 m soil cover, the Sherwood number will asymptotically approach unity for long periods of time.

CONCLUSIONS

The volatilization fluxes to the atmosphere were calculated in the absence of soil cover, and for 1 m and 4 m soil cover depths. The presence of a soil cover decreases the pollutant flux to the atmosphere by one order of magnitude (Tables A.5 and A.7). The presence of the 1 m soil cover (Case B) retards the initial pollutant flux and decreases the maximum Sherwood number compared with Case A, in the absence of a soil cover (Tables A.4 and A.6). Also, the presence of a 4 m soil cover (1 m thicker than the unsaturated tar zone) significantly decreases the initial dimensionless pollutant flux to the atmosphere.

After sufficient time elapsed for the Sherwood number to approach unity in the three examples, the upper-bound fluxes of benzene, toluene, and xylenes are on the order of 0.10 to 1.0 ng/cm²/s. Finally, the time for the Sherwood number to approach unity increases from a few days for a 1 m soil cover to at least a year for the 4 m soil cover. Although the residence time for the pollutants in the soil is significant, the order of magnitude for the residence time is uncertain, because it depends on the soil diffusivity.

Unfortunately, the precise values of the diffusivities cannot be determined because the actual air and water contents of the soil are unknown. If the soil is closer to a dry soil

Table A.8

POLLUTANT FLUX (SHERWOODS) TO THE ATMOSPHERE—4 M SOIL COVER

Organic Chemical	Soil Diffusivity × 10 ⁴ (cm ² /s)	Pollutant Flux (Sh)		
		Year 2	Year 4	Year 6
Benzene	5.46	0.68	0.88	0.94
Toluene	4.94	0.62	0.86	0.93
Xylene	1.40	0.04	0.31	0.54
Naphthalene	0.088	< 10 ⁻¹⁰	< 10 ⁻¹⁰	10 ⁻¹⁰

compared with the value assumed in the current analysis, the pollutant flux will be higher than currently estimated. Furthermore, if the actual soil is saturated with water, the pollutant flux will decrease significantly compared with the results presented. In the former case, its persistence in the soil will decrease, but in the latter case, its persistence in the soil will increase significantly. The additional effect of rainfall, however, would tend to increase the persistence (in the soil) of the chemical by pushing (by convection) the organics deeper into the soil toward the saturated soil zone.

Measurements of pollutant fluxes at the air/soil interface can provide data for comparison with the predictions shown in Tables A.5 and A.7. Such a comparison will help to determine whether the tar zone is indeed saturated. Data on pollutant concentrations with depth would also be helpful in calculating chemical volatilization fluxes. Improved model predictions for the expected transport of the chemicals in the soil will require physical data for the soil (porosity, organic carbon content or type of soil classification, air and water content, etc.).

Additional limitations of the current analysis include the neglect of biodegradation in the soil and water evaporation from the soil to the atmosphere. If biodegradation is occurring it will lead to a decrease in the volatilization flux while water evaporation may lead to increased contaminant volatilization flux. Currently, however, there is no established method for estimating the above effects. One could speculate that the high concentrations of the various organics in the tar pond may be too high for bioactivity, but at this point it can only be speculation. Water evaporation will be significant only for compounds with a preferred partition toward the air phase, thus, for the compounds considered in this analysis it is unlikely that water evaporation will significantly affect the volatilization flux.

In closure, the present analysis should provide a reasonable upper-limit order-of-magnitude estimate of the volatilization flux for the four contaminants considered. The results represent several scenarios to illustrate the potential magnitude of the volatilization fluxes. Equations (A.1) through (A.7) are analytical and should allow the calculation of the volatilization fluxes for other scenarios.

Appendix B

TOXICITIES FROM INGESTION OF XYLENE, TOLUENE, NAPHTHALENE, BENZO(A)PYRENE, AND BENZENE

Toxic endpoints resulting from exposure to xylene, toluene, naphthalene, benzo(a)pyrene, and benzene are numerous, including acute death, central nervous system toxicity, fetotoxicity, teratogenicity, hemolysis, carcinoma, and leukemia. For a particular agent, the endpoint exhibited depends on route of administration, dose rate, and total time of exposure. However, of these five toxic substances, the group consisting of xylene, toluene, and naphthalene may be classified as noncarcinogenic whereas the group of benzo(a)pyrene and benzene is clearly carcinogenic.

In the following sections, we briefly review the toxicities associated with each substance and identify the one most appropriate for quantitative risk estimation. We then present relevant experimental data and an analysis of the data. It is apparent that the data bases for all of these substances, especially naphthalene, are quite limited and that, as a result, only rough estimates of response are possible. The illustrative quantitative response calculated for each substance will be an upper-level steady-state number of toxic cases per year per 10^5 of the total population, given that each member of the population is exposed to a constant dose rate over his or her lifetime. In the case of reproductive toxicity assessment, it will be assumed that this population has a zero growth rate and that the average life span is 70 years.

XYLENE

Toxicity Review

Fishbein (1985) has recently reviewed the environmental and toxicological aspects of exposure to xylene. Of note is the observation that exposure is to mixed xylenes rather than individual isomers. In the general environment, exposure to each of the individual isomers is probably of the same order of magnitude as found in commercial xylene (40 percent meta, 20 percent ortho, 20 percent para, and 20 percent ethylbenzene). Exposure to xylenes from utility sources would also be expected to be a mixed exposure.

Metabolism plays an important role in the pharmacokinetics of the mixed xylenes, accounting for the fate of 95 percent of the compounds absorbed (Fishbein, 1985). Xylene metabolism in both humans and animals is relatively well understood; the primary route of transformation is oxidation of the absorbed isomers to the corresponding toluic acids followed by their conjugation to glycine to form o-, m-, and methylhippuric acids. These latter compounds are excreted in the urine and account for 95 percent of the metabolized xylene. A minor pathway (< 2 percent of absorbed xylene) involves ring hydroxylation to produce xylenols and their excretion either as unchanged components or glucuronide conjugates.

Of possible relevance to risk estimation are two modifications of this scheme at high dose. First, metabolism of xylenes occurs in the liver via the microsomal mixed function oxidase system and cytoplasmic dehydrogenases (Carlone and Fouts, 1974; Harper et al., 1975). At very high doses, the capacity of this system may be overwhelmed and both xylene and its metabolite concentrations in target tissues may become nonlinear functions of dose. Second, the depletion

of rat liver glutathione (an electrophile scavenger) by very high doses of o-xylene (Van Doorn et al., 1980a and 1980b) suggests that a highly reactive intermediate may exist, whose concentration depends on administered dose in a highly nonlinear fashion because of glutathione saturability.

Both of these effects may occur at the high dose levels employed in the xylene toxicity studies described below. Thus, for those experiments, toxic response is probably more closely related to the target tissue dose of reactive metabolite than to the administered dose, suggesting that extrapolations of response from high to low dose should be based upon local reactive metabolite concentrations. However, although these considerations must eventually be incorporated into refined models of xylene dose response, we have not done so in the present study for two reasons. First, it is not known which metabolite or metabolites to associate with a given toxic endpoint. Second, if we ignore these effects and perform dose-response extrapolations with a linear model, we are likely to obtain upper-bound estimates of expected risk, and these will be adequate for our risk assessments. Only if the experimental response data were taken in a region of extreme saturation of mixed function oxidase activity—and ours does not appear to be in such a region—would such upper estimates not be obtained (Hoel et al., 1983).

Quantitative toxicity data for xylene are very limited relative to that for benzene and even toluene (Fishbein, 1985). At very high doses, narcosis is observable. The acute LD₅₀ of rats following oral exposure to xylene (in olive oil) was reported by Wolf et al. (1956) as 4.3 g/kg body weight and by Hine and Zuidema (1970) as 10 ml/kg. Qualitative toxicities involving liver, kidney, and heart damage have been reported for both acute and chronic inhalation exposures, but little long-term xylene epidemiology exists for quantification (Von Burg, 1982; Fishbein, 1985). Central nervous system toxicity following 70-minute inhalation periods at 435 and 1300 mg/m³ has been reported for exercising humans but not for those at rest. Short-term memory and reaction time were not affected (Gamberale et al., 1978; Olson et al., 1985).

As of 1985, no animal data existed that adequately demonstrated carcinogenicity (Fishbein, 1985). Furthermore, many short-term genetic tests indicate that xylene is not a mutagen. This absence of mutagenic response was observed in assays employing bacteria, mouse lymphoma cells, yeast cells, rat hepatocytes, and human lymphocytes (Litton Bionetics, 1978; Bos et al., 1981; Gerner-Smidt and Friedrich, 1978).

Although xylene's embryotoxic and teratogenic effects may be responsible for irreversible damage, it is clearly not a selective teratogenic agent (in the sense that major effects occur at doses well below the maternal toxic dose) (Hood and Ottley, 1985). Kucera (1968) noted that xylene induced visible malformations in chick embryos. Inhalation studies in CFY rats over days 9–14 of pregnancy led Hudak and Ungvary (1978) to conclude that significant increases occurred in the number of fused sternbrae and extra ribs in fetuses continually exposed to 1,000 mg/m³ of mixed xylenes. These skeletal anomalies were not scored as true malformations by these investigators, thus they concluded that mixed xylene was not teratogenic. The skeletal anomalies plus retarded fetal development were interpreted as embryotoxic. In later inhalation studies of individual xylene isomers, Ungvary et al. (1980) again confirmed the presence of retarded fetal development (all isomers) and extra ribs (m- and p-xylene) but denied teratogenicity. On the other hand, two studies involving the oral dosing of CD-1 mice concluded that mixed xylenes were teratogenic (Marks and Ledoux, 1982; Nawrot and Staples, 1981). This teratogenicity is weak and has been observed only at doses that are also toxic to the mother. The principal malformation observed was cleft palate. No studies have yet explained the differences between the inhalation and oral exposure experiments in terms of species-specific pharmacokinetics or target tissue sensitivity.

Rate of Toxicity Induction by Xylene Ingestion

The toxic endpoint used to derive a quantitative estimate of the response of a population exposed to xylene is the teratogenicity induced by ingestion of this compound. This endpoint was chosen because carcinogenicity has not been demonstrated, because teratogenicity is irreversible, unlike most central nervous system impairment, and because teratogenicity, once present, is not expressed over a response continuum like liver damage. We begin with the mouse data of Marks and Ledoux (1982), select a dose-response function to fit the data, and, from this fit and interspecies scaling, estimate an incidence rate Q , the number of malformations induced per year per standard dose per 10^5 people. The Marks and Ledoux (1982) data appear in Table B.1.

In this assay, pregnant CD-1 mice were dosed by gavage with mixed xylenes in cottonseed oil over days 6 to 15 of gestation. On day 18 of gestation, each dam was killed and external anomalies (recorded in footnote to Table B.1) were counted. The primary nonskeletal malformation observed was cleft palate; cleft palates appearing in stunted fetuses (less than 0.5 g) were not counted.

The dose-response model used to analyze these data was the one-hit model. This simple model is subject to the criticism that it is formulated upon the assumption that transition to the final toxic state is irreversible, yet progression toward the teratogenic outcome in utero may not be irreversible. If teratogenicity resulting from exposure to a particular substance involved a mutation, irreversible by definition, this model, for instance (or a multistage model), would be appropriate. If there were hormonal regulation, a reversible mechanism could be

Table B.1

RESPONSE OF CD-1 MICE TO ORALLY ADMINISTERED MIXED XYLENE

Dose ^a (mg/kg/day)	Response ^b	
	Malformed	Total
2,580	12	224
2,060	7	227
1,030	3	232
520	2	208
0	2	658

SOURCE: Marks and Ledoux (1982)

NOTE: The litter is the usual unit for analysis (used to avoid a biased result due to a concentration of malformations in a very few litters); however, in these data, concentration in a few litters is slight and the number of fetuses was used as a statistical unit.

^aDoses of 3,100 and 4,130 excluded because of high maternal/fetal death and Marks and Ledoux's exclusion of dead fetuses from the assay.

^bNumber of malformed fetuses and total fetuses observed, where malformations are defined as cleft palate + fused arches and ribs + fused sternbrae + missing ribs and arches; bilateral wavy ribs were excluded.

involved up to birth, and other models might be considered. In the case of xylene, no molecular detail regarding teratogenicity is known. Hence, lacking other information, the one-hit model was chosen for analysis because it provided an adequate empirical fit to the data in Table B.1, and because it was mathematically simple.

The one-hit model (Bolten et al., 1987) is given by

$$P(d) = 1 - \exp(-q_0 - q_1 d) \quad (\text{B.1})$$

where $P(d)$ is the cumulative incidence of toxic response (malformations), just after the end of the exposure period (day 18 of gestation) to dose d . $P(d)$ may be considered the cumulative response following an entire pregnancy, since extension of the exposure beyond day 15 would be well past the time interval when cleft palate may be induced. $P(0) = 1 - \exp(-q_0)$ is the background rate of toxic response.

After taking the log of Eq. (B.1), $-\ln[1 - P(d)]$ was fit to the data of Table B.1 using a weighted linear least squares technique. Identifying each experimental dose-response pair by index i , and defining each experimental response as

$$E_i = -\ln(1 - P_i) \quad (\text{B.2})$$

and the corresponding theoretical response from Eq. (B.1) as

$$T_i = q_0 + q_1 d_i \quad (\text{B.3})$$

the weighted least squares objective function S is

$$S = \sum_i (E_i - T_i)^2 / \sigma_i^2 \quad (\text{B.4})$$

where σ_i^2 is the estimated variance of E_i . The variance weighting was required to account for the different sample sizes used at each dose. Because each response is a ratio, σ_i^2 was estimated from the experimental response as the variance of a proportion, i.e., as $P_i(1 - P_i)/n_i$ where n_i is the number of animals tested at dose d_i . (Strictly this variance applies to $P(d_i)$ rather than E_i , but the low $P(d_i)$ range in Table B.1 makes $P(d_i)$ a good approximation to E_i .)

Because T_i can be written as a linear function of the q 's, S may be easily minimized, and analytical expressions for the mean and variance of each q parameter are available (Mathews and Walker, 1965). Introducing parameters C_{im} defined by

$$T_i = \sum_{m=1}^2 c_{im} q_{m-1} \quad (\text{B.5})$$

where, from Eq. (B.3), $c_{i1} = 1$ for all i , $c_{i2} = d_i$, and variables M_{lm} and X_m are defined by

$$M_{1m} = M_{m1} = \sum_{i=1}^N C_{im} C_{i1} / \sigma_i^2 \quad 1 \leq m, 1 \leq 2 \quad (\text{B.6})$$

$$X_m = \sum_{i=1}^N C_{im} E_i / \sigma_i^2 \quad (\text{B.7})$$

where N is the number of doses ($N = 5$ for xylene including control), the expressions for the means and variances of q_0 and q_1 are, respectively,

$$\begin{bmatrix} q_0 \\ q_1 \end{bmatrix} = \frac{1}{\det[M]} \begin{bmatrix} M_{22} & -M_{12} \\ -M_{21} & M_{11} \end{bmatrix} \begin{bmatrix} X_1 \\ X_2 \end{bmatrix} \quad (\text{B.8})$$

$$\begin{bmatrix} \text{var}(q_0) \\ \text{var}(q_1) \end{bmatrix} = \frac{1}{\det[M]} \begin{bmatrix} M_{22} \\ M_{11} \end{bmatrix} \quad (\text{B.9})$$

Accordingly, best fit values to the mouse xylene data of Table B.1 were obtained as $q_0 = 2.68 \times 10^{-3}$, $q_1 = 1.52 \times 10^{-5} \text{ (mg/kg/day)}^{-1}$, $\sigma_{q_0} = [\text{var}(q_0)]^{1/2} = 2.08 \times 10^{-3}$, and $\sigma_{q_1} = [\text{var}(q_1)]^{1/2} = 3.66 \times 10^{-6} \text{ (mg/kg/day)}^{-1}$. The 95 percent upper confidence value for q_1 , applicable to the mouse, is

$$q_{1,\text{mouse}}^* = q_1 + 1.96 \sigma_{q_1} = 2.24 \times 10^{-5} \text{ (kg/day)}^{-1} \quad (\text{B.10})$$

Because we are ultimately interested in assessing human risk, this $q_{1,\text{mouse}}^*$ must be scaled to its human counterpart. Based upon simple one-compartment pharmacokinetic considerations (Bolten et al., 1986 and 1987), this may be accomplished approximately by the body weight scaling

$$q_{1,\text{human}}^* = q_{1,\text{mouse}}^* \left[\frac{W_{\text{human}}}{W_{\text{mouse}}} \right]^{1/3} \quad (\text{B.11})$$

We chose the human and mouse weights to be the average weights of each species during pregnancy; for humans, $50 \text{ kg} \times 1.1 = 55 \text{ kg}$; for mouse $0.022 \text{ kg} \times 1.35 = 0.03 \text{ kg}$. Thus

$$q_{1,\text{human}}^* = 2.75 \times 10^{-4} \text{ (mg/kg/day)}^{-1} \quad (\text{B.12})$$

The excess human risk over background at low dose, $A(d)$, for $q_0 \ll 1$ is approximately $P(d) - P(0)$ (Bolten et al., 1987) or

$$A^*(d) = q_{1,\text{human}}^* d \quad (\text{B.13})$$

where the asterisks signify 95 percent upper confidence estimates. For teratogenic outcomes, $A^*(d)$ may be regarded as the excess risk of a malformed fetus per pregnancy.

Because $A^*(d)$ applies to a single pregnancy, it is not directly useful in assessing the risk to an entire exposed population. We therefore compute a final metric Q^* , the upper 95 percent confidence estimate of the incidence rate of malformations expected in a human population of 10^5 reproducing at a zero net growth rate and consuming 2 l/day of drinking water containing a unit concentration (1 mg/l) of toxic agent. This unit concentration was chosen to be well above most surface water concentrations of xylene (on the order of $1 \mu\text{g/l}$ (Fishbein, 1985)), yet less than the solubility limit in water (196 mg/l (Stephen and Stephen, 1963)). In turn, this corresponds to a daily dose rate, $d_{\text{unit conc}}$, of 0.036 mg/kg/day for a 55 kg woman. The assumption of zero net growth rate allows one to estimate the number of successful pregnancies per year per 10^5 population as 1,429/year ($10^5 \text{ persons} \times 1 \text{ child/person/70-year lifetime}$). Hence, the incidence rate Q^* may be derived as $A^*(d_{\text{unit conc}})$ from Eq. (B.13) multiplied by the average pregnancy rate of 1,429/year, i.e.,

$$Q^* = 2.75 \times 10^{-4} (0.036)(1429) = 0.015 \quad (\text{B.14})$$

where Q^* is the number of malformations per year per 10^5 people drinking water of 1 mg/l xylene concentration, and there are $2.75 \times 10^{-4} (0.036)$ malformations per pregnancy.

This suggests an upper-bound cumulative incidence of only one malformation over a 70-year period, a low rate for this population size.

TOLUENE

Toxicity Review

A toxicologic review of toluene has been presented by Cohr and Stockholm (1979). A narrower review focusing on neurotoxicity was presented by Benignus (1981). A substantial literature exists dealing with exposure to toluene by inhalation, including detailed uptake and plasma kinetics and acute response data. However, in this brief review, we will focus primarily on oral exposure to this compound.

Absorption of toluene via this route is efficient; nearly 100 percent of the ingested amount eventually is excreted in urine (75 to 80 percent as benzoic acid conjugates) or in expired air (18 percent as unchanged toluene). Absorption from the gut to plasma is aided by the binding of toluene to plasma lipids and lipoproteins. From a whole body viewpoint, the compound accumulates in fatty tissues in accordance with its high oil-to-water partition coefficient, and distribution of toluene to various organ groups is rapid on the time-scale of toxicologic experiments. According to Cohr and Stockholm (1979), there are no data available on the transport of toluene through the placenta.

Metabolism of toluene proceeds primarily by oxidation in the microsomes of the liver parenchymal cells. The greatest fraction of the absorbed compound is oxidized at the methyl group to benzoic acid via benzyl alcohol. In turn, benzoic acid is conjugated with glycine to form hippuric acid and with glucuronic acid to form benzoylglucuronide, and both conjugates are excreted in the urine. A smaller fraction of absorbed toluene proceeds by arylmonooxygenase oxidation to form o- and p-cresols, followed by conjugation to glucuronic acid and sulfate and urinary excretion of these conjugates. This route to phenolic derivatives, although a small fraction of the total mass flux, commands attention because the presence of reactive epoxide intermediates has been demonstrated (Jerina and Daly, 1974; Jerina et al., 1968). Nonlinearities in this metabolic scheme that affect risk estimation may be imagined at high dose levels (such as saturation of microsomal enzyme activity), but direct evidence is lacking.

Toxicity resulting from toluene exposure may be considered with respect to lethality, organ effects, genotoxic effects, and reproductive toxicity. Wolf et al. (1956) reported an oral LD_{50} for toluene of 7.0 g/kg body weight in Wistar rats. The highest LD_{50} was measured among those for benzene, toluene, xylene, ethyl and diethyl benzene, isopropyl benzene, styrene, α -methyl styrene, and vinyl toluene. In repeated dose experiments, these authors again found toluene to be the least toxic of all compounds tested. Kimura et al. (1971) reported a lower LD_{50} of 0.64 g/kg for adult male Sprague Dawley rats, whereas Withey and Hall (1975) reported a value (5.6 g/kg) similar to Wolf et al. (1956).

Of organ systems affected by toluene, the nervous system is most clearly affected (Cohr and Stockholm, 1979). Both central and peripheral nervous systems may exhibit toluene toxicity but these effects are generally observed only after large exposures. The most significant toxicities have been found in the central nervous system where toluene typically acts as a depressant. Under acute dosing conditions, drowsiness, tiredness, headache, nausea, impaired coordination, impaired light accommodation, confusion, and staggering gait have all been observed. Short-term tests (Gamberale and Hultengren, 1972; Olson et al., 1985) in human volunteers at rest have shown that simple reaction time, choice reaction time, and perceptual speed show a significant dose-response relationship. Lasting and possibly irreversible central nervous system toxicity, including disturbances of intellectual, emotional, and neurovegetative functions, may also exist following long exposures to toluene at high doses. However, irreversibility has been difficult to demonstrate in humans because most data are anecdotal and involve co-exposure of subjects to other solvents as well. Furthermore, some animal experiments involving toluene exposure indicate that changes in impaired psychomotor test performance

may not be truly irreversible but rather are indicators of long-term adaptation (Tahti et al., 1983). The possibility of irreversibility is assumed primarily by analogy to the behavior of other solvents (Cohr and Stockholm, 1979).

Hematological toxicity resulting from toluene exposure does not appear to be significant. The early literature reported toxicities similar to those of benzene, but, because of benzene contamination in these early studies, they have not been confirmed in the recent literature. Histochemical changes following exposure to pure toluene have been reported in both humans and rats (e.g., increased number of leukocyte granules) but no direct connection between these observations and clinical toxicity has been made.

Liver toxicity has been investigated primarily by examination of serum chemistry and histological/histochemical observations of liver tissue. Although changes in serum enzymes indicative of hepatotoxicity were reported for rats, significant changes were not reported for humans. Alterations in liver cytology, such as an increased number of mitochondria and accumulation of vacuoles, have been observed following exposure to toluene, but these changes were reversible and quickly disappeared after termination of exposure. Kidney tissue may be damaged by toluene at high doses as evidenced by hematuria, proteinuria, tubular acidosis, and hypokalemia but irreversible toxicity at lower doses remains undemonstrated. Likewise heart tissue may be affected at high doses. Toluene is also suspected of affecting the diencephalon-hypophysis-adrenal cortex complex resulting in irregular menstruation; however the epidemiologic data upon which this suspicion is based are quite limited and coexposure with other solvents may have occurred.

Genotoxic effects of toluene are absent or very slight. Mutagenesis caused by toluene has not been observed in submammalian systems (Cohr and Stockholm, 1979). A significantly increased number of chromosomal breaks in typographers exposed to toluene has been reported as have increased chromosomal aberrations in rats exposed subcutaneously to about 1 g/kg/day for 12 days. However, toxicities resulting from associated phenotypic changes have not been demonstrated, and these observations are important only in their qualitative similarity to benzene effects at lower doses. Carcinogenicity resulting from toluene has not yet been shown, either in skin application experiments or in 18-month inhalation experiments with maximum concentrations of 300 ppm (EPA, 1980a).

Prenatal toluene apparently has an effect on offspring (Benignus, 1981). Hudak and Ungvary (1978) reported that CFY rats, exposed via inhalation to toluene at 1500 mg/m³ on days 1-8 of gestation, exhibited reduced fetal weight. They also reported significantly increased skeletal retardation ($P < 0.05$) but no malformations. Nawrot and Staples (1979) reported that CD-1 mice, exposed by gavage to 783 to 2,610 mg/kg/day of toluene, exhibited increased embryonic lethality at all dose levels and significant fetal weight reduction at the two higher dose levels. Furthermore, a statistically significant increase in cleft palate was observed at the highest dose level.

Rate of Toxicity Induction by Toluene Ingestion

To characterize the response of a human population exposed to toluene via ingestion, we consider two endpoints. The first is the no-effect endpoint, because EPA based its 1980 water quality criterion for toluene on the maximum-no-effect level observed in the subchronic six month toxicity experiments of Wolf et al. (1956). These experiments sought to observe changes in growth, mortality, appearance, and behavior, hematology, organ weights, histopathology, and bone marrow counts in female rats. The second endpoint is the teratogenic outcome of cleft palate (Nawrot and Staples, 1979), chosen because it is a positive observation and because it presently appears to be the most likely irreversible outcome.

In the Wolf et al. experiment, Wistar female rats were dosed by stomach tube with toluene in olive oil solution five days a week for six months at dose rates ranging from 0 to 590 mg/kg/day. No toxic effect was observed by these investigators at any dose level. The number of animals in each dosage group was reported (Table B.2), but the proportions of responders in the control and dosage groups were not. However, by analogy to other chronic and subchronic rat and mouse studies (Schroeder and Mitchener, 1975; Reeves et al., 1967; Schopp et al., 1984), it is reasonable to assume that mortality in the control population of 20 females was absent at six months (assuming no infection or cannibalism in the group) and that the proportional response was 0/20.

From this control group response and the number of animals tested, we may estimate the maximum proportion of animals at a particular dose that have responded (died), yet shown no statistically significant difference from controls. We estimated this maximum response for the highest dose level relative to control (zero dose) using Fisher's Exact Method for the comparison of two binomial proportions (Ostle, 1963).

The largest proportional response consistent with a significance level of 0.05 was 0.3085. The 0.05 significance level was used by Wolf et al. (1956) in assessing significance of other measures in their research. Hence if the one-hit model is again used to describe toxic response,

$$P(d) = 1 - \exp[(-q_0 - q_1 d)t] \quad (\text{B.15})$$

where time dependence of the response has been shown as the fractional lifetime t . The largest proportional response (0.3085) at an average maximum dose of 422 mg/kg/day is

$$P(d_{\max}) = 0.3085 = 1 - \exp(-q_{1,\text{rat}} d_{\max} t) \quad (\text{B.16})$$

The value of q_0 in Eq. (B.15) has been taken as zero so that all uncertainty of response resides in q_1 and t is 193 days/730 days (lifetime of rat) = 0.264. From Eq. (B.16),

$$q_{1,\text{rat}} = 0.0033 (\text{mg/kg/day})^{-1}$$

Table B.2

NUMBER OF FEMALE WISTAR RATS TESTED
FOR RESPONSE TO TOLUENE

Dose ^a (mg/kg/day)	Number of Animals ^b
422	10
253	10
84	10
0	20

SOURCE: (Wolf et al., 1956)

^aDose tabulated is the actual daily dose rate (range of 0 to 590 mg/kg/day) averaged over an administration schedule of five out of seven days. Total exposure period was 193 days.

^bNo effect over several toxic endpoints (see text), including mortality, was reported for toluene-exposed animals.

Scaling this rat parameter to its human counterpart, we again invoke the scaling rule of Eq. (B.11) with mouse reference replaced by rat. Using a standard human male weight of 70 kg and a rat weight of 0.2 kg,

$$q_{1,\text{human}}^* = 0.023 \text{ (mg/kg/day)}^{-1}$$

The excess lifetime toxic risk per person at some dose d is given by Eq. (B.13), $A^*(d) = q_{1,\text{human}}^* d$. For comparability to other compounds, we again estimate the upper-limit toxic-response incidence rate R^* for a population of 10^5 people continually exposed to toluene by drinking 2 l/day of water containing a unit concentration of 1 mg/l toluene:

$$\begin{aligned} R^* &= A^*(d_{\text{unit conc}})(10^5/70) \\ &= q_{1,\text{human}}^* d_{\text{unit conc}} (10^5/70) = 0.95 \end{aligned} \quad (\text{B.17})$$

where R^* is the number of deaths per year in a population of 10^5 people. Here $d_{\text{unit conc}} = 1 \text{ mg/l} \times 2 \text{ l/day}/70 \text{ kg body weight} = 0.29 \text{ mg/kg/day}$, and the factor of 70 is the average human life span in years. The unit concentration of 1 mg/l is much higher than that found in most surface waters (from 1 to $10 \mu\text{g/l}$ (EPA, 1980a)), yet less than its solubility limit in water (500 mg/l (Stephen and Stephen, 1963)).

This response rate is relatively high but may be more a reflection of experimental uncertainty than actual risk of mortality. The Wolf et al. experiments did not observe any toxic response and sample sizes were small, two experimental factors which greatly expand the confidence limits about mean estimated response. Overestimation of toxic response may also be a consequence of using a linear dose-response model; however the lack of positive observed responses does not allow the parameterization of nonlinear models.

Reproductive Toxicity

In the experiment of Nawrot and Staples (1979), CD-1 mice were dosed by gavage with toluene in cottonseed oil over days 6 to 15 of gestation at three dose levels. The incidence of cleft palate (in excess of that caused by general retardation in growth rate) was recorded by litter and by fetus (Table B.3). Because the cleft palating was spread over several litters (the 10 at the highest dose occurred in five litters), little bias in litter occurrence was indicated. As a result, we can perform all calculations using the number of fetuses as the analytical unit.

As with xylene teratogenicity, we analyze the cumulative incidence of cleft palate using the one-hit model combined with a weighted linear least squares technique. The approach of Eqs. (B.1) through (B.9) is again followed to derive estimates for the one-hit model parameters q_0 and q_1 ; the only exception is that the zero response proportion (0/265) of the 783 mg/kg/day dose necessitates that its variance be estimated from exact confidence limits of zero proportions (Ostle, 1963).

The results of these computations yield $q_0 = 1.57 \times 10^{-3}$, $q_1 = 7.17 \times 10^{-6} \text{ (mg/kg/day)}^{-1}$, $\sigma_{q_0} = [\text{var}(q_0)]^{1/2} = 1.35 \times 10^{-3}$, and $\sigma_{q_1} = [\text{var}(q_1)]^{1/2} = 2.83 \times 10^{-6} \text{ (mg/kg/day)}^{-1}$. The 95 percent upper confidence value for q_1 , applicable to the mouse, is

$$q_{1,\text{mouse}}^* = q_1 + 1.96\sigma_{q_1} = 1.27 \times 10^{-5} \text{ (mg/kg/day)}^{-1}$$

Applying the same scaling rule from Eq. (B.11) and using the same pregnancy body weights as employed in the xylene calculation,

Table B.3
RESPONSE OF CD-1 MICE TO ORALLY
ADMINISTERED TOLUENE

Dose (mg/kg/day)	Response ^a	
	Malformed	Live
2,610	10	274
1,305	2	290
783	0	265
0	2	1,037

^aNumber of fetuses—fetal data supplied by Dr. R.E. Staples.

$$q_{1,\text{human}}^* = 1.56 \times 10^{-4} (\text{mg} / \text{kg} / \text{day})^{-1} \quad (\text{B.18})$$

This value may now be used to compute a Q^* for toluene teratogenicity, the upper 95 percent confidence estimate of the incidence rate of malfunctions expected in a human population of 10^5 reproducing at zero net growth rate and consuming 2 l/day of water containing 1 mg/l of toluene. As derived for xylene above in Eq. (B.14),

$$Q^* = (q_{1,\text{human}}^* d_{\text{unit conc}})(1429) = 0.0081$$

This incidence rate is small, like that calculated for xylene.

NAPHTHALENE

Toxicity Review

Toxicity data for this compound are few. In preparing its 1980 water quality criteria document for naphthalene, EPA stated that there were no chronic animal toxicity studies nor epidemiologic studies adequate to serve as a basis for criterion setting (EPA, 1980b). Since that time a new study has appeared characterizing the general toxicology and immune toxicology of naphthalene in mice following acute and subchronic (90 day) exposures (Schopp et al., 1984). Other studies have appeared investigating the naphthalene-induced cytotoxicity observed in mouse pulmonary bronchiolar epithelium (Griffin et al., 1981; Warren et al., 1982; Buckpitt et al., 1984; O'Brien et al., 1985).

The older naphthalene literature recorded two principal responses of human subjects to large naphthalene exposures. The first major manifestation was hemolytic anemia with associated jaundice and the second was cataract formation (Schopp et al., 1984). Hemolytic anemia was observed after massive ingestion of moth balls (Zeulzer and Apt, 1949), or exposure of newborns to clothing heavily impregnated with naphthalene (Dawson et al., 1958). The susceptibility to naphthalene-induced hemolytic anemia appears to be associated with a glucose-6-phosphate dehydrogenase (G6PD) deficiency. Newborns represent a special group in that some are sensitive in spite of no G6PD deficiency (Zinkham and Childs, 1958). Although sufficient data do not exist to fully describe hemolytic response with dose, we do know that the degree of hemolytic anemia in an individual rapidly drops off with dose as red cell glutathione becomes available for detoxification. Cataract formation probably results from a catechol-

reductase-catalyzed formation of 1,2-dihydroxynaphthalene which, in turn initiates a reaction chain leading to cataracts (Van Heyningen, 1979).

Like hemolytic anemia, insufficient data exist to model this continuous response with dose. Human and rat data (e.g., Fitzhugh and Buschke, 1949) both suffer from a nonquantitative scoring of cataract development and use of very limited dosage levels (often just one). Rabbit data are more extensive (Van Heyningen and Pirie, 1976) but still suffer from nonquantitative scoring of cataract. Furthermore, not all strains of the same species are equally sensitive to naphthalene-induced cataracts, and, without further data, it is not clear which best models human response.

Recent literature (Reid et al., 1973; Mahvi et al., 1977; Warren et al., 1982, Buckpitt et al., 1984) has identified and examined the specific necrosis of noniliated bronchiolar epithelial cells (Clara cells) and bronchial epithelial cells by naphthalene. Evidence was found that Clara cells, possessing substantial levels of cytochrome P-450 activity, oxidize naphthalene to reactive intermediates that bind to macromolecules. This binding or perhaps associated redox reactions (Buckpitt et al., 1984) are thought to lead to cell death and sloughing of the epithelial cells. Warren et al. (1982) showed that extensive necrosis occurred in mice near the LD_{50} dose, with large numbers of exfoliated epithelial cells observed in the bronchiolar lumen. These authors also determined the relation between dose and level of covalent binding of reactive naphthalene metabolites to lung tissue.

Unfortunately it is not possible at present to pass from this biochemical dose-response relationship to a quantitatively meaningful dose-clinical response relationship; covalent binding needs to be correlated with a clinical endpoint such as death or some level of impaired respiration. In addition, it is not clear that destruction of bronchiolar epithelium is the most appropriate endpoint for use in human risk estimation since it is known that significant species sensitivity to pulmonary toxicity exists (O'Brien et al., 1985). It is relatively clear, however, that if pulmonary epithelial necrosis were used as an endpoint, the dose-response relationship would be highly nonlinear; substantial binding of naphthalene metabolites to lung macromolecules occurs only after depletion of the detoxification agent, glutathione.

Naphthalene has not been identified as a mutagen (McCann et al., 1975) or as a complete carcinogen (EPA, 1980b). Naphthalene carcinogenicity reported by Knake (1956) was rejected by EPA (1980b) on the grounds that the naphthalene used contained 10 percent coal-tar-derived impurities and that co-exposure to known carcinogens occurred in all experiments.

Rate of Toxicity Induction by Naphthalene Ingestion

As was true at the time of formulation of the naphthalene water quality criterion, good data bases do not exist for organ-specific toxicology. However, subchronic general toxicity results have become available for oral administration of agent (Schopp et al., 1984), and we will employ these to crudely characterize the human population response to naphthalene ingestion.

In the Schopp et al. (1984) experiments, CD-1 mice were dosed for 90 days by oral gavage with naphthalene in a corn oil vehicle. Table B.4 summarizes the mortality results for female mice. Results for male mice were similar except that survival ratios were more strongly affected by aggression in this group. The males, unlike the females, did not exhibit any significant changes in organ weights. These mice neither exhibited hemolytic anemia nor cataract formation under the dosage conditions employed. Cataract formation may not occur because these mice are albino; albino rats are resistant to this lesion, whereas rats with pigmented eyes are susceptible. Furthermore, gross examination of the lungs revealed no evidence of necrosis

of the bronchiolar epithelium as might have been expected from the observations of Warren et al. (1982) and Buckpitt et al. (1984). Hence the ultimate causes of death in the Schopp et al. study remain undetermined.

Cursory examination of the response data in Table B.4 shows that no trend with dose emerged during the 90 days study even though doses were as high as one-quarter of the acute male LD_{50} . Hence a response model providing a good fit to the data of Table B.4 would be expected to yield no dose-dependence in the mean; however its upper confidence limit will provide an upper bound to expected toxic response.

The naphthalene data have been fit with the one-hit model given in Eq. (B.15). This equation is identical to Eq. (B.1) except for the exposure time t ($= 90$ days/730 days). Hence if the q parameters of Eq. (B.1) are redefined to include t , the weighted least squares analysis of Eqs. (B.1) through (B.9) can again be employed to fit data. The results of this computation are

$$q_0 = [Eq. (B.1)]/t = 0.172,$$

$$q_1 = q_1[Eq. (B.1)]/t = -7.90 \times 10^{-5} (\text{mg/kg/day})^{-1},$$

$$\sigma_{q_0} = [\text{var}(q_0)]^{1/2} = 9.9 \times 10^{-2}, \text{ and}$$

$$\sigma_{q_1} = [\text{var}(q_1)]^{1/2} = 1.27 \times 10^{-3} (\text{mg/kg/day})^{-1}.$$

The best mean q_1 is a negative number but is so small as to be negligibly different from zero. Note that the standard deviation σ_{q_1} is much larger than the mean. Such a low value of q_1 merely reflects the absence of a dose trend in the raw data. The upper confidence limit on this mouse q_1 is

$$q_{1,\text{mouse}}^* = q_1 + 1.96 \sigma_{q_1} = 0.0025(\text{mg/kg/day})^{-1}$$

and scaling this to the human using Eq. (B.11) and human and mouse weights of 70 kg and 0.029 kg gives

$$q_{1,\text{human}}^* = 0.033(\text{mg/kg/day})^{-1}$$

Table B.4

SUBCHRONIC RESPONSE OF FEMALE CD-1 MICE
TO ORALLY ADMINISTERED NAPHTHALENE

Dose (mg/kg/day)	Response ^a	
	Dead	Total
133	1	60
53	2	40
5.3	3	40
0	1	76

^aMortality of female mice following exposure for 90 days.

As used in the previous toluene calculations, we can compute R^* , the upper-limit toxic-response incidence rate for a population of 10^5 people continually exposed to naphthalene by drinking 2 l/day of water containing a unit concentration of 1 mg/l naphthalene:

$$\begin{aligned} R^* &= A^*(d_{\text{unit conc}}) \times 10^5 / 70 \\ &= q_{1,\text{human}}^* d_{\text{unit conc}} 10^5 / 70 = 1.4 \end{aligned}$$

where R^* = number of deaths per 10^5 people per year,
 $d_{\text{unit conc}} = 0.029 \text{ mg/kg/day}$.

The average human lifetime is again 70 years. The 1 mg/l concentration is far above observed surface water concentrations of $1.4 \mu\text{g/l}$ (EPA, 1980b) but below the solubility of naphthalene in water (12.5 mg/l) (Stephen and Stephen, 1963).

The response rate is relatively high, but like the result for toluene, may be more a reflection of experimental uncertainty than actual risk of mortality. Because no positive trend was observed in the experimental data, the actual response rate could be substantially below this estimated upper confidence limit.

BENZO(A)PYRENE

Toxicity Review

Benzo(a)pyrene (BP) has been the subject of intense cancer research since it was observed to be the major carcinogenic component of coal tar (Cook et al., 1933). A review of the 50 years of research leading to the conclusion that the ultimate carcinogen formed from BP is (+)-anti-benzo(a)pyrene-7,8-diol-9,10-epoxide was recently presented by Phillips (1983). A synopsis of the human and animal toxicology of BP is found in the IARC evaluation of carcinogenic risk of chemicals to humans (IARC, 1983) and in review by DePierre and Ernster (1978) and Pelkonen and Nebert (1982). We therefore provide only a brief overview of the metabolism and toxicity of this compound, leaving the description of details to the references contained within the review just cited.

Early work (Pullman and Pullman, 1955) made the presumption that parent BP was biologically active and that metabolism (to about 20 compounds plus conjugates) only served to detoxify the compound. However, it is now known that the opposite is true, that the cell's attempt to detoxify the compound leads to the production of biologically active electrophilic species (Miller, 1970). The principal pathway for metabolic activation of BP was shown to involve oxidation of BP to (+)-BP-7,8-oxide by mono-oxygenase, followed by epoxide hydrolase-catalyzed hydrolysis to (-)-trans-BP-7,8-dihydrodiol, and further oxidation by mono-oxygenase to (+)-anti-BP-7,8-diol-9,10-epoxide (Simms et al., 1974). This diol-epoxide is highly reactive and capable of forming adducts with the guanine residues in DNA. At present, it is believed that this DNA-bound diol-epoxide is responsible for causing mutations or initiating a carcinogenic one, perhaps by base mispairing or frameshift induction (Phillips, 1983). However, adducts with RNA or proteins cannot absolutely be ruled out as causative (Pelkonen and Nebert, 1982). Overall, the picture emerges that in the body's attempt to oxidize BP to readily cleared compounds, a compound is produced along one path that is highly toxic because it is a poor substrate for the hydrolase enzyme that would complete its detoxification.

Benzo(a)pyrene is a known carcinogen to experimental animals (IARC, 1983) and, as part of the polycyclic hydrocarbon complex, involved in the development of occupational cancers involving exposure to coal tar. It is a strong carcinogen at the site of administration, inducing its own metabolism there and leading to epidermal carcinomas following skin painting, fibrosarcomas following subcutaneous injection, squamous cell carcinomas following ingestion and various pulmonary tumors following inhalation. BP may also induce tumors at locations distant to the site of administration. Leukemia and lymphatic tumors, for example, may be induced after oral or subcutaneous administration of agent (Pelkonen and Nebert, 1982).

Mutagenicity of BP has been amply demonstrated in a variety of test systems, and, in fact, is often used as a positive control. It induced lesions in bacterial DNA leading to repair; it was active in assays indicating bacteriophage induction, bacterial and *Drosophila* mutation, and sister chromatid exchange, chromosomal aberration, and point mutation in cultured mammalian cells. It was also shown to produce these same mammalian cell results *in vivo*, and, in addition, sperm abnormality and positive specific locus tests (IARC, 1983).

Benzo(a)pyrene is embryotoxic and teratogenic to mice (IARC, 1983). If the mother has an easily induced cytochrome P-450 mono-oxygenase system, active BP metabolites will form and cross the placenta, and may lead either to fetal death or malformation. If the mother lacks the inducible genotype but the fetus possesses it, embryotoxicity and teratogenicity may still result.

Other reproductive toxicities have been noted (IARC, 1983). Following very high oral doses of BP to pregnant mice (40 mg/kg body weight/day), the offspring of both sexes exhibited almost complete sterility. Application of BP to the skin of pregnant mice over four generations resulted in sensitization of the offspring to BP, leading to increased rates of papilloma and carcinoma induction in these animals over control rates. Finally in both mice and rabbits, transplacental carcinogenesis has been demonstrated.

Rate of Toxicity Induction by Benzo(a)pyrene Ingestion

To characterize the response of a population exposed to BP by ingestion, we draw on the same data as used by EPA (1980c) in establishing the water quality criterion (Neal and Rigdon, 1967). These data are superior to others because the route of administration was oral (feeding *ad libitum*), rather than the much more common dermal route, and because relatively large numbers of animals were used in the experiment.

The Neal and Rigdon data are shown in Table B.5. In these experiments, male and female CFW mice were orally dosed with benzo(a)pyrene mixed into a laboratory feed for a period of 110 days and then immediately sacrificed for examination. Response was scored as the cumulative incidence of gastric tumors.

These data were analyzed by EPA using the multistage model as described in EPA (1980c). The upper three doses were excluded from their analysis because of lack of fit to the model. The low dose limit of this multistage model for lifetime exposure is the one-hit result shown previously in Eq (B.1). EPA (1980c) reported a 95 percent upper confidence limit for the human q_1 of

$$q_{1,\text{human}}^* = 11.5(\text{mg} / \text{kg} / \text{day})^{-1}$$

For comparison with other compounds, we use this value to again estimate R^* , the upper limit toxic-response incidence rate for 10^5 people exposed to benzo(a)pyrene by drinking 2 l/day of water containing a unit concentration, 1 mg/l, of compound. As before,

Table B.5
RESPONSE OF CFW MICE TO ORALLY
ADMINISTERED BENZO(A)PYRENE

Dose (mg/kg/day)	Response ^a	
	Carcinoma	Total
0	0	289
0.13	0	25
1.3	0	24
2.6	1	23
3.9	0	37
5.2	1	40
5.85	4	40
6.5	24	34
13.0	19	23
32.5	66	73

^aCumulative incidence of papillomas and squamous cell carcinomas over a 110 day exposure period. CFW mouse lifetime was taken as 630 days.

$$\begin{aligned}
 R^* &= A^*(d_{\text{unit conc}})10^5/70 \\
 &= q_{1,\text{human}}^* d_{\text{unit conc}} 10^5/70 \\
 &= 490
 \end{aligned}$$

where R^* = number of gastric tumor cases per 10^5 people per year.

This R^* value is much larger than the corresponding values obtained for toluene and naphthalene, reflecting its much greater cancer toxicity. However, these 490 toxic cases must be considered only a number for comparison of toxic potencies with other compounds. The unit concentration of 1 mg/l apparently exceeds the solubility limit of BP in water. If this limit is estimated from the solubilities of pyrene (0.18 mg/l) and fluoranthene (0.26 mg/l) as about 0.2 mg/l, then the human dose rate at BP saturation in water becomes

$$d = 2 \text{ l/day} \times 0.2 \text{ mg/l} \times 1/70 \text{ kg} = 0.0057 \text{ mg/kg/day}$$

and R^* becomes

$$R^* = q_{1,\text{human}}^* d (10^5/70) = 92$$

This is a very high toxic response incidence. Unlike the estimated incidences for toluene and naphthalene, this R^* is not greatly overestimated because of the absence of positive responses in the observed data. However, it is subject to over- or underestimation because we cannot account for saturation of detoxification and actuation pathways, as are all of our other R^* and Q^* estimates.

BENZENE

Toxicity Review

Benzene metabolism was reviewed by Snyder et al. (1981). Although a number of metabolic pathways have been identified in animal studies, the data on pharmacokinetics are not sufficient to model metabolism in animals (Anderson et al., 1980). Pharmacokinetic models are not available to model human metabolism or distribution. There is no conclusive evidence that the carcinogen benzene oxide—a possible metabolic intermediate of benzene—exists (Goldstein, 1983).

Mutagenicity and Chromosomal Effects. According to the IARC, benzene is not mutagenic in bacterial systems (IARC, 1982). In mammalian cells, the mutagenicity of benzene has generally been negative as well (IARC, 1982). Benzene did not appear to induce enhancement in the frequency of sister chromatid exchange in cultures (IARC, 1982). The chemical has shown significant increases in micronuclei in five studies (Lyon, 1976; Diaz et al., 1980; Hite et al., 1980; Meyne and Legator, 1980; and Erexson et al., 1984). Benzene does induce chromosomal aberrations in bone marrow cells from mice, rats, and rabbits (IARC, 1982; CDHS, 1984). Benzene also causes DNA damage in mammalian cells in culture and chromosomal aberrations in workers (CDHS, 1984).

Carcinogenicity. Benzene is carcinogenic in both animals and human beings. In what follows, we summarize the relevant studies.

Tables B.6 through B.8 summarize the effects of benzene from animal studies taken from CDHS (1984).

Table B.6 shows the available data through 1976. These data, according to the IARC (1974) were not sufficient to demonstrate animal carcinogenicity. By 1982, the IARC (1982) had concluded that there was limited evidence of carcinogenicity because of additional studies.

The results of a gavage study are presented in Table B.7 as Experiment 1 (Maltoni, 1983). Mortality was higher in the benzene treated group and was correlated to dosage. Table B.7 also shows the 92 week interim results of a second gavage study, Experiment 3. There was an increase in zymbal gland tumors¹ in the benzene administered animals. In Experiment 2, an inhalation study, there were increased zymbal gland carcinomas, leukemias, oral cavity carcinomas, and mammary gland carcinomas but only the zymbal gland carcinomas, the leukemias, and oral cavity carcinomas showed statistical significance according to the Cochran-Armitage linear trend test.

The results of a NTP rat gavage study are summarized in Table B.8. There was a statistically significant increase in the neoplasm incidence at multiple sites. It is worth noting here that zymbal and preputial gland carcinomas² are rare in mice.

There is ample evidence that benzene causes leukemia in humans (Goldstein, 1977; Goldstein and Snyder, 1982; van Raalte and Grasso, 1982; IARC, 1982; Maltoni, 1983; EPA, 1984). The 23 principal epidemiological studies on benzene are summarized in Table B.9. In 1984, the IARC concluded that the epidemiological studies demonstrate that benzene is carcinogenic to humans (IARC, 1982).

One large study not shown in Table B.9 is by the Environmental Health Associates (1983). It was a historical perspective investigation of 4,602 male chemical workers who worked with benzene for six months or more over the period 1946 to 1975. There was a statistically significant dose-response relationship between cumulative exposure to benzene and mortality from leukemia and from all lymphatic/hematopoietic cancer combined (CDHS, 1984).

¹Zymbal glands are large modified sebaceous glands in rats surrounding the external ear canal. Humans have no counterpart.

²Preputial glands lie beneath the skin of the prepuce and open into its cavity in the male; in the female, they are located in the prepuce of the clitoris.

Table B.6
LONG-TERM CARCINOGENICITY BIOASSAYS ON BENZENE: AVAILABLE DATA UP TO THE MID-1970S

Authors	Animals			Treatment and Other Experimental Details		Results	Observations
	Species	Strain	Sex	No. ^a			
Lignac (1932)	Mice	"Albino"	M,F	33 T	Subcutaneous injection of 0.001 ml of benzene in 0.1 ml of olive oil, once weekly for 17-21 weeks (total dose, about 1 mg/kg body weight)	8 leukemias (from 4 to 8 months from the start of treatment)	No control group
Kirschbaum and Strong (1942)	Mice	F	?	30 T	Subcutaneous injection of 0.001 ml of benzene in 0.1 ml of sesame oil	6 leukemias (30%) (from 200 to 300 days of age) 29 leukemias (14%) (before 300 days of age)	Leukemia increase in treated animals is not statistically significant
Amiel (1960)	Mice	DBA2 C3H C57BL6	M	30 T 30 T 30 T	Subcutaneous injection of 0.001 ml of benzene in 0.1 ml of olive oil, for the whole life span	0 0 0	Maximum survival of DBA2, C3H, and C57BL6 mice: 730 days
		AKR	M	30 T		16 leukemias (between the 7th and the 16th month of treatment); 8 animals died before the 9th month of treatment	Major survival of animals of control group
Hiraki et al. (1963)	Mice	Swiss	M,F	35 C 10 T	Subcutaneous injection of 0.001 ml of benzene in 0.1 ml of olive oil, for 10 weeks	30 leukemias 5 subcutaneous sarcomas (at autopsy, performed between the 162nd and the 253rd day from start of treatment)	2 animals died within the first 8 weeks of treatment no control group
Many	Mice Rats Rabbits	Various	M,F A	great many	Skin applications	No effects	Nonsystematic and non ad hoc planned experiments

SOURCES: Malkoni et al. (1983), CDHS (1984), and Goldstein (1977).
^aT = treated; C = control.

Table B.7

MALTONI ET AL. BENZENE RAT BIOASSAY SUMMARY

Organ (Site)	Tumor Type	Route of Exposure	Sex	Dosage	Cochran-Armitage Linear Trend Test	Fisher Exact Test	Difference in Cancer Attack Rate per 100 (Dose - Control)
Zymbal gland	Carcinomas	Gavage ^a	F	13.9 mg/kg-day 66.7 mg/kg-day	P < 0.001 —	P = 0.25 (NS) P = 0.003	6.7 25.0
Hemolympho-reticular	"Leukemias"	Gavage ^a	M	13.9 mg/kg-day 66.7 mg/kg-day	— —	P = 1.0 (NS) P = 0.078	(NS) 12.1
Mammary	Carcinoma	Gavage ^a	F	13.9 mg/kg-day 66.7 mg/kg-day	P = 0.091 (NS) —	P = 0.50 (NS) P = 0.178 (NS)	3.3 11.9
Zymbal gland	Carcinomas	Gavage ^b	F	321.4 mg/kg-day	N/A (single dose level)	P = 0.007	15.0
Zymbal gland	Carcinomas	Gavage ^b	M, F	321.4 mg/kg-day	N/A (single dose level)	P < 0.001	14.0
Oral cavity	Carcinomas	Gavage ^b	M	321.4 mg/kg-day	N/A (single dose level)	P = 0.003	17.5
Oral cavity	Carcinomas	Gavage ^b	M, F	321.4 mg/kg-day	N/A (single dose level)	P < 0.001	13.7
Zymbal gland	Carcinoma	Inhalation ^c	F	17 mg/kg-day	N/A (single dose level)	P = 0.27 (NS)	3.9
Zymbal gland	Carcinoma	Inhalation ^d	M, F	16.4 mg/kg-day	N/A (single dose level)	P = 0.002	5.1
Liver	Hepatomas	Inhalation ^e	F	1.42 mg/kg-day	N/A (single dose level)	P = 0.022	5.1

SOURCE: CDHS (1984).

NOTES: NS = Not Statistically Significant, $p > 0.05$; N/A = Not Applicable.^aExperiment 1: (#BT901), animals dosed for 52 weeks.^bExperiment 3: (#BT902), 92 week interim results, dosing to be carried out for 104 weeks, 118 week interim results.^cExperiment 2: (#BT4004), inhalation exposure of 13 week old breeder rats for 104 weeks, 118 week interim results.^dExperiment 3: (#BT4004), inhalation exposure of 12 day old embryos for 104 weeks. Dose in utero not considered, 118 week interim results.^eExperiment 3: (#BT4006), inhalation exposure of 12 day old embryos for 15-weeks. Dose in utero not considered, 118 week interim results.

Table B.8

SUMMARY OF NTP BIOASSAY FOR SIGNIFICANT NEOPLASMS

Organ (Site)	Tumor Type	Species	Sex	Cochran-Armitage Trend Test	Fisher Exact Test	Difference in Cancer Attack Rate per 100 (Dose - Control)
Zymbal gland	Squamous cell carcinoma	Rat	M	P < 0.001	P < 0.001	30
		Rat	F	P < 0.001	P < 0.001	28
		Mouse	M	P < 0.001	P < 0.001	43
		Mouse	F	P = 0.022	P = 0.121 (NS)	6
Skin	Squamous cell carcinoma	Rat	M	P = 0.007	P = 0.003	16
		Mouse	M	P = 0.028	P = 0.121 (NS)	6
Lip	Squamous cell carcinoma	Rat	M	P = 0.012	P = 0.003	16
Tongue	Squamous cell carcinoma	Rat	M	P = 0.078	P = 0.059	8
		Rat	F	P = 0.078	P = 0.059	8
Oral cavity	Squamous cell carcinoma	Rat	M	P = 0.006	P = 0.006	14
		Rat	F	P = 0.011	P = 0.028	10
Hematopoietic	Malignant lymphomas or leukemia	Mouse	M	P = 0.006	P = 0.005	23
Harderian gland	Carcinoma	Mouse	F	P = 0.004	P = 0.059	8
Lung	Alveolar/bronchiolar carcinoma	Mouse	M	P = 0.028	P = 0.020	19
		Mouse	F	P = 0.021	P = 0.013	12
Preputial gland	All carcinomas	Mouse	M	P < 0.001	P < 0.001	63
Mammary gland	Carcinomas	Mouse	F	P < 0.001	P < 0.001	20
	Carcinosarcoma	Mouse	F	P = 0.006	P = 0.059 (NS)	8
Harderian gland	Adenoma or carcinoma	Mouse	M	P = 0.001	P < 0.001	27
	Carcinoma	Mouse	F	P = 0.004	P = 0.059 (NS)	8
Ovary	Granulosa cell tumor or carcinoma	Mouse	F	P = 0.003	P = 0.017	15

SOURCE: CDHS (1984).

NOTE: Fisher Exact Test - comparison of highest dose group with control, one-tailed test. Mice of both sexes and female rats dosed at 71.4 mg/kg/day, male rats dosed at 143 mg/kg/day.

Table B.9
EPIDEMIOLOGIC STUDIES OF CARCINOGENICITY IN HUMANS

Year	Author	Population Studied	Duration	Results
1974	Tabershaw Cooper Assoc.	Petrol industry workers		Increase in rate of lymphomas but not statistically significant (NS)
1974	Thorpe	Petrol industry workers	1962-1972	Leukemia SMR = 121 (NS) (SMR in worker controls = 60)
1974 1977	Aksoy et al.	Shoemakers exposed to 210-650 ppm benzene	1967-1975	Annualized crude rate of acute leukemia 2-fold greater than expected
1975	McMichael et al.	Rubber industry workers		Excesses in mortality from: chronic lymphatic leukemia myelogenous leukemia lymphosarcoma
1976	Vigliani	Patients with benzene hemopathy. Exposures estimated at 200-500 ppm	1942-1975 1959-1974	Leukemia incidence: 66 13/135 Estimated relative risk (RR) = 20
1977	Infante et al.	748 rubber industry workers followed to 7/75		1940-1949 7 cases of myeloid/monocytic leukemia SMR = 506 (U.S. white males) SMR = 474 (worker controls)
1978	Ott et al.	Benzene workers (DOW); 594 workers followed to 1973	1938-1970	2 deaths from anemia: one pernicious one aplastic 3 deaths from leukemia Mortality results NS but leukemia rates exceed TNCS expectation ($P < .05$)
1978	Fishbeck et al.	10 chemical workers exposed to benzene	1953-1963	Changes in blood but "... no persisting significant adverse health effects."
1978	Brandt et al.	Case-control study of 50 acute nonlymphocytic leukemia	1969-1977	History of exposure to petroleum products among cases
1979	Vianna & Polan	Workers in New York State exposed to benzene		RR = 2.1 lymphosarcoma RR = 1.6 reticulum cell sarcoma RR = 1.6 Hodgkin's Disease For workers older than 45, the observed number of cases was statistically significant (SS) greater than the expected number
1979	Greene et al.	U.S. Government Printing Office workers		Significantly higher proportion of deaths from multiple myeloma, leukemia, and Hodgkin's disease and related to exposure to benzene
1980	Linos et al.	Case-control study of 138 leukemias		4 cases found, 3 were chronic lymphocytic leukemia RR = 3.3 (NS)
1981	Flodin et al.	Case-control study of 42 acute myeloid leukemia		Six-fold (SS) increase in risk related to exposure to solvents including petroleum products
1981	Schottenfeld et al.	Worker cancer registry compiled by American Petrol Institute		Incidence of lymphocytic leukemia and multiple myeloma increased (SS)
1981	Rushton and Alderson	Petrol refinery workers		Risk of leukemia increased 2-fold in high and medium benzene exposed vs. low exposed ($P = 0.05$)
1981	Rinsky et al.	Continuation of follow-up of Infante study		SMR = 560 leukemia SMR = 2100 leukemia in workers with 5 or more years exposure

Table B.9 (Cont.)

Year	Author	Population Studied	Duration	Results
1982	Thomas et al.	Refinery workers		PMRs for multiple myeloma and other lymphomas elevated (SS)
1982	Hanis et al.	Refinery and chemical workers		SMR for cancer of the lymphopoietic tissues elevated but NS
1983	Decoufle et al.	Chemical plant workers 259 men followed through 1977	1960-1974	SMR-377 (SS) lymphocytic and hematopoietic cancer (L&H) SMR = 682 (SS) leukemia
1983	Tsai et al.	454 refinery workers		No deaths observed from L&H cancer; 0.42 expected (NS)
1983	Arp et al.	Rubber industry workers		For lymphocytic leukemia: RR = 4.5 (NS) benzene exposure RR = 4.5 (NS) other solvent exposures
1983	Wong et al.	Chemical workers	1946-1975	SMRs elevated (but NS) for L&H cancer leukemia, non-Hodgkin's lymphoma. RR = 3.2 (SS) L&H cancer (vs. worker control) Dose-response trend found SMRs for lung cancer and several other cancers elevated (NS)

SOURCE: CDHS (1984).

Rate of Toxicity Induction by Benzene

The data we choose are from the Rinsky et al. reevaluation of the Infante et al. (1977) epidemiologic study (CDHS, 1984; EPA, 1984). From this work we find that

$$q_{1,\text{human}}^* = 5.2 \times 10^{-2} (\text{mg/kg/day})^{-1}$$

For ingestion, we again assume a unit concentration of 1 mg/l, a consumption level of 2 l/day, and a standard human male weight of 70 kg. From the dose rate and $q_{1,\text{human}}^*$, we may estimate the upper-limit leukemic incidence rate, R^* , for a population of 10^5 people continually exposed to benzene as,

$$\begin{aligned} R^* &= A^*(d_{\text{unit conc}}) \times 10^5 / 70 \\ &= q_{1,\text{human}}^* d_{\text{unit conc}} 10^5 / 70 = 2.13 \end{aligned}$$

where R^* = number of leukemias per 10^5 people per year.

This response rate is relatively high. Because this is based on human epidemiologic response, it represents an actual risk of mortality. This is in contrast to the case of toluene where such a high dose rate was obtained as a consequence of experimental uncertainty.

For inhalation, we assume a unit volume concentration in air of 1 ppb, approximately the average ambient benzene concentration in the Los Angeles basin (CDHS, 1984). We also assume an average respiratory rate of 20 m³/day, an absorption coefficient of 0.5, and a body weight of 70 kg. From the dose rate and $q_{1,\text{human}}^*$, we may again calculate the upper-limit leukemic incidence rate for a population of 10^5 people continually exposed to benzene as,

$$R_{\text{inhalation}}^* = q_{1,\text{human}}^* \times d_{\text{unit conc}} \times 10^5 / 70 = 0.03$$

where R^* = number of leukemias per 10^5 people per year.

For an individual exposed to benzene at higher or lower than normal outside air level, the risk would be proportionately higher or lower.

BIBLIOGRAPHY

- Aksoy, M., et al., "Types of Leukemia in Chronic Benzene Poisoning. A Study in Thirty-Four Parts," *Acta Haematologica*, Vol. 55, pp. 65-72, 1977.
- Aksoy, M., et al., "Leukemia in Shoe-Workers Chronically Exposed to Benzene," *Blood*, Vol. 44, pp. 837-841, 1974.
- Amiel, J. L., "Negative Test for the Induction of Leukaemia in Mice by Benzene," *Revue Française d'Etudes Biologie Clinique*, Vol. 5, pp. 198-199, 1960.
- Anastos, G. J., et al., "Coal Tar Disposal: Case Study," Paper presented at the sixth annual Edison Electric Institute and Envirosphere Company conference on Environmental Licensing and Regulatory Requirements Affecting the Electric Utility Industry, Washington, D.C., October 30 to November 1, 1983.
- Anderson, M. W., D. G. Hoe, and N. L. Kapland, "A General Scheme for the Incorporation of Pharmacokinetics in Low-Dose Risk Estimation for Chemical Carcinogenesis," *Toxicology and Applied Pharmacology*, Vol. 55, p. 154, 1980.
- Arbuckle, W. B., "Estimating Activity Coefficients for Use in Calculating Environmental Parameters," *Environmental Science and Technology*, Vol. 17, No. 9, pp. 537-542, September 1983.
- Arp, E. W., et al., "Lymphocytic Leukemia and Exposures to Benzene and Other Solvents in the Rubber Industry," *Journal of Occupational Medicine*, Vol. 25, pp. 598-602, 1983.
- Beckman, D. A., and R. L. Brent, "Mechanisms of Teratogenesis," *Annual Review of Pharmacology and Toxicology*, Vol. 24, pp. 483-500, 1984.
- Benignus, V. A., "Health Effects of Toluene: A Review," *Neurotoxicology*, Vol. 2, pp. 567-588, 1981.
- Berkow, Samuel Gordon, "A Method of Estimating the Extensiveness of Lesions (Burns and Scalds) Based on Surface Area Proportions," *Archives of Surgery*, Vol. 8, pp. 138-148, 1924.
- Bolten, J. G., et al., *Risk Assessment of Toxic Pollutants from Fossil Fuel Power Plants*, Electric Power Research Institute, EPRI EA-5358, August 1987.
- Bolten, J. G., et al., *A Case Study of Beryllium Emissions from a Coal-Fired Power Plant*, The RAND Corporation, N-2484-EPRI, August 1986.
- Bolten, J. G., et al., *Alternative Models for Risk Assessment of Toxic Emissions*, The RAND Corporation, N-2261-EPRI, April 1985.
- Bos, R. P., et al., "Nonmutagenicity of Toluene, o-, m-, p-Xylene, o-Methylbenzyl Alcohol, and o-Methylbenzyl Sulfate in the Ames Assay," *Mutation Research*, Vol. 88, pp. 273-279, 1981.
- Brandt, L., et al., "Occupational Exposure to Petroleum Products in Men with Acute Non-Lymphocytic Leukaemia," *British Medical Journal*, Vol. 1, pp. 553-554, 1978.
- Brent, R. L., "Editorial Comment: Definition of a Teratogen and Relationship of Teratogenicity to Carcinogenicity," *Teratology*, Vol. 34, pp. 359-360, 1986.
- Brubaker, K. L., P. Brown, and R. R. Cirillo, *Addendum to User's Guide for the Climatological Dispersion Model*, U.S. Environmental Protection Agency, EPA-450/3-77-015, Washington, D.C., May 1977.
- Brutsaert, W., "A Theory for Local Evaporation (or Heat Transfer) from Rough and Smooth Surfaces at Ground Level," *Water Resources Research*, Vol. 11, No. 4, pp. 543-550, August 1975.

- Buckpitt, A. R., L. S. Bahnson, and R. B. Franklin, "Hepatic and Pulmonary Microsomal Metabolism of Naphthalene to Glutathione Adducts: Factors Affecting the Relative Rates of Conjugate Formation," *Journal of Pharmacological and Experimental Therapeutics*, Vol. 231, pp. 291-300, 1984.
- Busse, A. D., and J. R. Zimmerman, *User's Guide for the Climatological Dispersion Model*, U.S. Environmental Protection Agency, EPA-R4-73-024, Washington, D.C., December 1973.
- California Department of Health Services and Air Resources Board (CDHS), *Report to the Scientific Review Panel on Benzene*, Sacramento, September 1984.
- Carlone, M. F., and J. R. Fouts, "In Vitro Metabolism of p-Xylene by Rabbit Lung and Liver," *Xenobiotica*, Vol. 4, p. 705, 1974.
- Carslaw, H. S., and J. C. Jaeger, *Conduction of Heat in Solids*, 2nd ed., Oxford University Press, Oxford, 1959.
- Cohen, Y., and P. Ryan, "Multimedia Modeling of Environmental Transport," *Environmental Science and Technology*, Vol. 19, No. 5, pp. 412-417, 1985.
- Cohr, K. H., and J. Stokholm, "Toluene: A Toxicologic Review," *Scandinavian Journal of Work and Environmental Health*, Vol. 5, pp. 71-90, 1979.
- Cook, J. W., C. L. Hewett, and I. Hieger, "The Isolation of a Cancer-Producing Hydrocarbon from Coal Tar," *Journal of the Chemical Society*, Part 1, pp. 395-405, 1933.
- Dawson, J. P., W. W. Thayer, and J. F. Desforges, "Acute Hemolytic Anemia in the Newborn Infant due to Naphthalene Poisoning," *Blood*, Vol. 13, pp. 1113-1125, 1958.
- Decoufle, P., et al., "Mortality Among Chemical Workers Exposed to Benzene and Other Agents," *Environmental Research*, Vol. 30, pp. 16-25, 1983.
- DePierre, J. W., and L. Ernster, "The Metabolism of Polycyclic Hydrocarbons and Its Relationship to Cancer," *Biochimie Biophysique Acte*, Vol. 473, pp. 149-186, 1978.
- Diaz, M., A. Reiser, L. Braier, and J. Diaz, "Studies on Benzene Mutagenesis, I. The Micronucleus Test," *Experientia*, Vol. 36, p. 297, 1980.
- Environmental Health Associates, "An Industry-Wide Mortality Study of Workers Exposed to Benzene," submitted to the Chemical Manufacturers Association, Berkeley, Ca., 1983.
- Environmental Protection Agency (EPA), "National Emission Standards for Hazardous Air Pollutants: Regulation of Benzene; Response to Public Comments," Part II, *Federal Register*, Vol. 40, p. 23478, June 6, 1984.
- Environmental Protection Agency (EPA), *Treatability Manual*, Vol. I, *Treatability Data*, EPA-600/-2-82-001a, Washington, D.C., 1981.
- Environmental Protection Agency (EPA), *Ambient Water Quality Criteria for Toluene*, EPA-440/5-80-075, Washington, D.C., October 1980a.
- Environmental Protection Agency (EPA), *Ambient Water Quality Criteria for Naphthalene*, EPA-440/5-80-059, Washington, D.C., October 1980b.
- Environmental Protection Agency (EPA), *Ambient Water Quality Criteria for Polynuclear Aromatic Hydrocarbons (PAHs)*, EPA-440/5-80-069, Washington, D.C., October 1980c.
- Environmental Protection Agency (EPA), *Ambient Water Quality Criteria for Benzene*, EPA-440/5-80-018, Washington, D.C., October 1980d.
- Environmental Protection Agency (EPA), "Water Quality Criteria Documents; Availability," *Federal Register*, Vol. 45, No. 231, pp. 79318-79378, November 28, 1980e.
- Environmental Protection Agency (EPA), *Water-related Environmental Fate of 129 Priority Pollutants*, EPA-440/4-79-0296, Washington, D.C., 1979.
- Erexson, G. L., J. L. Wilmer, C. D. Auman, and A. L. Kligerman, "Induction of Sister Chromatid Exchanges and Micronuclei in Male DBA/2 Mice by the Inhalation of Benzene," Abstract No. Cb-19, *Environmental Mutagenesis*, Vol. 6, p. 408, 1984.

- Finch, Caleb E., and Leonard Hayflick (eds.), *Handbook of the Biology of Aging*, Van Nostrand Reinhold Company, New York, 1977.
- Fishbeck, W. A., et al., "Effects of Chronic Occupational Exposure to Measured Concentrations of Benzene," *Journal of Occupational Medicine*, Vol. 20, pp. 539-542, 1978.
- Fishbein, Lawrence, "An Overview of Environmental and Toxicological Aspects of Aromatic Hydrocarbons: III. Xylene," *Science of the Total Environment*, Vol. 43, pp. 165-183, 1985.
- Fitzhugh, O. G., and W. H. Buschke, "Production of Cataract in Rats by Beta-tetralol and Other Derivatives of Naphthalene," *Archives of Ophthalmology*, Vol. 41, pp. 572-582, 1949.
- Flodin, U., et al., "A Case-Referent Study on Acute Myeloid Leukemia, Background Radiation and Exposures to Solvents and Other Agents," *Scandinavian Journal of Work and Environmental Health*, Vol. 7, pp. 169-178, 1981.
- Gamberale, F., and M. Hultengren, "Toluene Exposure: II. Psychophysiological Functions," *Work Environment Health*, Vol. 9, pp. 131-139, 1972.
- Gamberale, F., G. Annwall, and M. Hultengren, "Exposure to Xylene and Ethylbenzene: III. Effects on Central Nervous Functions," *Scandinavian Journal of Work and Environmental Health*, Vol. 4, pp. 204-211, 1978.
- Gehan, Edmund A., and Stephen L. George, *The Estimation of Human Body Surface Area from Height and Weight*, University of Texas at Houston, M.D. Anderson Hospital and Tumor Institute, Houston, Tx., n.d.
- Gerner-Smidt, P., and U. Friedrich, "The Mutagenic Effect of Benzene, Toluene, and Xylene Studied by the SCE Technique," *Mutation Research*, Vol. 58, pp. 313-316, 1978.
- Gillette, D. A., and C. Cowherd, "The Concept of Resuspension Rates Applied to Problems of Fugitive Dust Emissions and Wind Erosion," in H. R. Pruppacher et al. (eds.), *Precipitation Scavenging, Dry Deposition, and Resuspension*, Vol. 2, *Proceedings of the Fourth International Conference*, Elsevier, New York, N.Y., 1982.
- Goldstein, B. D., *Benzene Toxicity: Review of Recent Literature*, prepared for the American Petroleum Institute, Department of Environmental and Community Medicine, CMDNJ-Rutgers Medical School, New Brunswick, N.J., 1983.
- Goldstein, B. D., "Hematotoxicity in Humans," in S. Laskin and B. Goldstein (eds.), "Benzene Toxicity, A Critical Evaluation," *Journal of Toxicology and Environmental Health (Supplement)*, Vol. 2, pp. 69-105, 1977.
- Goldstein, B. D., and C. A. Snyder, "Benzene Leukemogenesis," in R. R. Tice, D. L. Costa, and K. M. Schaich (eds.), *Genotoxic Effects of Airborne Agents*, Plenum Press, New York, 1982.
- Greene, M. H., et al., "Cancer Mortality Among Pringing Plant Workers," *Environmental Research*, Vol. 20, pp. 66-73, 1979.
- Griffin, K. A., C. B. Johnson, R. K. Breger, and R. B. Franklin, "Pulmonary Toxicity Hepatic and Extrahepatic Metabolism of 2-Methylnaphthalene in Mice," *Toxicology and Applied Pharmacology*, Vol. 61, pp. 185-196, 1981.
- Guess, H. A., and K. S. Crump, "Can We Use Animal Data to Estimate 'Safe' Doses for Chemical Carcinogens?," in A. Whittemore (ed.), *Environmental Health, Proceedings of SIAM Institute*, Philadelphia, Pa., 1977.
- Hanna, Steven R., Gary A. Briggs, and Rayford P. Hosker, Jr., *Handbook on Atmospheric Diffusion*, National Oceanic and Atmospheric Administration, Washington, D.C., 1982.
- Hannis, N. M., et al., "Epidemiology Study of Refinery and Chemical Plant Workers," *Journal of Occupational Medicine*, Vol. 24, pp. 203-212, 1982.

- Harper, C., R. T. Drew, and J. R. Fouts, in D. J. Jollow, J. J. Kocsis, R. Snyder, and H. Vainio (eds.), *Biological Reactive Intermediates*, Plenum Press, London, pp. 302-311, 1975.
- Hawley, J. K., "Assessment of Health Risk from Exposure to Contaminated Soil," *Risk Analysis*, Vol. 5, No. 4, pp. 289-302, 1985.
- Hine, C., and H. Zuidema, "The Toxicological Properties of Hydrocarbon Solvents," *Industrial Medicine and Surgery*, Vol. 39, pp. 215-224, 1970.
- Hiraki, K., et al., "Development of Subcutaneous Sarcomas in Swiss Mice Given Repeated Injections of Benzene in Olive Oil," *Gann-Japanese Journal of Cancer Research*, Vol. 54, p. 427, 1963.
- Hite, M., I. Smith, and S. Thornton, "Effect of Benzene in the Micronucleus Test," *Mutation Research*, Vol. 77, p. 149, 1980.
- Hoel, D. G., H. L. Kaplan, and M. W. Anderson, "Implication of Nonlinear Kinetics on Risk Estimation," *Science*, Vol. 219, pp. 1032-1038, 1983.
- Holzworth, G. C., *Mixing Heights, Wind Speeds, and Potential for Urban Air Pollution Throughout the Contiguous United States*, Environmental Protection Agency, Office of Air Programs Report No. AD-101, Research Triangle Park, N.C., January 1972.
- Hood, R. D., and M. S. Ottley, "Developmental Effects Associated with Exposure to Xylene: A Review," *Drug and Chemical Toxicology*, Vol. 8, pp. 281-297, 1985.
- Hudak, A., and G. Ungvary, "Embryotoxic Effects of Benzene and Its Methyl Derivatives: Toluene, Xylene," *Toxicology*, Vol. 11, pp. 55-63, 1978.
- Infante, P. F., et al., "Leukemia in Benzene Workers," *Lancet*, Vol. 2, pp. 76-78, 1977.
- International Agency for Research on Cancer (IARC), *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Polynuclear Aromatic Compounds, Part 1, Chemical, Environmental, and Experimental Data*, Vol. 32, Lyons, France, December 1983.
- International Agency for Research on Cancer (IARC), *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans: Some Industrial Chemicals and Dyestuffs*, Vol. 29, Lyons, France, 1982.
- International Agency for Research on Cancer (IARC), *IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans: Some Anti-Thyroid and Related Substances*, Vol. 7, Lyons, France, 1974.
- Jerina, D. M., and J. W. Daly, "Arene Oxides: A New Aspect of Drug Metabolism," *Science*, Vol. 185, pp. 573-582, 1974.
- Jerina, D. M., J. W. Daly, and B. Witkop, "The Role of Arene Oxide-Oxepin Systems in the Metabolism of Aromatic Substrates: II. Synthesis of 3,4-Toluene-4-2H Oxide and Subsequent 'NIH Shift' to 4-Hydroxytoluene-3-2H," *Journal of the American Chemical Society*, Vol. 90, pp. 6523-6525, 1968.
- Jori, A., et al., "Ecotoxicological Profile of Xylenes," *Ecotoxicology and Environmental Safety*, Vol. 11, pp. 44-80, 1986.
- Jury, W. A., W. F. Spencer, and W. J. Farmer, "Behavior Assessment Model for Trace Organics in Soil: I. Model Description," *Journal of Environmental Quality*, Vol. 12, No. 4, pp. 558-564, 1983.
- Kern, Mark J., and Moira McCue, *New Haven Waterwatch Project*, Connecticut Fund for the Environment, Inc., October 1985.
- Kimura, E., D. Ebert, and P. Dodge, "Acute Toxicity and Limits of Solvent Residue for Sixteen Organic Solvents," *Toxicology and Applied Pharmacology*, Vol. 19, pp. 699-704, 1971.
- Kirschbaum, A., and L. C. Strong, "Influence of Carcinogens on the Age Incidence of Leukemia in the High Leukemia F Strain of Mice," *Cancer Research*, Vol. 2, pp. 841-845, 1942.

- Knake, E. "Über Schwache Geschwulster-zengende Wirkung von Naphthalin und Benzol," *Virchows Archiv Pathologie, Anatomie, und Physiologie*, Vol. 329, pp. 141-148, 1956.
- Kreyszig, E., *Advanced Engineering Mathematics*, 3rd ed., John Wiley & Sons, Inc., New York, N.Y., 1972.
- Kucera, J., "Exposure to Fat Solvents: A Possible Cause of Sacralagenesis in Man," *Journal of Pediatrics*, Vol. 72, p. 857, 1968.
- Leighton, D. T., Jr., and J. M. Calo, "Distribution Coefficients of Chlorinated Hydrocarbons in Dilute Air-Water Systems for Groundwater Contamination Applications," *Journal of Chemical and Engineering Data*, Vol. 26, No. 4, pp. 382-385, 1981.
- Lignac, G.O.E., "Benzene Leukaemia in Humans and Albino Mice," *Krankheitsforsch*, Vol. 9, pp. 403-453, 1932.
- Linos, A., et al., "A Case-Control Study of Occupational Exposures and Leukaemia," *International Journal of Epidemiology*, Vol. 9, pp. 131-135, 1980.
- Litton Bionetics, *Mutagenicity Evaluation of Toluene*, Final Report to the American Petroleum Institute, Washington, D.C., LBI Project No. 20847, Litton Bionetics, Kensington, Md., 1978.
- Lyon, J. P., "Mutagenicity Studies with Benzene," Abstract, Ph.D. Dissertation, University of California, *Dissertation Abstracts*, B36:5537, 1976.
- MacKay, D., and S. Patterson, "Calculating Fugacity," *Environmental Science and Technology*, Vol. 15, No. 9, pp. 1006-1014, September 1981.
- MacLeod, Rod, Fisheries Biologist, Department of Environmental Protection, State of Connecticut, personal communication, 1986.
- Mahvi, D., H. Bank, and R. Harvey, "Morphology of a Naphthalene-Induced Bronchiolar Lesion," *American Journal of Pathology*, Vol. 86, pp. 559-571, 1977.
- Maltoni, C., "Myths and Facts in the History of Benzene Carcinogenicity," in M. A. Mehlman (ed.), *Carcinogenicity and Toxicity of Benzene*, Advances in Modern Toxicology, Vol. IV, Princeton Scientific Publishers, Inc., N.J., 1983.
- Maltoni, C., et al., "Benzene: A Multipotential Carcinogen. Results of Long-Term Bioassays Performed at the Bologna Institute of Oncology," *American Journal of Industrial Medicine*, Vol. 4, pp. 589-630, 1983.
- Marks, T. A., and T. A. Ledoux, "Teratogenicity of a Commercial Xylene Mixture in the Mouse," *Journal of Toxicology and Environmental Health*, Vol. 9, pp. 97-105, 1982.
- Mathews, J., and R. L. Walker, *Mathematical Methods of Physics*, W. A. Benjamin, Inc., New York, N.Y., 1965.
- McCann, J., E. Choi, E. Yamasaki, and B. M. Ames, "Detection of Carcinogens or Mutagens in the Salmonella/Microsome Test," *Proceedings of the National Academy of Science*, Vol. 72, pp. 5135-5139, 1975.
- McMichael, A. J., et al., "Solvent Exposure and Leukemia Among Rubber Workers: An Epidemiologic Study," *Journal of Occupational Medicine*, Vol. 17, pp. 234-239, 1975.
- Merian, E., and M. Zander, "Volatile Aromatics," in O. Huntzinger (ed.), *Handbook of Environmental Chemistry*, Part B, Vol. 3, Springer-Verlag, Berlin, pp. 117-161, 1982.
- Meyne, J., and M. S. Legator, "Sex-Related Differences in Cylogenetic Effects of Benzene in the Bone Marrow of Swiss Mice," *Environmental Mutagens*, Vol. 2, p. 43, 1980.
- Miller, J. A., "Carcinogenesis by Chemicals: An Overview," GHA Clowes Memorial Lecture, *Cancer Research*, Vol. 30, pp. 559-576, 1970.
- National Oceanic and Atmospheric Administration (NOAA), United States Department of Commerce, *Fisheries of the United States, 1985*, Current Fishery Statistics No. 8368, Washington, D.C., April 1986.

- Nawrot, P. S., and R. E. Staples, "Embryofetal Toxicity and Teratogenicity of Isomers of Xylene in the Mouse," *The Toxicologist*, Vol. 1, p. A22, 1981.
- Nawrot, P. S., and R. E. Staples, "Embryofetal Toxicity and Teratogenicity of Benzene and Toluene in the Mouse," *Teratology*, Vol. 19, p. A41, 1979.
- Neal, J., and R. H. Rigdon, "Gastric Tumors in Mice Fed Benzo(a)pyrene: A Quantitative Study," *Texas Reports on Biology and Medicine*, Vol. 25, pp. 553-557, 1967.
- Nuclear Regulatory Commission (NRC), "Calculation of Annual Doses to Man from Routine Releases of Reactor Effluents for the Purpose of Evaluating Compliance with 10 CFR, Part 50, Appendix I," *Regulatory Guide 1.109*, Washington, D.C., October 1977.
- Nunes, P., and P. E. Benville, Jr., "Uptake and Depuration of Petroleum Hydrocarbons in the Manila Clam, *Tapes semidecussata* Reeve," *Bulletin of Environmental Contamination and Toxicology*, Vol. 21, pp. 719-726, 1979.
- O'Brien, K., L. Smith, and G. Cohen, "Differences in Naphthalene-Induced Toxicity in the Mouse and Rat," *Chemical and Biological Interaction*, Vol. 55, pp. 109-122, 1985.
- Olson, B. A., F. Gamberale, and A. Iregren, "Coexposure to Toluene and p-Xylene in Man: Central Nervous Functions," *British Journal of Industrial Medicine*, Vol. 42, pp. 117-122, 1985.
- Ostle, B., *Statistics in Research*, 2nd ed., Iowa State University Press, Ames, 1963.
- Ott, M. G., et al., "Mortality Among Individuals Occupationally Exposed to Benzene," *Archives of Environmental Health*, Vol. 33, pp. 3-9, 1978.
- Pelkonen, O., and D. W. Nebert, "Metabolism of Polycyclic Aromatic Hydrocarbons," *Pharmacology Review*, Vol. 34, pp. 189-222, 1982.
- Petito, Catherine T., *Physical/Chemical/Biological Properties Influencing the Transport and Fate of Coal Tar Compounds in the Groundwater*, Gradient Corporation, Cambridge, Mass., May 1986.
- Phillips, D. H., "Fifty Years of Benzo(a)pyrene," *Nature*, Vol. 303, pp. 468-472, 1983.
- Pullman, A., and B. Pullman, "Electronic Structure and Carcinogenic Activity of Aromatic Molecules. New Development," *Advances in Cancer Research*, Vol. 3, pp. 117-169, 1955.
- Reeves, A. L., D. Deitch, and A. J. Vorwald, "Beryllium Carcinogenesis: I. Inhalation Exposure of Rats to Beryllium Sulfate Aerosol," *Cancer Research*, Vol. 27, pp. 439-445, 1967.
- Reid, R., J. Prausnitz, and T. Sherwood, *The Properties of Gases and Liquids*, 3rd ed., McGraw-Hill Book Co., New York, N.Y., 1977.
- Reid, V.V.D., K. F. Illet, J. M. Glick, and G. Krishna, "Metabolism and Binding of Aromatic Hydrocarbons in the Lung: Relationship to Experimental Bronchiolar Necrosis," *American Review of Respiratory Disease*, Vol. 107, pp. 539-551, 1973.
- Rinsky, R. A., et al., "Leukemia in Benzene Workers," *American Journal of Industrial Medicine*, Vol. 2, pp. 217-245, 1981.
- Rushton, L., and M. R. Alderson, "A Case-Control Study to Investigate the Association Between Exposure to Benzene and Deaths from Leukaemia in Oil Refinery Workers," *British Journal of Cancer*, Vol. 43, pp. 77-84, 1981.
- Schopp, G. M., et al., "Naphthalene Toxicity in CD-1 Mice: General Toxicology and Immunology," *Fundamentals of Applied Toxicology*, Vol. 4, pp. 406-419, 1984.
- Schottenfeld, D., et al., "A Prospective of Morbidity and Mortality in Petroleum Industry Employees in the United States—A Preliminary Report," in R. Peto and M. A. Schneiderman (eds.), *Banbury Report No. 9: Quantification of Occupational Cancer*, Cold Spring Harbor Laboratory, Cold Spring Harbor, N.Y., pp. 247-265, 1981.

- Schroeder, H. A., and M. Mitchener, "Life-Term Studies in Rats: Effects of Aluminum, Barium, Beryllium, and Tungsten," *Journal of Nutrition*, Vol. 105, pp. 421-427, 1975.
- Sehmel, G. A., "Resuspension Rates from Aged Inert-Tracer Sources," in H. R. Pruppacher et al. (eds.), *Precipitation Scavenging, Dry Deposition, and Resuspension*, Vol. 2, *Proceedings of the Fourth International Conference*, Elsevier, New York, N.Y., 1982.
- Sims, P., et al., "Metabolic Activation of Benzo(a)pyrene Proceeds by a Diol-epoxide," *Nature*, Vol. 252, pp. 326-328, 1974.
- Snyder, R., et al., "Biochemical Toxicology of Benzene," in E. Hodgson et al. (eds.), *Reviews in Biochemical Toxicology*, Vol. 3, Elsevier/North Holland Publishing Co., New York, N.Y., pp. 123-153, 1981.
- Stephen, H., and T. Stephen, *Solubilities of Inorganic and Organic Compounds*, Vol. 1, Part 1, Pergamon Press, Oxford, The Macmillan Company Distributors, New York, N.Y., 1963.
- Tabershaw Cooper Associates, *A Mortality Study of Petroleum Workers*, American Petroleum Institute Medical Research Report No. EA 7402, Washington, D.C., 1974.
- Tahti, H., R. K. Aaran, and H. Vapaatalo, "An Inhalation Method for Testing the Toxicity of Volatile Compounds in Small Laboratory Animals. A Study on Short-Term and Long-Term Toluene Inhalation in Rats," *Methodology and Findings in Experimental Clinical Pharmacology*, Vol. 5, pp. 667-671, 1983.
- Thomas, T. I., et al., "Mortality Patterns Among Workers in Three Texas Oil Refineries," *Journal of Occupational Medicine*, Vol. 24, pp. 135-141, 1982.
- Thorpe, J. J., "Epidemiologic Survey of Leukemia in Persons Potentially Exposed to Benzene," *Journal of Occupational Medicine*, Vol. 16, pp. 375-382, 1974.
- Tsai, S. P., et al., "Retrospective Mortality and Medical Surveillance Studies of Workers in Benzene Areas of Refineries," *Journal of Occupational Medicine*, Vol. 25, pp. 685-692, 1983.
- Ungvary, G., et al., "Studies on the Embryotoxic Effects of o-, m-, and p-Xylene," *Toxicology*, Vol. 18, pp. 61-74, 1980.
- United States Department of Commerce (USDC), Bureau of the Census, *Census of Population and Housing, 1980, Census Tracts*, Washington, D.C., July 1983.
- United States Geological Survey (USGS), *Water Resources Data*, Vol. 2, September 1985.
- United States Naval Weather Service (USNWS), *World-Wide Airfield Summaries*, Vol. III, Part 7, Washington, D.C., March 1970.
- Van Doorn, R., et al., "Effect of Toluene and Xylenes on Liver Glutathione and Their Urinary Excretion as Mercapturic Acids," *Archives of Toxicology*, Vol. 43, pp. 293-304, 1980a.
- Van Doorn, R., et al., "Electrophilic Intermediates in the Metabolism of Toluene and Xylenes in the Rat, a Metabolic Pathway Involving Sequential Side-Chain Oxidation, Sulphation and Glutathione Conjugation," *Toxicology Letters*, SI No. 1, p. 219, 1980b.
- Van Heyningen, R., "Naphthalene Cataract in Rabbits: A Resume," *Experimental Eye Research*, Vol. 28, pp. 435-439, 1979.
- Van Heyningen, R., and A. Pirie, "Naphthalene Cataract in Pigmented and Albino Rabbits," *Experimental Eye Research*, Vol. 22, pp. 393-394, 1976.
- Van Raalte, H.G.S., and P. Grasso, "Hematological, Myelotoxic, Clastogenic, Carcinogenic, and Leukemogenic Effects of Benzene," *Regulatory Toxicology and Pharmacology*, Vol. 2, p. 153, 1982.
- Vianna, N. J., and A. Polan, "Lymphomas and Occupational Benzene Exposure," *Lancet*, Vol. 1, pp. 1394-1395, 1979.

- Vigliani, E. C., "Leukemia Associated with Benzene Exposure," *Annals of the New York Academy of Science*, Vol. 271, pp. 143-151, 1976.
- Von Burg, R., "Toxicology Updates. Xylene," *Journal of Applied Toxicology*, Vol. 2, pp. 269-270, 1982.
- Warren, D. L., D. L. Brown, Jr., and A. R. Buckpitt, "Evidence for Cytochrome P-450 Mediated Metabolism in the Bronchiolar Damage by Naphthalene," *Chemical and Biological Interactions*, Vol. 40, pp. 287-303, 1982.
- Wilson, J. G., "Mechanisms of Teratogenesis," *American Journal of Anatomy*, Vol. 136, pp. 129-132, 1973.
- Withey, R., and J. Hall, "The Joint Toxic Action of Perchloroethylene with Benzene or Toluene in Rats," *Toxicology*, Vol. 4, pp. 5-15, 1975.
- Wolf, M. A., et al., "Toxicological Studies of Certain Alkylated Benzenes and Benzene," *AMA Archives of Industrial Health*, Vol. 14, pp. 387-398, 1956.
- Wong, O., *An Industry-Wide Mortality Study of Workers Exposed to Benzene*, Environmental Health Associates, Report submitted to the Chemical Manufacturers Association, Berkeley, Ca., 1983.
- Zeulzer, W. W., and L. Apt, "Acute Hemolytic Anemia due to Naphthalene Poisoning," *Journal of the American Medical Association*, Vol. 141, p. 185, 1949.
- Zinkham, W. J., and B. Childs, "A Defect of Glutathione Metabolism in Erythrocytes from Patients with a Naphthalene-Induced Hemolytic Anemia," *Pediatrics*, Vol. 22, pp. 461-471, 1958.