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Ranking Environmental Health Risks
in Bangkok, Thailand
Volumes 1 & 2

This report was co-financed by A.I.D. and the U.S. Environmental Protection Agency (EPA). The objectives of this study were twofold. First the study ranked urban environmental problems in Bangkok, Thailand, using adverse health effects information to help establish priorities among the myriad of urban environmental problems which beset Bangkok. Second, the study was designed to test whether the "Comparative Risk Assessment Methodology" developed by the EPA, which had previously been applied only in the U.S., was equally applicable to analysis of urban environmental problems in the developing world.

The methodology for this study is known as "comparative risk analysis." As adapted to Bangkok, it has three major components: a) to estimate the number and severity of cases of disease caused by each environmental problem, b) to develop a common denominator for all illnesses and injuries to provide a basis for ranking the different health problems, and c) to rank the problems in the order of the health risk caused by each.

Environmental data for Bangkok was collected and the health risks likely to be associated with the environmental problems in the city were assessed and assigned to one of three categories of risk. Airborne particulate matter, lead and infectious and parasitic organisms causing microbiological diseases were determined to be highest risk environmental problems. Although the principle purposes of the study were to describe and rank environmental health risks for Bangkok and to test the existing methodology, brief recommendations are offered for managing major environmental hazards.

Volume 1 contains the body of the report. Volume 2 consists of technical appendices which contain the complete environmental data used in ranking Bangkok's health risks.

Ranking Environmental Health Risks in Bangkok, Thailand

Volume 1

December 1990

OFFICE OF
HOUSING AND URBAN
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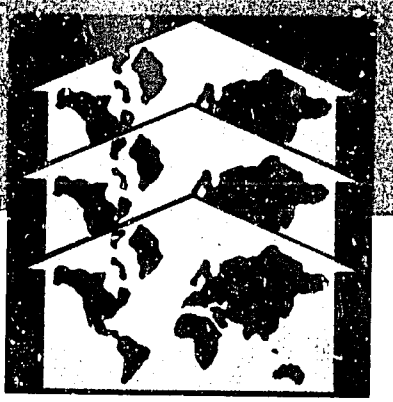
U.S. AGENCY FOR INTERNATIONAL DEVELOPMENT



W O R K I N G P A P E R

Ranking Environmental Health Risks in Bangkok, Thailand

December, 1990

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Preface and Acknowledgements

This report summarizes a project conducted during the summer and fall of 1990 for two U.S. government agencies: the U.S. Agency for International Development (USAID) (Office of Housing and Urban Programs) and the U.S. Environmental Protection Agency (USEPA) (Office of Policy, Planning and Evaluation). The project assesses and ranks the health risks from environmental problems in Bangkok, Thailand. The project represents the first application of USEPA's comparative risk analysis methods to the environmental problems of a large urban area in a developing country. It is hoped that this methodology will prove useful in establishing environmental priorities for Bangkok, as well as in future applications to other cities in the developing world.

This report was submitted to fulfill USAID Contract No. PDC-1008-I-00-9066-00, Delivery Order #8, and USEPA Contract No. 68-W9-0077, Work Assignment #39. The project team was directed by Stuart Sessions of Sobotka & Co.. The primary authors were Susan Keane of Abt Associates, Inc., and Mr. Sessions. Additional significant contributions were made by Al McGartland, Kirk O'Neal and Kathleen Bell of Abt Associates, Inc., and Kathryn Steucek of Sobotka & Co.. Questions or comments should be addressed to the primary authors.

The authors gratefully acknowledge the assistance of David Foster of the Research Triangle Institute in making arrangements for and contributing to the project team's research in Bangkok, and for his hospitality. Elaine Haemiseggar of USEPA and formerly of Abt Associates, Inc. contributed extensively in the design and research for the project. Numerous Thai and USAID Mission officials provided unselfish assistance in conducting the project, including particularly Donald Hubbard and David Painter of USAID, and Dr. Dhira Phantumvanit, Khun Phanu Kritiporn and Khun Krerkpong Charnpratheep of the Thailand Development Research Institute.

Special thanks go to Alexi Panehal of USAID and Dan Beardsley of USEPA for having

the vision to initiate the project, and to Ms. Panehal for timely guidance throughout its course. Alexi Panehal of USAID and Debra Gutenson and Pam Stirling of USEPA have served as project officers. Helpful comments on the project methodology, findings and summary report were provided by Matthew Auer and Antoinette Sebastian of USAID. The views expressed in the report, however, are those of its authors and do not necessarily reflect those of the two sponsoring agencies.

Contents -- Volume 1

Contents -- Volume 2 -- Technical Appendices

List of Tables

List of Figures

Executive Summary

In the summer of 1990, the United States Agency for International Development (USAID) and the United States Environmental Protection Agency (USEPA) financed a project to evaluate the human health risks associated with urban environmental problems in Bangkok, Thailand. The study team adapted and tested a methodology developed by USEPA. Preceding this study, the methodology had been used solely in the United States. The study had two purposes: 1) to determine whether the USEPA methodology could be adapted for application to the developing world, and, more specifically, 2) to establish a priority ranking of urban environmental health risks in Bangkok.

USAID and USEPA used health risks as the basis for the priority ranking of urban environmental problems rather than focusing on damage to the nation's natural resource base, ecology, or economy. A focus on the other varieties of risks would probably lead to a different relative ranking of problems.

The study team collected environmental data for Bangkok and analyzed the health risks likely to result from the different environmental problems in the city. Ultimately, each environmental problem was assigned to one of three categories of risk -- higher risk, medium risk, and lower risk. Airborne particulate matter, lead, and infectious and parasitic organisms causing microbiological diseases were determined to be higher risk environmental problems. Medium risks included airborne carbon monoxide, and metals other than lead. The lower risk category consisted of several environmental problems including toxic air pollutants, airborne sulfur dioxide, nitrogen dioxide and ozone, surface and ground-water contamination, food contamination with pesticides and metals, and solid and hazardous waste disposal.

With regard to the higher risk from exposure to particulate matter in air, the study suggests that inhabitants of Bangkok might suffer over 50 million restricted activity days and up to 1400 excess mortalities per year. Over half a million adults and children may be adversely

affected by exposure to lead, experiencing cardiovascular ailments and learning disabilities. Individuals are exposed to lead through all media including air, water and food. Probably sources of lead in the environment include gasoline lead emissions, industrial point source emissions, corroded water distribution pipes, and leaded paint and soil. Lead content in many foodstuffs is high. Foods may pick up lead if grown in contaminated soil or if irrigated with contaminated water. In addition, foods may absorb lead from canning or may be exposed to leaded automobile exhaust and dust during transportation or while lying in the food stalls of markets and street vendors. Microbiological diseases such as acute diarrhea, dengue fever and dysentery may affect over one and a half million urban dwellers a year and account for over six percent of all deaths in the city. The diseases are promoted by a lack of sewage conveyance and treatment, and a lack of clean, reliable water supplies for all households.

The team identified two environmental problems as posing medium risks: airborne carbon monoxide and metals other than lead. On an average day, carbon monoxide poses a minor health risk (e.g., headaches, difficulty in concentrating) to hundreds of thousands of people, and a more serious threat to the health of people with pre-existing heart disease. The team found that data on metals such as manganese and cadmium were insufficient for projecting the resulting numbers and varieties of adverse health effects. However, limited data on the levels of these metals in the hair, blood and urine of residents of Thailand appear to: a) exceed levels found to be of toxicological concern elsewhere, and b) exceed levels found among highly exposed metal workers in other countries. The team concluded that if these limited studies are indicative of the population at large in Bangkok, there is reason for concern.

In the grouping of lower risks, the team found that toxic air pollutants such as benzene, formaldehyde and asbestos could cause cancer in several dozen individuals per year, while sulfur dioxide, ozone and nitrogen oxides were expected to cause little or no adverse health impacts. Risks are also relatively low from drinking water contamination, with the exception of problems associated with microbiological contamination in parts of the water distribution network or from storage at households. Most Bangkok residents are served by public supplies from a river intake upstream of the city. The quality of the surface water at the intake is acceptable and the water

receives adequate treatment. However, contaminants may subsequently enter the drinking water through leaks in the distribution system or when households store water to compensate for erratic water supplies. This contamination contributes to the incidence of microbiological diseases in the city. Ground water is generally acceptable for drinking. Natural clay layers provide some protection of ground-water sources from contamination, though improperly constructed wells may provide occasional conduits for contaminants to reach aquifers. Generally, contamination of food by pesticides or metals (other than lead) poses low risks. Health risks posed by solid and hazardous wastes are probably small to the general population of Bangkok. However, the limited populations in close contact with large quantities of waste -- collection laborers, scavengers at the dump sites, and residents near the three major dump sites -- are more likely subject to substantial risks of respiratory, microbiological and chronic diseases.

The methodology for the study is known as "comparative risk analysis". As adapted to Bangkok, it has three major components: a) to estimate the number and severity of cases of disease caused by each environmental problem among Bangkok's population, b) to develop a common denominator for all illnesses and injuries to provide a basis for ranking the different health problems, and c) to rank the problems in the order of the health risk caused by each. The team identified several sources of uncertainty in the comparative risk analysis. Among the more important were the lack of sufficient data on certain pollutants, the limited data on rates at which Bangkok residents are actually exposed to pollutants, and incomplete scientific understanding of cause-and-effect relationships for exposure to pollutants and health.

In analyzing the results of the risk assessment, the team identified areas for further research -- information that is necessary for proper risk management. The team recommends that before specific control measures are instituted for managing particulates in air, a thorough emission inventory is necessary that will establish the relative contributions of different sources. Companion studies to investigate the volume and size of the particles, (small particles pose greater health risks to humans than large particles), and surveys to document human exposure to pollutants in different microenvironments are also warranted. The team also recommends the

undertaking of an air toxics sampling effort, as current data on air toxics are insufficient for a rigorous assessment of health risks.

Evidence from this study reveals that lead exposure is a serious problem in Bangkok, however, a large blood lead sampling study is necessary to confirm its severity. The sampling results should be correlated with socioeconomic data to determine which groups are most severely affected. Furthermore, a comprehensive study of exposure pathways is needed to determine the routes by which individuals receive the greatest doses of lead. Surveys of industrial lead emissions are also required.

To address the problem of microbiological diseases, the team suggests that the Bangkok Metropolitan Water Authority investigate various methods for increasing the amount and reliability of water supplied to households. Reliable water supplies and adequate water pressure throughout the entire distribution system will reduce the ability of external contaminants to infiltrate into the water supply system and will minimize the need for water storage by Bangkok residents. In addition, feasibility studies are required for small sewage conveyance projects.

Although the principal purposes of the study were to describe and rank environmental health risks for Bangkok and to test the existing methodology, the team made brief recommendations for managing some major environmental hazards. Control measures for particulate matter emissions should focus on major industrial sources and diesel vehicles. Tighter control of automobile emissions and incentives to retire old vehicles and replace them with technologically advanced, less polluting cars and trucks will alleviate the problems associated with carbon monoxide. The team encourages the Government of Thailand's continuing effort to reduce the lead content of gasoline.

In Thailand, the results of this study will be incorporated into USAID's Management of Natural Resources Project. The Thailand Development Research Institute, a prestigious organization that studies environmental and natural resource policy issues, plans to use this study in preparing its own reports for the Government of Thailand. Finally, the study will be analyzed

to determine whether USEPA's comparative risk methodology is useful in urban areas outside of the United States, and whether additional applications should be pursued elsewhere.

1. Introduction

In recent years, the United States Agency for International Development (USAID) has made substantial contributions to improving environmental conditions in developing nations. Most USAID projects to date have aimed at improving rural environmental conditions and increasing the degree to which a country's natural resources are managed for sustainable economic development. Yet urban environmental conditions may be equally problematic, and equally in need of improvement if a country is to realize its potential for long run economic growth. This study represents an innovative attempt to understand and prioritize urban environmental problems in Bangkok, Thailand.

The Bangkok Metropolitan Area has been the engine driving Thailand's impressive economic growth for the past two decades. Despite comprising only about 10% of the nation's population of 60 million, Bangkok has contributed half of the growth in Thai GNP between 1970 and 1986.

Poor environmental conditions in Bangkok threaten the continuation of this growth. Environmental problems in Bangkok are numerous and severe. Only about 2% of Bangkok's population is served by sewage treatment facilities. Human waste from the remainder of the population is treated inadequately in cesspools and septic tanks or not at all, and most quickly finds its way into surface or ground water. Largely as a result of the high human waste loads, the major river (Chao Phraya) through Bangkok is nearly dead, with conditions approaching anaerobic through the city and downstream.

About three quarters of Bangkok's population is provided with piped potable water from upstream surface sources. Although the water is well treated in modern plants, problems in the distribution system allow contaminants to infiltrate and many of the water system customers must boil their water and/or purchase bottled water to reduce risks of waterborne disease. Most of the remainder of the population relies on ground water from private wells and unlicensed sources of

uncertain quality.

Bangkok is at a mean elevation of 1.5 meters above sea level, and is subject to serious and costly flooding. Many of the klongs that formerly provided drainage have now been covered and filled. Excessive pumping of ground water appears to contribute to land subsidence, exacerbating the flooding in the city. Flooding is a serious problem; the floods of 1983, for example, are estimated to have resulted in over $200 million of damages.

About 1/5 of Bangkok's solid waste is uncollected. Most of that which is collected is placed in open dumps, supporting rats and insects and generating highly contaminated leachate and runoff. There is little provision for identification and separate management of hazardous waste. Inadvertent human contact with toxic wastes has resulted in several reported incidents of serious illnesses.

Air pollution and noise are growing problems with rapid increases in motor vehicle use and industrial and construction activity. National air quality standards for particulate matter are frequently exceeded, and are occasionally exceeded for carbon monoxide.

The combination of unmanaged human and solid waste and other unsanitary conditions has led to a substantial incidence of environmentally related infectious and parasitic disease among Bangkok residents. Toxic chemicals from industrial and agricultural sources are being found frequently in fish, other foodstuffs, river water and sediments. They are accumulating to worrisome levels in exposed humans, threatening additional adverse health effects.

The sheer magnitude of Bangkok's environmental problems in relation to the resources available to address them appears to discourage environmental progress. At least 5 studies have been conducted of the city's sewage treatment needs, the latest estimating a cost of $1.4 billion (in 1980 dollars) for a full system. Such a funding commitment is well beyond current local capabilities and possibly also beyond current national capabilities. Perhaps because solving the entire problem seems so infeasible, little progress appears to be occurring in sewage treatment.

In such a situation of staggering environmental needs and sharply limited resources to address them, careful establishment of environmental protection priorities is a necessity. It is critical that the Thai government use wisely the resources available for environmental protection from domestic sources and the international donor community, investing so as to buy the most environmental protection possible with the resources available. Faced with an excess of environmental problems needing attention, how is a government to make the choice of which to address first?

Over the past several years, the U.S. Environmental Protection Agency (USEPA) has developed methods to assist governments that find themselves in such circumstances. USEPA has assisted about twenty U.S. cities, counties, states and regions in establishing environmental priorities, and has incorporated this experience into a general methodology. USEPA's approach typically involves two steps:

1. Identifying, evaluating, and ranking in priority order the environmental problems facing the subject geographical area. The first question to be answered is: "What are the most serious environmental problems facing us today?" This is known as the risk assessment phase of the process.

2. Developing and analyzing cost-effective policy measures to mitigate the highest priority environmental problems. The government asks itself: "What can we do about these problems?" This is known as the risk management phase of the process.

In this project, USAID and USEPA have co-funded application of the first step of this process to Bangkok and its environmental problems. The project is intended both to aid Thailand specifically, and to test whether the USEPA methodology is likely to be applicable generally to environmental problems in large cities throughout the developing world. The U.S. agencies sponsoring this work are concerned with both aims. They hope that the project will be useful to the Royal Thai government and to the USAID Mission in Thailand in establishing priorities for Thai environmental programs. The sponsoring agencies are also interested in learning how

USEPA's methodology -- which to date has been employed only for environmental problems in the U.S. -- could be adapted to provide a basis for priority-setting in foreign, less developed countries. This report focuses on the project findings in Bangkok. A separate memorandum provides an evaluation of how well USEPA's methods have worked in this application to Bangkok and suggestions about how the approach might be further modified for application elsewhere.[1]

1.1 Objectives of the Project

The following are the explicit objectives of the project:

1. Identify and briefly describe the major environmental problems affecting the health of Bangkok's population.

2. Rank these problems in priority order, in terms of the relative magnitude of the adverse impacts on human health caused by each.

3. Identify the major data gaps in understanding the health impacts of Bangkok's environmental problems.

4. As time and resources permit, suggest promising options for cost-effective mitigation of the high priority problems.

5. Develop and test a methodology for comparative assessment of environmental problems that may be applicable to other urban areas in developing nations throughout the world.

1.2 Organization of this Report

[1]. See the memorandum: "Assessment of Comparative Risk Analysis Methodology as Applied to Bangkok, and Suggestions Regarding its Future Application to Cities in Developing Countries"; to Alexi Panehal, USAID/PRE/H; from S. Sessions and A. McGartland; December, 1990.

This overview report summarizes the findings and methods of the project. Chapter 2 presents a summary ranking of the environmental problems in Bangkok based on the relative severity of the health risks they pose. Chapter 3 describes each of the health risks in more detail. Chapter 4 discusses the comparative risk analysis methodology used to develop these findings. Chapter 5 concludes with some brief suggestions regarding promising environmental management strategies for Bangkok and key data gaps that should be addressed. Most of the technical details supporting the findings of the project are contained in a series of technical appendices. Six technical appendices describe the data, analysis and conclusions for the individual environmental problem areas. Two final technical appendices describe the process of health risk assessment as conducted by USEPA.

2. Relative Ranking of Environmental Problems

2.1 Summary Ranking of Problems

In this project, we have acquired a large amount of data on emissions and concentrations of pollutants in Bangkok, and human exposure to them. These data have been combined with information on the health effects that may be caused by environmental pollutants to estimate the likely number of adverse health impacts resulting from each environmental problem in Bangkok. We then rank environmental problems relative to each other based on the number and severity of adverse impacts they cause.

Although grounded in science, this process of comparative risk analysis is ultimately qualitative and subjective. The available information is limited and somewhat uncertain, with regard to both the levels of pollutants and their potential health effects. Our summary judgment about each environmental problem in Bangkok thus involves combining our estimate of the number and severity of health effects it causes with an understanding of the quality and biases in the data underlying this estimate.

We reflect our level of certainty in these judgments by classifying each environmental problem into one of only three categories: higher risk, medium risk, or lower risk. We feel quite confident in assigning problems to one of these three ranking categories, but less confident in making further distinctions within a category about the relative risks posed by the different problems. We believe there are very substantial differences in risk between problems in different categories, of at least one and probably more orders of magnitude. In our view, a problem in the "higher risk" category is at least 100 times as serious from a health risk perspective as a problem in the "lower risk" category. Table 2.1 summarizes our relative ranking of environmental problems in Bangkok based on the health risks they pose.

Table 2.1 Summary Ranking of Environmental Problems

Higher Risk:	Particulate Matter
	Lead
	Microbiological Diseases
Medium Risk:	Carbon Monoxide
	Other Metals
Lower Risk:	Toxic Air Pollution
	Other Criteria Air Pollutants (SO_2, NO_2, O_3)
	Surface Water Contamination
	Ground Water Contamination
	Food Contamination (pesticides and metals)
	Solid & Hazardous Waste Disposal

Note: Ranking is based on relative health risks to the population of Bangkok. Problems are unranked within risk categories.

2.2 Higher Risk Problems

Three problems are ranked as higher risk: particulate matter air pollution, lead, and microbiological diseases. Each of these three problems causes very widespread health effects, including a substantial number of deaths. Data supporting these estimates are of good quality.

Particulate matter concentrations in Bangkok's ambient air substantially exceed both Thai and U.S. health-based ambient standards. Ambient levels have worsened in recent years. The levels of particulate matter are estimated to cause 9 - 51 million days per year of restricted activity for respiratory reasons[2] for Bangkok residents, and up to 1,400 deaths per year. The high end of these ranges correspond with the most recent ambient monitoring data. These estimates are based on extensive Thai ambient monitoring data and numerous studies performed throughout the world relating incidence of human health impacts to particulate matter concentrations.

Lead levels in Bangkok appear high in air, drinking water and food. Our estimates of health effects stem from sampling data on levels of lead in the blood of residents across the entire country. We have no blood lead sampling data specifically for Bangkok, but because lead is typically an urban pollutant, levels in Bangkok might be even higher. There is also some difference across the studies we reviewed in the average blood lead levels found, with the most recent study appearing to show the highest levels. Again, the high end of the estimated risk range seems most likely. Our estimates of health effects among adult males include several hundred thousand cases of hypertension per year, and stemming from them up to 800 cases of heart attack and stroke, and up to 400 deaths. Among children, significant reduction in mental capacity is expected; the loss of IQ points is estimated at 700,000 for each cohort of children, or 3 - 5 points for the average child through the age of seven. These estimates are based on

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[2]. A respiratory restricted activity day is a day on which an individual experiences sufficient respiratory distress or illness to cause a significant reduction in normal daily activity. The individual typically remains inactive at home. In the U.S., more than half of all restricted activity days result in absence from work.

extensive recent U.S. studies of the health effects of lead exposure, which are providing the impetus for very sharp reductions of lead in the U.S.. We have not calculated any effects on women or fetuses from lead in Bangkok, as the scientific relationships in this area are still speculative. If proven, these effects would further increase the already large health impacts estimated to result from lead.

Microbiological diseases are a longstanding major source of health problems in Bangkok, as well as in nearly every developing country of the world. We estimate that between 850,000 and 1.7 million cases of these diseases (primarily acute diarrhea, dengue fever, dysentery and helminthiases) occur in Bangkok each year. This estimate is quite uncertain, as it is based on scaling up the reported number of cases of such diseases by a factor of 5-10 to account for under-reporting. We are more confident in the reporting of deaths in Bangkok; about 6% of the deaths are attributed to microbiological diseases. Environmental causes are responsible for only a fraction of the cases of these diseases. Other problems outside the environmental scope of this project, such as inadequate health care and education, are also clearly important in determining the prevalence of these diseases.

Each of these three environmental problems causing high health risks will also cause large economic losses in Bangkok. The cost of absences from work and medical care for the large number of cases of these diseases are substantial. Lead also appears to impose a high economic cost in significantly reducing the mental capacity and future productivity of children. Further research to quantify these economic losses might provide an important stimulus to improved control of environmental pollution in Bangkok.

2.3 Medium Risk Problems

We have ranked two problems as medium risk: carbon monoxide air pollution and metals other than lead. These two problems both potentially affect a large fraction of Bangkok's population, but are ranked medium for somewhat different reasons.

Carbon monoxide is a ubiquitous air pollutant in Bangkok resulting from heavy use of vehicles with poorly controlled emissions in congested areas. The levels of this pollutant at curbside often exceed levels at which health effects may be expected, but levels found at monitors for the general ambient air are below thresholds of concern. This finding corresponds with the typical experience that carbon monoxide concentrations fall off rapidly as one moves away from busy roads and intersections. Our estimate that a substantial fraction of Bangkok's population is at risk from this pollutant is based, in effect, on a very conservative and inaccurate assumption that all Bangkok residents spend their day at curbside. The number of individuals among the estimated population at risk that will actually experience the adverse health impacts (angina pain for those with heart disease; headaches and an inability to concentrate for others) is unknown, but presumably rather low. These health effects are not severe; they are treatable and transient. In sum, despite the widespread high levels of carbon monoxide in Bangkok, we rank it as only a moderate health risk because of several levels of conservatism in our analysis and because of the relatively mild health effects at issue.

Metals other than lead are ranked as a moderate health risk in Bangkok, based upon limited information suggesting potential health problems across the general population, but insufficient evidence to warrant ranking it as a major problem. Average levels of manganese and cadmium in body tissues of Thai residents have been found to be comparable to those in highly exposed workers in metal plants in other countries. Body levels of these metals in Thailand exceed levels found to be of toxicological significance elsewhere. However, health effects studies are not sufficiently advanced for us to be able to evaluate the risks posed by these levels. There is also some uncertainty because we have no specific data on metal concentrations for residents of Bangkok.

2.4 Lower Risk Problems

The other environmental pollution problems in Bangkok that we have examined in this study have all been ranked as posing lower health risks. They include: other criteria air pollutants (sulfur dioxide, nitrogen dioxide and ozone), toxic air pollutants, surface water contamination, ground-water contamination, food contamination (pesticides and metals), and solid and hazardous waste disposal. This is primarily because contaminants from the lower ranked problems rarely exceed health-based thresholds for much of Bangkok's population. For some small, specific groups of individuals, though, one of the lower ranked problems may pose significant health risks (e.g., residents near one of Bangkok's waste dumps may face greater risks from solid and hazardous waste disposal than from one of the problems ranked as higher risk for Bangkok's population in general).

Other criteria air pollutants (sulfur dioxide, nitrogen dioxide and ozone) occur in Bangkok at levels well below health-based thresholds. Toxic air pollutants have been estimated in a rough analysis to cause 60 - 70 cancer cases annually among the city's population, but these calculations are quite conservative and the cancer potency factors that have been used sharply overestimate the actual number of resulting cancers. On the other hand, our air toxics analysis considered the contribution from mobile sources only, ignoring that from industry. While we believe that air toxics are a lower health risk problem, it is possible that additional studies could find them to pose somewhat greater risks.

Surface water contamination appears to present low risks. The quality of the surface water obtained for most of Bangkok's drinking water supply is acceptable, the treatment given to it is good, and the very limited at-the-tap monitoring data we obtained were acceptable also. Exceptions to this low risk finding for drinking water involve lead and microbiological contaminants. Lead in drinking water provides a moderate contribution to the serious problem of lead in the aggregate. Microbiological contamination of drinking water is probably a moderate contributor to the total incidence of microbiological disease. Other pathways by which surface water contamination can affect health -- direct contact, irrigation, fish consumption -- all appear

to present minimal health risks except, again, for their probably rather small contribution to microbiological diseases.

We have very little data with which to assess the health risks associated with ground-water contamination in Bangkok. The monitoring data we acquired were for areas surrounding Bangkok, and not for Bangkok itself. Data were not available for several key classes of potential contaminants. The available data suggest no health problems from ground-water contamination. More important than the limited monitoring data, however, is the geology underlying Bangkok. Multiple clay layers provide the aquifers with good protection from surface contamination. Ground-water contamination may be a threat only in limited areas where an improperly constructed ground-water well provides a conduit for contamination from the surface to penetrate the clay layers and reach an aquifer.

Food contamination from environmental sources seems to pose relatively low health risks. Pesticide residues are estimated to result in 14 excess cancer cases annually, but there are several reasons why even this moderate number is probably an overestimate. Metals are found in foods at levels of from 9 - 120% of their RfDs[3], but adverse health impacts are unlikely to result unless levels become substantially higher than these.

Finally, solid and hazardous waste disposal are judged to result in lower risks. Data here are quite limited, both on the toxic constituents in Bangkok's wastes and on the degree to which they are released from the dump sites. Information is sufficient to estimate substantial risks to scavengers and the communities adjacent to the three dumps. Since, ambient concentrations of pollutants typically decline greatly as one moves away from dump sites due to dilution, attenuation and degradation of the contaminants, we doubt strongly that the dumps could result

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[3]. RfD is the abbreviation for Reference Dose. The RfD for a chemical is the dose which, when consumed daily by an individual for a lifetime, is sufficiently safe to yield only a trivial risk of adverse health effects. The RfD is also known as the Acceptable Daily Intake. RfDs have a margin of safety built into them. A dose less than the RfD of a chemical is nearly certainly safe. A dose exceeding the RfD is not necessarily unsafe.

in substantial risks to Bangkok's general population. Uncollected solid waste may contribute in a minor way to the incidence of microbiological diseases.

2.5 Factors Considered in Ranking Environmental Problems

Table 2.2 displays our conclusions and judgments about the several factors we considered in comparing the risks of the different environmental problems. For each problem, the table covers:

- Estimated health affects. In most cases, we have estimated both the number and type of health effects across Bangkok's population. In several cases, we can only estimate the number of people at risk of a particular health effect.

- Severity of the health effects. The particular health effects caused by the problem are classed as severe, moderate, or mild. This information derives from a severity index described in Section 4.7 and Appendix H.

- Quality of the exposure data. This column indicates both the general quality of the data on emissions, ambient concentrations and doses of pollutants and the nature of any biases likely in our analysis of the data. For example, important classes of contaminants may be omitted from the analysis, resulting in an underestimate of risks. Alternatively, very conservative assumptions might be made about contaminant fate, transport and exposure, resulting in an overestimate of risks.

- Nature of the health data. This column indicates whether the nature of the underlying health effects data used in the risk assessment yields best estimates or conservative estimates. In general, epidemiologically derived relationships yield unbiased "best" estimates. Estimates of the number of cancer cases and the number of people "at risk" because of doses exceeding RfDs will typically

substantially exceed the actual number of cases.

The final column of the table provides a summary judgment about the health risk posed by the problem relative to the health risks posed by the other environmental problems in Bangkok. The final column relies on the information from the other columns.

TABLE 2.2 Comparative Health Risks From Environmental Problems

Problem	Estimated Health Effects	Severity of Each Effect	Quality of Exposure Data	Nature of Health Data	Summary
Air Pollution					
Particulate Matter	9 - 51 million restricted activity days/yr	Mild	Good	Best estimate	Higher risk
	300 - 1400 deaths/yr	Severe			
Carbon monoxide	Avg day: 20,000 people at moderate risk of angina	Mild	Good. Risk is over-estimated by assuming that everyone is exposed at curbside levels.	Number of people actually suffering the effects will be much lower than the number at risk.	Medium risk
	900,000 people at slight risk of headache	Mild			
Lead	---------- Lead in all media discussed below ----------				
Sulfur dioxide, nitrogen oxides, ozone	None	---	Good	Best estimate	Lower risk
Toxic substances	70 - 80 cancers/yr	Severe	Poor. Estimate covers toxics from mobile sources only.	Number of actual cancers likely to be far lower.	Lower risk, perhaps medium
Water Pollution					
Surface water as drinking water source	3 cancers/yr Population not at risk from other contaminants, as doses < RfDs.	Severe	Fair. No data on several classes of contaminants.	Number of actual cancers likely to be far lower.	Lower risk. Contaminants with no data probably don't add much risk.
Ground water as drinking water source	No cancers Population not at risk from other contaminants, as doses < RfDs.	---	Poor. No data on several classes of contaminants.	---	Lower risk. Contaminants with no data probably don't add much risk.

TABLE 2.2 Comparative Health Risks From Environmental Problems (cont'd)

Problem	Estimated Health Effects	Severity of Each Effect	Quality of Exposure Data	Nature of Health Data	Summary
Water Pollution (cont'd)					
Other water pathways: direct contact, crop irrigation, fish consumption	Virtually no risks except through microbiological agents.	--- contribute to Microbiological Diseases and Lead, discussed below ---			Lower risk
Contamination of drinking water in distribution system or at point of use	---------- contributes to Microbiological Diseases and Lead, discussed below ----------				
Lead	Adult males:				
	200,000 - 500,000 cases hypertension/yr	Medium	Fair. Levels in Bangkok are perhaps higher than the country's averages we use.	Best estimate	Higher risk
	300 - 800 cases heart attack & stroke/yr	Severe			
	200 - 400 deaths/yr	Severe			
	Children:				
	500 - 60,000 need medical attention/yr	Medium			
	400,000 - 700,000 points IQ lost/yr	Medium			
Other metals					
Manganese & cadmium	Uncertain. Body levels exceed those of toxicological significance.	Uncertain	Fair	Health data not sufficient to permit quantitative risk assessment.	Uncertain importance. Ranked medium risk.
Mercury	None	---	Questionable	---	Lower risk, but uncertain because of questionable data on body levels.

TABLE 2.2 Comparative Health Risks From Environmental Problems (cont'd)

Problem	Estimated Health Effects	Severity of Each Effect	Quality of Exposure Data	Nature of Health Data	Summary
Microbiological Diseases	6% of deaths in Bangkok .85 - 1.7 million cases/yr of diarrhea, dengue, etc.	Severe Nearly all mild	Both environmental and non-environmental factors contribute importantly to these diseases. Case estimate highly uncertain due to difficulty of estimating rates of under-reporting.		Higher risk
Food Contamination					
Pesticides	14 cancers/yr	Severe	Fair	Number of actual cancers likely to be far lower.	Lower risk
Metals	6 metals at 9 - 120% of RfDs	Variable	Fair	Doses moderately exceeding RfDs unlikely to pose significant risks.	Lower risk, but would become more important if other exposure routes also contributed significant metal doses.
Solid & Hazardous Wastes	Less than 1 cancer/yr No other effects	Severe ---	Very limited data on only a few contaminants. Large overestimate of likely exposure to these contaminants.	Number of actual cancers likely to be far lower.	Lower risk (But significant risk to some small groups)

2.6 Interpreting the Rankings

Several points about interpreting the results of this study should be made. These points stem from choices made in establishing the methodology for this project, and they are described in more detail in Chapter 4.

First, the rankings of environmental problems in Bangkok are based on their health risks. A focus on other varieties of risks -- risks to ecological systems, economic losses, or damages to natural resources -- would probably lead to a different relative ranking of problems.

Second, the rankings are intended to reflect "residual risks", or those that result from environmental problems as they currently exist. Trends may change the relative magnitude of these risks over time. Environmental control programs take time to implement, and it will be important to re-examine the remaining risks as control programs progress. Several researchers argue that developing countries such as Thailand will undergo a transition in the types of environmental risks they face.[4] A rising standard of living will reduce traditional problems like microbiological disease, but increased industrial and consumer activity will intensify problems such as toxic emissions and effluents and hazardous wastes.

Third, the rankings are based on an estimate of the aggregate health effects caused by the problems to the population of Bangkok. Severe effects of an environmental problem on a small sub-population are treated as less important than moderate effects on a very large population. A problem that is geographically widespread and affects many people will rank high under this approach. A problem that is localized and affects few people, even though it may affect them severely, will rank lower.

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[4]. See K.R. Smith, "The Risk Transition," Working Paper no. 10 (Honolulu, Hawaii: Environment and Policy Institute, East-West Center, 1988) and Murray L. Cohen, et al, Health Consequences of Industrialization and Urban Development in Thailand, a Project Report Submitted to USAID/Thailand, September, 1985.

Fourth, several environmental problems of potential importance to the health of Bangkok residents have not been covered in our analysis. They include problems that the Thai government is unlikely to be able to do much about (e.g., global climate change), problems for which basic data were not available (e.g., indoor air pollution), and problems that we judged to relate more to public or occupational health than to environmental pollution (e.g., malnutrition, food additives, worker safety and exposure to toxic substances).

Finally, a ranking of problems according to their risk will not necessarily match the priority that should be given to control programs for each problem. Management of environmental risks -- what one chooses to do about environmental problems -- will depend on many factors in addition to the magnitude of the risks. Important risk management factors that are not addressed in this report include the cost and technical feasibility of control measures for the different environmental problems, public opinion, political concerns, legal authority and institutional issues. In setting priorities for control efforts, all of these factors should be considered. One might find that there are particularly good opportunities to reduce risks associated with a relatively lower risk problem, while there is nothing very effective that might be done about some higher risk problem.

On balance, we believe that there is likely to be a general correlation between the environmental problems posing higher residual risks and the problems for which additional control efforts will prove most cost-effective and that comparative risk analysis is a useful tool to start with in setting priorities. In moving from comparative risk analysis to establishing priorities and designing control programs, however, another level of analysis is necessary. Candidate control measures must be evaluated against all the risk management factors.

3. Findings for Each Environmental Problem

3.1 Summary

In this chapter, we discuss the findings for each separate environmental problem that underlie the relative rankings. The findings are presented here in summary fashion. Technical appendices to this report explain in more detail the data, assumptions, calculations and references supporting the findings, and their limitations. Separate appendices are included for each major group of environmental problems: air pollution, water pollution, food contamination, solid and hazardous waste disposal, lead and other metals, and microbiological diseases.

In order to provide a reference point, ambient concentrations of environmental pollutants in Bangkok are compared, whenever possible, with both Thai and U.S. standards. In some instances, though, standards do not yet exist for some pollutants and exposures of concern. In other cases, the standards of one or both countries have not been updated to reflect the latest information on the health effects of some environmental pollutants. In general, an instance where the ambient level of a pollutant violates a standard usually does suggest a health risk. The converse, however, is not generally true. The fact that no standard is violated does not mean that there may not be an important health risk. In our analysis, we estimate health risks by reference to the basic health effects information on each pollutant, and not by reference to standards.

3.2 Air Pollution

Air pollution in Bangkok has increased in recent years as a result of growing population, industrial activity and motor vehicle use. Although a detailed emission inventory for Bangkok is not available, we suspect that mobile sources are a particularly large contributor to air pollution in the city, with industry, construction, general fuel burning (for cooking, etc.) and open burning of trash of lesser importance. Our risk assessment relies on ambient air monitoring data for six

common air pollutants: particulate matter, carbon monoxide, lead, sulfur dioxide, ozone, and nitrogen dioxide. Risks from a wide variety of toxic air pollutants (e.g., benzene, cadmium) are estimated roughly by extrapolating ambient concentrations from emissions estimates. A summary of our findings with regard to health risks from air pollution in Bangkok is provided in Table 3.1. Appendix A discusses the data, calculations and findings for air pollution in more detail.

Particulate Matter

Airborne particulates constitute a serious threat to public health in Bangkok. Ambient levels average 90 - 200 ug/m^3, well in excess of both the Thai standard of 100 ug/m^3 and the old U.S. standard of 75 ug/m^3.[5] Such high levels of particulate matter have been linked epidemiologically to a wide variety of adverse respiratory health effects. We estimate these levels result in between 9 million and 51 million "restricted activity days" annually among Bangkok's population. A restricted activity day is one on which an individual feels ill and cannot pursue a normal level of activity. In the U.S., more than half of all restricted activity days result also in days of work loss -- it is clear that this adverse health effect can result in substantial economic losses to Bangkok in terms of medical costs and lost productivity from the work force.

We also estimate that these levels of particulate matter cause 300 - 1400 excess mortality cases annually. The exact cause of the epidemiological correlation between high levels of particulate air pollution and death rates is not known with certainty. It is assumed that the excess mortality involves individuals who are already medically compromised (e.g., with respiratory

[5]. The U.S. standard for particulate matter has been revised to apply only to smaller, respirable particulate matter. The current U.S. standard cannot be compared directly with the ambient levels cited here or with the Thai standard.

TABLE 3.1 AIR POLLUTION

Pollutant	Ambient Levels	Adverse Health Effects	Notes
Particulate matter	90 - 200 ug/m3 Thai std. 100 US std. 75	9 - 51 million restricted activity days/yr 300 - 1400 excess mortality/yr	Sources uncertain: industry, construction, transportation, fuel burning, trash burning
Carbon monoxide	1 - 21 mg/m3 curbside Thai std. 20 US std. 10	Avg. day: 20,000 people with heart disease at moderate risk of angina pain 900,000 people at low risk of milder symptoms (e.g. headache)	Lax auto standards and enforcement
Lead	.2 - .7 ug/m3 ambient .3 - 3.0 curbside Thai std. 10 US std. 1.5	Substantial. See separate findings for lead in Table 3.4	
Sulfur dioxide	7 - 15 ug/m3 Thai std. 100 US std. 80	None	
Nitrogen oxides	16 - 34 ug/m3 Thai std. 320 US std. 100	None	
Ozone	up to 150 ug/m3 Thai std. 200 US std. 235 ug/m3	None	Levels likely to increase over time with traffic, industry
Toxic substances	----------	70 - 80 cancers/yr	Assessed for mobile sources only, through use of emission factors. In US, mobile sources contribute 58% of air toxics cancer incidence

ailments) who cannot survive the additional strain of high levels of air pollution.

No inventory of the source types responsible for Bangkok's particulate emissions is available. Likely major sources include industry, construction activities, transportation (particularly diesel engines), general fuel burning and open burning of trash. Developing an emission inventory for particulate matter in Bangkok would be worth while.

Carbon Monoxide

Carbon monoxide in Bangkok derives nearly exclusively from motor vehicles. Monitored levels are highest at curbside, and they fall off sharply-away from roads and intersections. Curbside levels have been monitored at 1 - 21 mg/m^3 -- nearly always less than the Thai standard of 20 mg/m^3, but often above the U.S. standard of 10 mg/m^3. Such levels of carbon monoxide have been associated with increased incidence of heart pain (angina) in persons with chronic cardiovascular disease, and with milder symptoms such as headaches and inability to concentrate among the general population. We estimate that carbon monoxide concentrations on an average day in Bangkok put about 20,000 people with heart disease at moderate risk of angina pain, and 900,000 members of the general population at low risk of milder symptoms. On the day with the highest carbon monoxide levels, about 50,000 people will be at moderate risk of angina pain, and over 2 million people will be at low risk of the milder symptoms.

By U.S. standards, the Thai limits on carbon monoxide emissions from motor vehicles are very lax. Enforcement of the existing standards is also reportedly weak.

Lead

Lead is found in Bangkok's ambient air at .2 - .7 ug/m^3, and in curbside locations at .3 - 3.0 ug/m^3. By contrast the Thai ambient standard is 10 ug/m^3 and the U.S. standard is 1.5 ug/m^3. The U.S. has been sharply lowering its standards for all routes of lead exposure in recent years based on mounting evidence of adverse effects of very low levels of lead. Information on

the health effects of lead indicates that total exposure to lead from all media (air, water, food) is critical, and that analysis of all media simultaneously is preferred to analysis of any one medium. The very extensive adverse health effects we find from total exposure to lead in Bangkok will be discussed in a subsequent section of this chapter.

Sulfur Dioxide

This pollutant usually derives from fossil fuel power plants and other major fuel burning industrial facilities. Its monitored levels in Bangkok are quite low at 7 - 15 ug/m^3, relative to the Thai standard of 100 ug/m^3 and the U.S. standard of 80 ug/m^3. No adverse health effects are expected from sulfur dioxide at these levels.

Nitrogen Dioxide

Nitrogen dioxide is emitted both by motor vehicles and major fuel burning installations such as power plants. Its levels are low in Bangkok at 16 - 34 ug/m^3 relative to the Thai standard of 320 ug/m^3 and the U.S. standard of 100 ug/m^3.[6] No adverse health effects are expected at these levels.

Ozone

Ozone at ground level is a pollutant formed by the interaction of hydrocarbons and nitrogen oxides in the presence of sunlight. (Ground level ozone as a pollutant should not be confused with stratospheric ozone, which is desirable and protects the earth from harmful ultraviolet radiation.) Ozone has been observed in Bangkok at levels up to 150 ug/m^3. The Thai standard is 200 ug/m^3 and the U.S. standard is 235 ug/m^3. There is some scientific debate over

[6]. The Thai and U.S. standards are not directly comparable, as the Thai standard is the maximum allowed over one hour, while the U.S. standard is the maximum allowed as an annual average.

whether adverse health effects among a sensitive population (asthmatics) might be expected at these levels. The USEPA decided recently not to tighten the U.S. standard, and that it is sufficiently protective. We will assume no adverse health effects from the ozone levels in Bangkok.

Ozone concentrations in Bangkok may have increased in recent years. Characterized by hot, sunny weather and low wind speeds, meteorological conditions in Bangkok facilitate production of ozone. With continued growth of motor vehicle traffic and increases in hydrocarbon emissions from industry, Bangkok may soon develop an ozone problem.

Toxic Air Pollutants

A very wide variety of other pollutants such as benzene, formaldehyde, cadmium and diesel particulates found in ambient air can cause adverse health effects. We were not able to obtain ambient monitoring data for such pollutants, but we did estimate ambient concentrations for some of them roughly by applying emission factors and dispersion modeling to estimates of vehicular miles traveled in Bangkok. Such estimates of ambient concentrations in Bangkok omit the effect of industrial and other area sources (e.g., dry cleaners).

Some of the health effects that can be caused by these pollutants, such as kidney damage from cadmium, are thought to involve threshold mechanisms (where the pollutant must be present at a level exceeding a certain threshold in order for adverse impacts to occur). We calculated that none of these pollutants were likely to occur at ambient levels in Bangkok high enough to exceed health effect thresholds. However, other health effects may occur without any threshold mechanism. It is thought that any exposure, no matter how small, to certain toxic air pollutants will increase an individual's risk of contracting lung cancer. Using our estimates of ambient concentrations and established carcinogenic potency factors for toxic air pollutants, we estimate 70 - 80 excess cancers per year among Bangkok's population from toxic air pollutants from mobile sources. U.S. studies have found 58% of cancer incidence from toxic air pollutants to derive from mobile sources. We have no information with which to judge whether mobile

sources contribute a greater share of cancers from toxic air pollutants in Bangkok than in the U.S.

3.3 Water Pollution

Polluted water may cause health risks in Bangkok by several means. Most obviously, risks can arise when individuals drink contaminated ground or surface water. The contamination may derive from the raw water supply, it may be added during the treatment process, or it may enter inadvertently in the water distribution system or as water is stored by the customer. Health risks may also arise from direct contact with contaminated water while individuals are bathing, swimming or washing. Water pollution may also cause human health risks through several less direct pathways, including consuming food crops irrigated with contaminated water, or consuming fish that have bioaccumulated pollutants while living in contaminated water. Each of these different risk pathways has been investigated separately. Table 3.2 summarizes the findings. Appendix B describes in detail the data, calculations and assumptions underlying the water pollution health risk estimates.

Surface Water as a Source of Drinking Water

The Chao Phraya River through Bangkok and the associated klongs are highly polluted, with very low levels of dissolved oxygen, high bacteriological contamination, substantial levels of toxic pollutants such as pesticides and heavy metals, and extensive floating garbage. To avoid this pollution, most of Bangkok's drinking water supply is drawn from the Chao Phraya by the Metropolitan Water Authority (MWA) about 40 km north of the city where water quality is much better. The MWA serves 75% of Bangkok's population with piped water, of which 95% derives from the upstream Chao Phraya intake. MWA's treatment plants for this water are relatively modern, providing treatment that is quite adequate and better than in many U.S. cities.

TABLE 3.2 WATER POLLUTION

Issue	Data Obtained	Risk Analysis Procedure	Adverse Health Effects	Notes
Surface water as drinking water source	Very limited sampling data at the tap Concentrations of contaminants in Chao Phraya intake	Assumed no removal of toxic contaminants in raw water by treatment plant	About 3 cancers/year Other contaminants < 5% of RfD Effects from lead may be substantial; see Lead findings	No data on disinfection byproducts; could add as many as a hundred more cancers
Contamination of drinking water in distribution system or at point of use		Important issue -- see Microbiological Disease and Lead findings		Infiltration of water containing microbiological agents into distribution system Leaching of lead
Ground water as drinking water source	Concentrations of contaminants in groundwater at locations around Bangkok	Assume no removal	No cancers Other contaminants < 10% of RfD	Man-made contaminants unlikely to affect Bangkok groundwater because of impervious clay layers
Direct contact with contaminated water	Concentrations in Chao Phraya and klongs	Compare with guidelines for safe contact	Risk of microbiological disease; concentrations exceed guidelines	Population at risk includes 121,000 residents in canal houses, those living along the river, and others during flooding
Irrigation of crops with contaminated water	Concentrations in river, ground water, and klongs	Compare with guidelines	None from chemicals; concentrations do not exceed guidelines	Microbiological contaminants may be a problem; see Food Contamination and Microbiological Disease findings
Consumption of contaminated fish and shellfish	Concentrations of metals and pesticides in fish and shellfish and Thai consumption of these foods	Assume no removal in food preparation	About .2 cancers/yr	See Food Contamination also No data on microbiological contamination
Protection of aquatic life, economic and aesthetic losses				Not within scope of this project Water quality is far worse than that necessary to support healthy aquatic communities

75% of population served by treated, piped water from MWA, 95% from river intake about 40 km upstream of Bangkok. Treatment is conventional and better than that in many U.S. cities. Remainder of population is served by private wells, typically untreated, or not served at all.

We calculated the risks to the portion of Bangkok's population that drinks this water, assuming that the water they drink is of the same quality as that leaving the treatment plants. (For the moment, in this portion of the risk assessment, we ignore contamination that enters drinking water in the distribution system, subsequent to treatment.) Making a highly conservative assumption that the MWA's treatment plants remove no toxic or heavy metal contaminants from the raw intake water, drinking this water is estimated to result in the following health effects:

o Up to 3 excess cancer cases per year, from carcinogenic pesticides.

o Trivial risks of non-carcinogenic health effects from most other contaminants such as metals. All such contaminants in drinking water occur at levels likely to contribute at most 5% of their RfDs.

o Lead constitutes the one exception. Lead concentrations average 20 - 50 ug/l, relative to the Thai and U.S. drinking water standards of 50 ug/l. Recent evidence indicates adverse effects from lead in drinking water at much lower levels. Consequently, the U.S. is in the process of lowering its standard, perhaps to 5 ug/l. A discussion of the health effects of lead in Bangkok's drinking water is included in a subsequent section of this chapter, which reports all the findings for lead.

o Data were not available for several classes of pollutants in drinking water supplied by the MWA. The one class of pollutants from which we might expect appreciable health risks is disinfection byproducts. Judging from risk analyses in U.S. cities with similar characteristics to Bangkok (similar chlorination treatment processes, and similar high levels of organic materials in the raw water), disinfection byproducts might cause up to a hundred excess cancer cases per year among the Bangkok population drinking the water.

Contamination of Drinking Water in the Distribution System or at the Point of Use

This is a serious problem. Bangkok's water supply distribution pipes often go through highly contaminated areas -- klongs, septic fields, backed-up storm drains, etc. -- and infiltration of external water from these areas through holes in the pipes introduces disease-causing microbiological agents into the drinking water. This infiltration is prompted by low water pressure in the water distribution system; MWA is unable to provide sufficient water to keep up with demand and water pressure falls. In some areas, the water in distribution pipes is under negative rather than positive pressure, as homeowners attach suction pumps to their water pipes to draw water to their home. Bangkok residents are quite aware of the frequent microbiological contamination of their tap water, inspiring many to purchase bottled water or boil tap water for drinking. They may still be exposed to waterborne disease agents through bathing or washing food, containers and utensils in contaminated water. Those who cannot afford to boil water or buy bottled water remain at substantial risk of contracting microbiological diseases.

Erratic water pressure and occasionally limited supplies also induce some Bangkok residents to store tap water or collect rain water, often in open vessels ("klong jars"). These vessels may provide a breeding ground for more microbiological agents and some disease vectors such as mosquitos.

The health risks from microbiological agents entering water distribution or storage equipment are undoubtedly large, but they cannot be estimated separately. A subsequent section of this chapter evaluates the risks of microbiological disease in Bangkok from environmental sources. It is impossible to apportion the total incidence of microbiological diseases among specific causes such as contaminated water, lack of sewage treatment, or poor personal hygiene practices. The rate at which microbiological diseases are transmitted depends on a combination of several factors, and contaminated water is only one of them.

Another pollutant likely to enter the drinking water supply through the distribution system is lead corroded from lead pipes and solder. Again, risks from lead are discussed in a separate section.

Ground Water as a Source of Drinking Water

Up to 25% of Bangkok's population may rely on ground water for their drinking water supply. The MWA has been phasing out the use of ground water for public water supply, because of land subsidence and worsened flooding resulting from excessive ground-water pumping. However, private and unlicensed withdrawal of ground water is substantial, and appears to be increasing. The combined effect of MWA and private actions appears to be a net reduction of ground-water pumping in the city, and a reduction in land subsidence.

We obtained limited data on ground-water quality at several locations outside of Bangkok. Minimal health risks were projected from consuming this water, but the available data did not cover several key classes of potential contaminants such as pesticides and radionuclides. On the whole we expect that Bangkok's ground water is sufficiently clean to result in low health risks, because impervious clay layers protect the ground water from man-made contamination from the surface. In some areas, though, improperly constructed or abandoned wells might provide a conduit for contamination to reach from the surface to ground-water aquifers.

Direct Contact With Contaminated Water

Swimming, bathing or washing articles in Chao Phraya and klong water may cause microbiological disease among those doing so. Concentrations of bacteria in these waters vary widely across different times and locations (particularly with proximity to sources of human waste). Many samples of river and klong water have drastically exceeded generally accepted guideline levels for direct contact, while many other samples are within the guidelines. Risks of microbiological disease from direct contact, like those from pathogens entering water distribution systems, also cannot be estimated separately from those due to other causes of microbiological disease. The risks from direct contact with contaminated surface water are greatest among those living in canal houses (perhaps 120,000 people) or along the river.

Irrigation of Crops With Contaminated Water

Concentrations of chemicals in river and klong water do not exceed guidelines for safe irrigation of food crops. However, no guidelines exist for bacterial contaminants. Concentrations of microbiological agents in river and klong waters are often substantial, and crop irrigation with this water may also contribute to the overall incidence of microbiological diseases in Bangkok.

Consumption of Contaminated Fish and Shellfish

Data on pesticide and heavy metal concentrations in marine life consumed by the average resident of Thailand were analyzed. Assuming the diet of Bangkok residents is similar, 0.2 excess annual cancer cases are projected to occur among Bangkok's population from pesticide residues in fish and shellfish. Consumption of fish and shellfish also contributes minimally to metal intake by residents of Thailand. Bacteriological contamination was not analyzed, but food poisoning commonly results from eating fish or shellfish taken from waters such as the Gulf of Thailand downstream of raw sewage discharges.

Other Impacts of Contaminated Surface Water

Although such effects are not within the scope of this project, it should be noted that the quality of Chao Phraya and klong waters are far worse than that necessary to support healthy aquatic ecosystems. Adverse ecological impacts from pollution of the Chao Phraya seem also to be extending to the marine waters of the Gulf of Thailand. Surface water pollution probably causes significant economic (to tourism, for example) and aesthetic (odors, unsightly floating trash) damages also. The health risks we analyze in this project may be less important than several other types of impacts from pollution of surface waters in Bangkok.

3.4 Food Contamination

The food consumed by Bangkok residents may be contaminated by several types of

substances from environmental sources that can cause health risks.[7] These include pesticides, metals, and microbiological agents. Microbiological contamination of food is covered generally in the discussion of microbiological diseases; in this section we discuss the findings regarding pesticides and metals. A summary of our findings with regard to health risks from food contamination in Bangkok is provided as Table 3.3. Appendix C discusses the data, calculations and findings for food contamination in more detail.

When pesticides are applied to crops in the field, residues of the pesticides may remain on the crop surface or may be incorporated systemically into the plant. Pesticides may also be applied to crops after harvest to prevent spoiling during transport and storage. Metal contamination of food may result from plant uptake of metals from the environment, from the application of pesticides containing metals, from processing and/or canning of foods, and from deposition of airborne metals on exposed foods in transport or storage. Some plants, fish and livestock can bioaccumulate pesticides and metals, concentrating them to levels beyond those at which they occur in the environment.

We obtained information on the concentrations of several dozen pesticides and metals

[7]. In this project we were not concerned with food contamination from non-environmental sources. We thus exclude from consideration such problems as natural carcinogens in food (e.g., aflatoxin in peanuts), food additives (preservatives, dyes, saccharin) and substances produced when foods are cooked (such as products of incomplete combustion from charcoal grilling).

TABLE 3.3 FOOD CONTAMINATION

Contaminants	Data Acquired	Adverse Health Effects	Notes
Pesticides	Residue levels in foods Average Thai consumption of different foods	14 cancers/yr	Nearly all from dieldrin, DDT, BHC. All have been banned. Levels now probably lower. Risks from meat, milk, oils.
Metals	Concentrations in foods Average Thai consumption of different foods	Lead at 97% of RfD 4 other metals at 9 - 37% of RfD Arsenic at 120% of RfD, but health data are suspect	Risks mostly from grains Sources uncertain
Microbiological	---------- Covered generally under microbiological disease ----------		
Natural, additives, from cooking, etc.	---------- Not within the scope of this project ----------		

in samples of Thai foodstuffs. A profile of the average Thai diet was also obtained. The food contamination and consumption data were combined to estimate the daily average dose of each contaminant ingested in food by the average resident of Thailand, and risks were calculated. We estimate that:

o Pesticide residues in food cause about 14 excess cancer cases annually in Bangkok's population. Nearly all of this risk is attributed to dieldrin, DDT and BHC, the sale of which was banned in Thailand in 1988, 1983, and 1980, respectively. Residue levels for these pesticides have probably declined since 1982 - 1985, when our data were generated. Levels in meat, milk and oils are primarily responsible for this risk.

o Four metals are present in foods at levels accounting for 9 - 37% of their RfDs. Lead, a fifth metal, is present at a level accounting for 97% of its RfD. Arsenic is present at a calculated 120% of its RfD, but for technical reasons this RfD is suspect. EPA is debating whether arsenic at low levels should be regarded as an essential human nutrient rather than a toxic substance, and the RfD for arsenic may be revised. Grains and flour appear to be the food commodities contributing the bulk of the dose of metals.

Despite several uncertainties in the data underlying these calculations (e.g., typically a food as consumed has lower contaminant levels than the same food when sampled because of washing, peeling or cooking; possible differences between food and diet for all of Thailand vs. Bangkok) we are reasonably confident that these estimates portray the general magnitude of risks from pesticides and metals in food. Pesticide residues appear to pose minor health risks. Metals may pose more substantial risks, but several questions need more investigation:

o How significant are the health effects that the RfDs for metals are intended to guard against?

o What safety margins are built into these RfDs?

o How much do routes other than food consumption contribute toward the RfDs for these metals?

o What sources are responsible for the seemingly high human exposure to metals?

3.5 Solid and Hazardous Wastes

About 80% of Bangkok's solid waste is collected and dumped at one of three large dump sites. Health risks can result from the uncollected solid waste (contributing generally to rodent and insect populations associated with microbiological diseases), and from conditions at the dump sites. Scavengers at the dump sites and nearby residents can suffer health damages from direct contact with toxic substances and disease vectors, and from contamination of air and surface and ground water. Most hazardous wastes in Bangkok are not separated from the remainder of the waste stream, instead they are co-disposed with municipal wastes at the dump sites. One existing dedicated facility now receives a small fraction of Bangkok's hazardous waste. More such facilities are planned. Appendix D discusses the data, calculations and findings for solid and hazardous wastes in detail.

Available data are not sufficient to perform a detailed assessment of risks from disposal of solid and hazardous wastes. Information necessary to do so would include data on the amounts of hazardous constituents in the waste sent to the dumps, engineering information about practices at the dumps, meteorological and hydrogeological information about the dumps' surroundings, and data on the populations at and around the dumps that are potentially exposed to hazardous constituents. Lacking this information, we have been able to perform only several cursory analyses.

Data were available on the concentrations of several important contaminants in leachate from one of the dumps. Assuming relatively little dilution of this leachate before it reaches a point where surface or ground water is used for drinking, we calculate that an individual drinking this contaminated water would face an excess lifetime cancer risk of 5×10^{-4} (i.e., such an

individual would have a 5 in 10,000 chance of contracting cancer over his or her lifetime from this source, in addition to whatever risk of cancer already exists from other sources). Non-carcinogenic metals would contribute less than their RfDs. In general, extensive contamination of water supplies by toxic substances from Bangkok's dumps is unlikely. Surface water supplies for Bangkok are drawn from far upstream of the dumps. Bangkok's ground water aquifers are substantially protected by several thick geological clay layers that will tend to immobilize heavy metals and many organic compounds before they can percolate as far underground as the aquifers.

Air monitoring data for several volatile organic compounds, particulate matter and nitrogen oxides were also available for a community adjoining one of the dumps. The air pollution levels in this community yield an estimated lifetime excess cancer risk of about 1×10^{-4} for the average resident. Risks from particulate matter in the community are substantial, as monitored concentrations are about five times higher than the Thai ambient standard. Nitrogen oxides and non-carcinogenic toxic air pollutants are estimated to cause no health risks.

Another study of individuals making a living scavenging from one of the dumps found a high incidence of several adverse health effects among the scavengers including lower than average results in lung function tests, high rates of respiratory diseases, high rates of infection by helminths, etc..

While these findings suggest at least moderate levels of health risks for those residing on and near Bangkok's dump sites, only a small fraction of Bangkok's population is so exposed. We estimate that perhaps 3,000 laborers, up to 6,000 scavengers, and several thousand more nearby residents might be subject to these levels of risk from collection and disposal of Bangkok's wastes. Nonetheless, the health risks to Bangkok's entire population are unlikely to exceed more than about one excess cancer case annually. In addition, solid and hazardous wastes are likely to contribute a small increment to the incidence of microbiological disease throughout the city.

3.6 Lead and Other Metals

Lead and several other metals are found in the blood, body tissues, hair, and urine of residents of Thailand at levels of toxicological significance. Assuming that residents of Bangkok show the same or higher levels, substantial health risks are likely to result. We cannot be certain which environmental pathways are contributing the bulk of Bangkok residents' exposure to heavy metals, but we can make some reasonable guesses. Our detailed calculations and findings about health risks from lead and other metals are described in Appendix E. A summary of these findings is provided in Table 3.4.

Lead

Adverse effects of lead on the central nervous system have been recognized for centuries and studied in great detail in the last few decades. Direct monitoring of an individuals' exposure to lead through inhalation and ingestion is difficult, but internal measures of exposure to lead can be obtained by sampling any of several biological tissues. The most common measure of exposure is the concentration of lead in the blood. Several large statistical studies have correlated blood lead levels with the incidence of various adverse health effects. We applied these relationships to blood lead concentrations among Thai residents to project health risks in Bangkok.

Reports by different researchers of average Thai blood lead levels vary from 16 to 45 micrograms per deciliter of whole blood. The highest level is reported in the most recent reference; this may be an indication of increasing lead concentrations or it may be explainable in some other manner. Because of this uncertainty about actual average blood lead levels, we provide both high and low estimates of the resulting health effects. Only some of the better studied health effects of lead are estimated; these include the effects of

TABLE 3.4 LEAD AND OTHER METALS

Pollutant	Levels in Body	Adverse Health Effects	Notes
Lead	Thai blood lead levels: 16 - 45 ug/dl, depending on source U.S. blood lead levels: 15 ug/dl in 1978; 4 - 8 ug/dl now, after phasedown of lead in gasoline	Adult males: 200,000 - 500,000 cases hypertension/yr 300 - 800 cases heart attack and stroke/yr 200 - 400 deaths/yr Children: 500 - 60,000 children/yr need immediate medical attention 400,000 - 700,000 total points IQ lost/yr	Sources of lead exposure: air, water, and food all contribute substantially
Other Metals			
Manganese and Cadmium	Levels in blood and hair: 1. Exceed levels of toxicological significance 2. Equal or exceed levels from highly exposed occupational settings elsewhere	Various neurological and renal effects	Sources uncertain
Mercury	Levels are well below those of toxicological significance		Data questionable

lead on blood pressure, heart disease and death in adult males, and on neurological development and the need for medical treatment among children. Some research has linked lead to several other health effects not included here, such as adverse effects on the developing fetus and possible increases in infant mortality, and impacts on exposed women.

The lowest estimate of Thai average blood lead levels (16 ug/dl) resembles average U.S. levels of blood lead in 1978, well before the U.S. reduction of lead in gasoline. U.S. average blood lead levels are now about 4 - 8 ug/dl. Because of difficulties in extrapolating health effects studies to blood lead levels below these, we estimate the health risks from lead in Bangkok only to the extent that lead levels in Bangkok exceed those currently prevailing in the U.S. Or, equivalently, we estimate the health benefits if blood lead concentrations in Bangkok could be reduced to levels comparable to those in the U.S. now.

We estimate that the following health effects would be avoided from such a reduction of blood lead levels in Bangkok:

o Among adult males:	200,000 - 500,000 cases/yr of hypertension
	300 - 900 cases/yr of heart attack and stroke
	200 - 400 deaths/yr
o Among children:	500 - 60,000 individuals/yr needing immediate medical attention if risk of serious brain damage is to be avoided
	400,000 - 700,000 total IQ points lost/yr (or an average of 3 - 5 IQ points lost by each child in Bangkok through the age of seven)

The exact sources of human exposure to lead in Bangkok are uncertain. Our calculations suggest that each of three sources -- ambient air, water and food -- is likely to be important for both children and adults. Occupational exposures and ingestion of soil and lead-based paint by

children are two additional potentially important routes that we did not investigate in our analysis.

The likely contributors to lead in ambient air are combustion of leaded gasoline and lead smelting. Limits on lead in gasoline in Thailand have declined from .84 g/l before 1984 to .45 g/l from 1985 to the present. A further reduction to .15 g/l is scheduled over 1991 to 1994. By contrast, lead in U.S. gasoline was at .29 g/l in 1984, .1 g/l in 1986, and .03 g/l now. Five of the seven lead smelting plants in Thailand are in Bangkok.

Lead in Bangkok's drinking water is probably derived from several sources: contamination of the raw water supply, deposition of airborne lead into the canals conveying the raw water to the treatment plants, leaching of lead from distribution pipes and solder, and deposition into uncovered water storage containers.

Lead levels in food consumed in the average Thai diet are surprisingly high. Lead in food alone amounts to 97% of the RfD for the substance. Our analysis suggests that food may be the primary pathway for human exposure to lead. How the lead gets into the food is uncertain. There are numerous possibilities, including: deposition of airborne lead onto soil and plant surfaces, naturally high soil lead content, irrigation of crops with contaminated water, deposition of lead onto food sold at roadside markets, or various sources in the food transport and processing system (e.g., from canning). We have no definitive information on the relative importance of these possibilities. We suspect that the combination of heavy traffic, substantial lead content in gasoline, and extensive consumption of food from road-side food stalls may be particularly important. Determining the major sources of lead in food is of high priority for further research.

Other Metals

Data from 1980 were obtained on the average levels of three other metals -- manganese, cadmium and mercury -- in blood, urine and hair of residents of Thailand. For manganese and cadmium, these levels were surprisingly high, even exceeding levels found elsewhere in studies

of individuals that were highly exposed to these metals in occupational settings (e.g., workers at smelters, battery and chemical plants). The average Thai levels also appear to exceed thresholds beyond which adverse health impacts have been observed in statistical studies in other countries. By contrast, the levels of mercury in residents of Thailand were well below the threshold for concern. (In fact, the reported mercury levels were so far below levels observed in other countries that we question whether the units of measurement for the Thai study have been correctly reported.)

These data on other metals raise both concerns and questions. Sufficient data are not available to estimate the number and type of adverse health effects that might result from these concentrations of metals. If, however, the general Thai population shows levels of these contaminants comparable to or greater than highly exposed workers, there is cause for worry. We do not know how the 1980 nationwide data would relate to current data for Bangkok residents. Because of the concentration of industrial activity in or near Bangkok, concentrations of these contaminants in Bangkok residents are likely to be higher than for the country as a whole. In addition, rapid industrial growth over the last 10 years is likely to have led to further elevation of these concentrations. This trend may be offset, however, by increased efforts to reduce environmental contamination.

We have little information on the sources of these metals, or the pathways through which exposure occurs. Analysis of contaminants in the average Thai diet (see the discussion of food contamination in a subsequent section) suggests that cadmium and mercury are present in food at levels contributing 33% and 30% of their RfDs, respectively. We surmise that smelters, battery manufacturers and other metals-related industries may be responsible for much of the releases of these metals into the environment.

3.7 Microbiological Diseases

There are substantial health risks in Bangkok from diseases that are related to environmental pollution and caused by microbiological agents. The diseases include acute diarrhea, dysentery, cholera, dengue fever, and many others. The agents responsible for the

diseases include bacteria, protozoa, viruses, and helminths (worms). A summary of our findings with regard to health risks from these diseases in Bangkok is provided in Table 3.5. Appendix F discusses the data, calculations and findings for environmentally-related microbiological diseases in more detail.

The incidence of these diseases is related to poverty, poor sanitation, poor housing, malnutrition, limited water supplies, lack of sewage disposal and treatment, and inadequate health care and education. Some of these contributing factors are associated with environmental pollution and are thus relevant to this project (e.g., lack of sewage treatment). For this reason, we are including some microbiological diseases among the health risks caused by environmental problems in Bangkok. However, many other factors contributing to these diseases are beyond the scope of this project (e.g., malnutrition). We evaluated the microbiological diseases that are common in Bangkok, and selected 14 for evaluation that are most closely related to environmental pollution. The microbiological diseases we have termed "environmentally related" include:

- o Acute diarrhea
- o Dysentery
- o Enteric fever (typhoid, paratyphoid)
- o Encephalitis
- o Tetanus
- o Acute poliomyelitis
- o Typhus and other rickettsioses
- o Dengue fever
- o Malaria
- o Cholera
- o Hepatitis A
- o Rabies
- o Leptospirosis
- o Helminthiases

These diseases in total are responsible for about 6% of the deaths in Bangkok. We estimate that there are about 850,000 - 1,700,000 cases per year of these diseases among Bangkok's population. This estimate is quite uncertain. We obtained data on the number of cases of these diseases among Bangkok residents reported by hospitals and clinics. We then scaled up this estimate to account for under-reporting. It is widely known that

TABLE 3.5 MICROBIOLOGICAL DISEASES

Key microbiological diseases that are environmentally related:

acute diarrhea	dengue fever
dysentery	malaria
enteric fever (typhoid, paratyphoid)	cholera
encephalitis	hepatitis A
tetanus	rabies
acute poliomyelitis	leptospirosis
typhus and other rickettsioses	helmlathiases

These disease are responsible for: 6% of deaths in Bankok
850,000 - 1,700,000 estimated cases/yr

Primary routes of transmission:
1. Human fecal to oral
2. Vectors (mosquitos, rats, flies)

Environmental factors in disease transmission:

Lack of water	More important
Lack of sewage conveyance	More important
Contaminated water	Important
Lack of sewage treatment	Important
Uncollected solid waste	Less important
Flooding	Less important

Non-environmental factors in disease transmisssion:

Poor personal hygiene
Inadequate health care and education
Lack of / non-use of toilets
Overcrowding and poor housing
Poor nutrition and food preparation

The "non-environmental" factors are as or more important than the environmental factors. In comparing the health risks from different environmental problems, not all the estimated cases of microbiological disease should be attributed to environmental causes.

individuals contracting less severe diseases, such as most of these, typically let their illnesses run their course or treat themselves with medicine purchased at a pharmacy, rather than seek a form of medical attention that will get them counted in the reported statistics. We used an estimate that only 10 - 20% of those ill with these diseases will seek medical attention; thus there are 5 - 10 times as many cases in reality as are counted in the health statistics. Uncertainty about this under-reporting factor is primarily responsible for the uncertainty of our overall estimate of incidence.

Environmental conditions in Bangkok with which we are concerned in this project can contribute to the spread of these diseases in important ways:

o Lack of water. Having a reliable water supply available in the home for bathing, washing and drinking is a key preventive measure for these diseases.

o Contaminated water. Many of the microbiological agents can live in water and are commonly transmitted when contaminated water is used for drinking or bathing.

o Lack of sewage conveyance. The bulk of the microbiological diseases of concern are transmitted by the fecal to oral pathway. This pathway can be broken by assuring that human excrement is conveyed away from where people are likely to come into contact with it.

o Lack of sewage treatment. Appropriate treatment of sewage, in a sewage treatment plant or in septic tanks and cesspools under proper loading and soil conditions, will remove nearly all of the harmful microbiological agents from waste water. After such treatment, waste water can then be safely discharged into waterways to which humans and animals will be exposed.

o Uncollected solid waste. About 80% of Bangkok's solid waste is collected and either scavenged or taken to one of three dump sites. The uncollected remainder can provide food and breeding ground for vectors such as rats and flies involved in transmission of

disease. Uncollected solid waste may also clog storm drains, contributing to accumulation of fecal matter and waste water, exacerbating flooding, and providing pools of stagnant water for mosquito breeding.

o Flooding. Periodic flooding in Bangkok can bring fecal material and people into closer contact. Klongs and storm drains that normally convey sewage away can back up and overflow during floods. Cesspools may overtop with a rising water table.

However, factors that we term "non-environmental", or beyond the scope of this study, are also important in transmission of these microbiological diseases. Critical non-environmental factors include:

o Poor personal hygiene. Cleaning, washing, wiping after defecating, keeping flies and mosquitos away, wearing shoes, etc. are all important.

o Inadequate health care and education. Several of these diseases may be prevented by immunization, and all can be treated effectively. Better knowledge about which practices to avoid and which to emphasize would be helpful. Street food vendors, for example, often wash their utensils and dishes quickly in water before use by another customer. The water is often contaminated, and it might be better instead to wipe the materials with a more sanitary and disposable paper towel.

o Lack of/non-use of toilets. Again, it is important to keep fecal material away from people. Lack of toilets or indiscriminate defecation, typically by children, either in the home or outside, can be a problem.

o Overcrowding and poor housing. Close living conditions increase person-to-person disease transmission. Lack of running water, toilets, screens on windows, refrigeration and garbage disposal facilities will all increase disease incidence.

o Poor nutrition and food preparation. Malnutrition makes individuals more susceptible to many of the microbiological diseases, or increases the severity of the diseases once they are contracted. Improper food preparation -- leaving foods out and exposed, insufficient cooking, etc., -- can contribute to many problems.

By reviewing the characteristics of each disease (e.g., typical pathways, ability of the agent responsible to survive outside the host, minimum infective dose), it is possible to estimate qualitatively the likely contribution of the various environmental and non-environmental factors to transmission and persistence of the disease. We conclude that non-environmental causes are as or more important than environmental causes for these diseases. In comparing the health risks posed by Bangkok's different environmental problems, we will not consider all cases of microbiological diseases as environmentally caused.

The most important environmental factors in preventing these diseases are probably: (1) Providing sufficient, reliable water to the population to support washing and other sanitary practices; and (2) Providing means -- toilets and sanitary sewers -- to get human excrement away from the immediate human environment. These approaches are highly effective in reducing the incidence of a broad range of microbiological diseases.

Other environmental factors are less critical, though still important. The quality of the water supplied is less important than having a reliable water supply in the first place. Treating human excreta properly (e.g., in sewage treatment plants) is less important than conveying it away from people. Although water quality and sewage treatment are not vital in controlling most types of microbiological disease, they are of significant value in reducing the traditional water-borne bacterial diseases (cholera, typhoid, shigella) and some helminth-related diseases, respectively.

More complete collection and better disposal of household refuse is another environmental factor that can be of value in reducing incidence of several vector-related diseases (those relating to rats and insects such as flies and cockroaches that can feed on garbage). We estimate this

value as rather low. Better drainage and reduced flooding can reduce the incidence of mosquito-related diseases such as malaria, dengue and bancroftian filariasis.

4. Methodology

4.1 General Approach

Several recent studies have analyzed environmental conditions in Thailand generally and in Bangkok particularly. Perhaps the most comprehensive is the Thailand Natural Resources Profile, prepared by the Thailand Development Research Institute in January, 1987 (TDRI, 1987).[8] This study reviews the information developed in previous studies and analyzes it in a different way in order explicitly to compare the various environmental health problems in Bangkok. Which environmental problems facing Bangkok are the most serious?

One might think that there is no objective way to compare the disparate environmental problems facing an area such as Bangkok. Consider just two of Bangkok's problems: air pollution from motor vehicles and untreated human waste. The pollutants associated with these two problems are different, the environmental media they affect are different, and the adverse health effects they can produce are different. How can one find a common denominator by which they may be compared?

For the past several years, the USEPA has been considering such issues in an attempt to improve the basis on which the U.S. sets its national environmental priorities. After participating in numerous projects to develop analytical support for establishing environmental priorities, USEPA has developed a technique known as "Comparative Risk Analysis". The results of U.S. comparative risk analysis projects have been both surprising and useful (see insert).

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[8]. See also, for example, Cohen, Murray L. op. cit.

Results of U.S. Comparative Risk Analyses

At the national level, USEPA found that several environmental problems to which the country devotes little effort constitute major environmental health threats, while others that are the subject of major Federal environmental programs present relatively small health risks:

- Indoor air pollution from naturally occurring radon, passive smoking and certain consumer products is one of the largest environmental health problems, yet is virtually unregulated.
- By a large margin, USEPA's largest single budget expenditure goes for cleanup of abandoned hazardous waste disposal sites. Yet such sites present relatively low health risks.
- USEPA's major water pollution control programs are nearly all directed at industrial plants and municipal sewage, yet runoff from farms, city streets and other areas constitutes a more significant problem.

USEPA explained this disparity between experts' assessment of the risks posed by environmental problems and the funding priority given to them by the fact that funding priorities are determined largely by the U.S. Congress, reflecting public opinion about the relative importance of different problems. Public opinion, in turn, does not typically focus on scientific and technical aspects of environmental risks. The public seems most concerned instead with qualitative aspects of environmental problems such as whether they involve risks that are commonplace or unusual, and whether or not there is a "villain" (e.g., a polluting industry) to blame for the risks. USEPA has subsequently increased its attention to "risk communication" in an effort to reach a common understanding between technical experts and the concerned public in how they perceive and rank environmental risks.

In a series of comparative risk studies for more limited geographic areas (cities, states or regions), the participants found additional results that contradicted what had been the conventional wisdom about various environmental problems. For example:

- Santa Clara County, California, found that substances introduced into public drinking water in the course of treating the water caused much greater health risks than the remaining levels of the contaminants that prompted the treatment in the first place.
- The State of Pennsylvania was surprised to find that health risks from an unregulated air pollutant were much greater than those from a regulated pollutant, even though the ambient concentration of the regulated pollutant exceeded air quality standards throughout more than half of the State.

A recent independent review of the comparative risk process by distinguished scientists in the U.S. supports its use for the critical task of focusing governmental attention on the most promising opportunities for reducing environmental risks.[9] The comparative risk process is now firmly established in the U.S. as the key first step in strategic planning for environmental protection.

There are many possible ways of measuring the relative seriousness of different environmental problems. Or, one might determine which problems involve the most extensive violations of national environmental quality standards. Or one might compare across problems the amounts of pollutants emitted, the ambient concentrations of pollutants, or the number of pollution sources in or out of compliance with pollution control requirements, for example.

[9]. Science Advisory Board to the U.S. Environmental Protection Agency. Reducing Risk: Setting Priorities and Strategies for Environmental Protection. September, 1990.

However, none of these measures capture the ultimate impacts of the problems. One does not build and operate treatment plants for domestic sewage merely to reduce biochemical oxygen demand and bacterial discharges to waterways or to improve the quality of receiving water -- one does it so that fewer people will get sick from coming in contract with human waste or from using contaminated water, and so that aquatic ecosystems will be healthier. The reason one takes pollution control actions is to prevent and abate the ultimate adverse impacts to humans and the environment caused by the environmental problems. The term "risk" is used to encompass the probability, magnitude and severity of these ultimate impacts.

Analysts often consider several varieties of risks: to human health, to ecological systems, and to economic welfare. For this project, we focus exclusively on the human health risks caused by environmental problems in Bangkok. Although ecological and welfare risks are certainly important, we have concentrated in this initial study on human health as the value of concern. In a separate effort, USAID is supporting a preliminary investigation of some of the damages to economic welfare caused by environmental problems in Thailand.[10]

The human health risks caused by environmental problems can be thought of as the cases of human disease or injury resulting from exposure to adverse environmental conditions. The range of adverse health effects that can be caused by environmental problems is very broad, including gastrointestinal disease (from pathogens in drinking water), angina pain (from carbon monoxide), learning disabilities (from exposure to lead), cancer (from chronic exposure to many carcinogenic substances) and many more. Our approach to judging the relative importance of Bangkok's environmental health problems was to:

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[10]. Contract in process with Research Triangle Institute to assess the economic losses to tourism from environmental pollution in Thailand. Note that this project, in estimating the number of adverse health effects in Bangkok from environmental pollution, will also provide information useful in assessing welfare risks. Analysts could convert our estimates of adverse health effects into estimates of economic costs by multiplying by appropriate factors to reflect the medical cost of treatment for these health effects and the loss of productivity from absences from work.

1. For each problem, estimate the number and severity of cases of disease or injury caused among Bangkok's population. (For some pollutants, knowledge about the human health effects they may produce is not yet sufficiently advanced to allow estimation of the number of cases likely to result. In such instances, we can only estimate the number of people in Bangkok that are exposed to the pollutant in amounts exceeding a threshold of concern.)

2. Develop a common denominator that allows one to compare and aggregate across the diverse set of diseases and injuries caused by each problem.

3. Rank the problems in order, based on the health risks caused by each.

In ranking the problems, we considered only the health risks that each causes. In this ranking we did not consider other attributes of the problems such as the cost and technical feasibility of controlling them, public opinion about them, politics, statutory mandates or institutional capabilities for dealing with them. These other factors may be equally or more important than the magnitude of the problems in deciding what should be done about them. However, we believe it is critically important to separate the process of assessing risks from the process of deciding how to manage the risks. Risk assessment is ideally a scientific and objective process performed by technical experts. Risk management, in contrast, is a judgmental process requiring public officials to balance a wide variety of concerns. Separating the two processes improves the objectivity and technical quality of the risk assessment. Obtaining an objective assessment of the risks facing a community is a key precondition to successfully managing those risks. This project concentrates on assessing the health risks posed by environmental problems in Bangkok. At the conclusion of the report, we will also offer some observations about managing these risks. Our focus on risk assessment is based upon our expertise relative to others -- we are experienced in risk assessment, whereas the Thai government and the USAID Mission are far better able to evaluate risk management concerns in Bangkok than are we.

4.2 Environmental Problems to be Analyzed

A first step in the project was to determine the set of environmental problem areas on which the study should focus. Two major issues had to be resolved:

o What scope to cover. Which environmental problems should be considered in the study? What would be left out?

o How to slice the pie. There are many possible ways to divide the universe of environmental problems, for example by pollutants (e.g., benzene, pathogens, pesticides), by sources (e.g., motor vehicles, industrial plants), by media (e.g., air, surface water, food), by pathway (e.g., inhalation, ingestion), by geographical region (e.g., Klong Toey, Lad Prao), or by other factors.

In making decisions on these issues, our primary criterion was to define the problems in a manner such that the results of our analysis and ranking could be translated easily into implications for environmental management. We structured problem definitions to correspond roughly to distinctions among potential environmental control programs. Thus, for example, we thought a comparison of the seriousness of water pollution and air pollution could be quite useful in establishing priorities among control programs. If instead we had defined problem areas basically by pollutant (e.g., benzene vs. cadmium) our ranking results would be much less useful. If we were to find, for example, that benzene in Bangkok poses substantially greater human health risks than does cadmium, we would not have a conclusion of much utility in a policy context. Thailand does not have a benzene or a cadmium program, and we would have little idea what other program to emphasize in order to address one or the other of these pollutants (drinking water treatment? industrial air pollution? ground-water contamination?) without substantial further work. Despite this general preference for defining problems in ways related to potential control programs, we found a few pollutants (lead and two other metals) to involve sufficient health risks to warrant addressing them as separate problems.

Several other criteria were used in deciding to exclude some problems from this analysis. We chose not to analyze:

o Problems that the government was unlikely to be able to do much about. Examples: global climate change, stratospheric ozone depletion.

o Problems for which basic data were unavailable or very difficult to acquire. Examples: indoor air pollution, occupational exposure to toxic substances.

o Problems that we judged to relate more to public or occupational health than to environmental pollution. Examples: malnutrition, food additives, occupational safety.

o Problems that we expected would pose very low human health risks in Bangkok. Example: radiation.

Table 4.1 lists the problems that we included in the study.

Table 4.1 Environmental Problems Covered in Bangkok Study

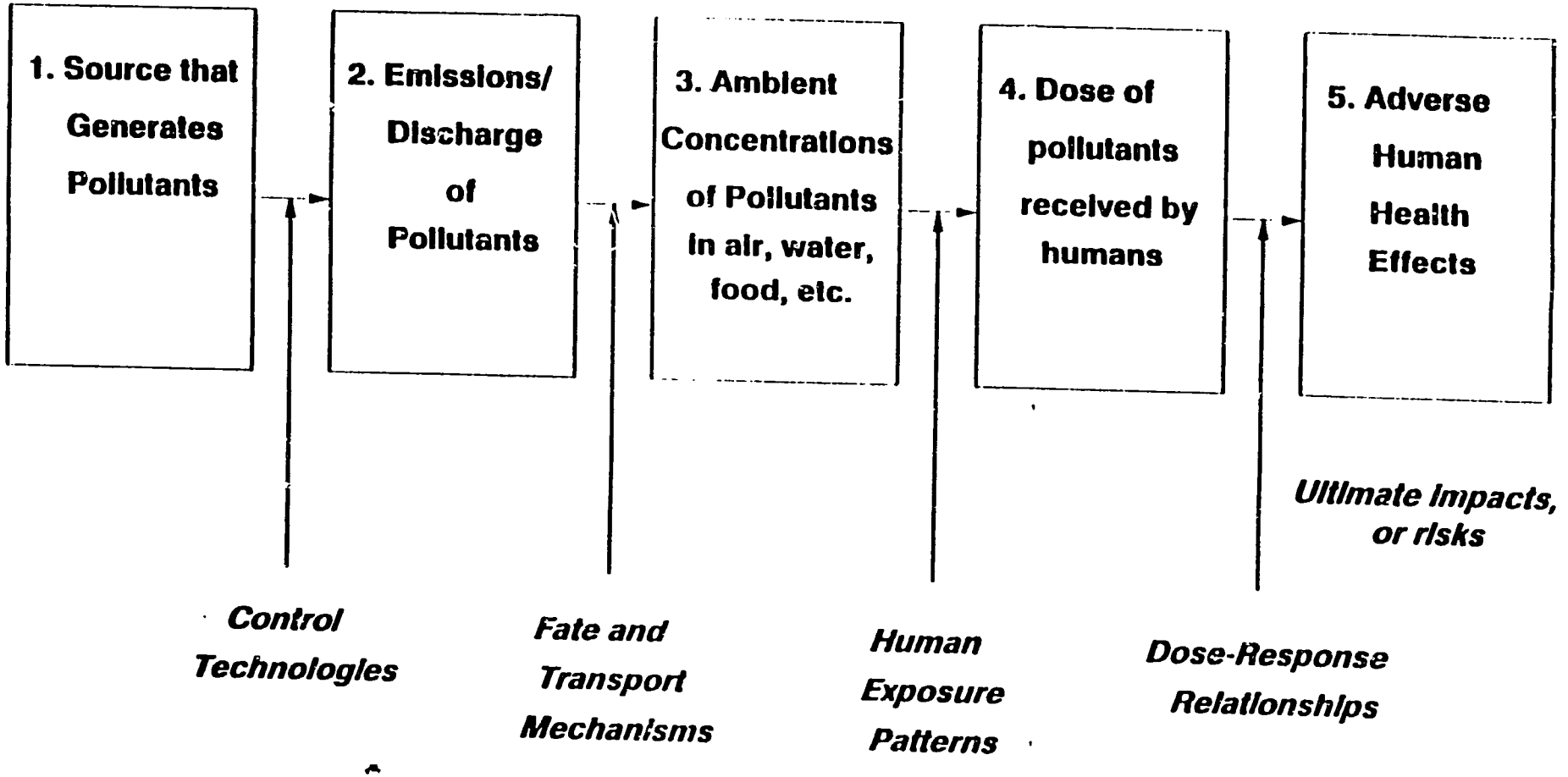
- o Air pollution
  - Criteria air pollutants: particulate matter, carbon monoxide, sulfur dioxide, ozone, nitrogen oxides. (Lead covered separately, in all media)
  - Toxic chemicals
- o Water pollution
  - Contamination of surface water
    - -- Effects on drinking water
    - -- Effects via direct contact, fish consumption, irrigation
  - Contamination of ground water
  - Drinking water treatment
- o Food contamination (pesticides and metals)
- o Solid and hazardous waste disposal
- o Lead and other metals
- o Microbiological disease (Can relate to water supply, human and solid waste disposal, etc.)

4.3 Overview of Risk Assessment Process for Individual Environmental Problems

Most environmental problems cause adverse human health effects through an identical series of steps (see Figure 4.1):

1. A source generates pollutants. For example motor vehicles generate particulate

Figure 4.1 General Process by Which Environmental Problems Create Adverse Human Health Effects



matter, carbon monoxide and several other pollutants as fuels are burned.

2. Some of the pollutants that are generated are emitted or discharged to the environment. The degree to which control technologies are installed and operated determines the fraction of generated pollutants that is emitted or discharged. For example, automobiles with catalytic converters will emit a much lower proportion of the carbon monoxide they generate than will autos without them.

3. Those pollutants that are emitted or discharged undergo various physical and chemical processes in the environment, resulting in ambient concentrations of the pollutants. For example, carbon monoxide is emitted by motor vehicles along roads, and in greatest amounts at busy intersections under congested conditions. Meteorological conditions typically result in rapid dispersion of carbon monoxide at several tens of meters distant from the primary emissions sources. Carbon monoxide concentrations thus tend to be highest at curbside, and they decline rapidly to low levels at a moderate distance from roads.

4. By breathing, drinking, eating and other activities, humans expose themselves to these ambient pollutants. The degree of exposure or dose received by humans depends primarily on where humans are located relative to the higher ambient concentrations of the pollutants. For example, exposure to carbon monoxide will be highest among those living, working or otherwise spending a large fraction of their time on or near heavily trafficked roads.

5. Human exposure to a pollutant produces some likelihood of a resulting adverse health impact. Epidemiological or toxicological studies have established "dose-response relationships" for many pollutants -- mathematical relationships specifying the probability of a human suffering an adverse health effect as a function of the dose of the pollutant he or she has received.

In assessing environmental problems, we are most interested in obtaining data on the ultimate impacts from the problems (Level 5 in Figure 4.1). Data at the fifth level can answer the question of how many cases of adverse human health effects are caused by the problem. Unfortunately, direct measurements of the number of adverse human health effects resulting from environmental causes are rarely available, for two reasons:

o Those who collect and maintain health statistics (e.g., of the number of people afflicted with upper respiratory tract infections, the number of cancer cases) are usually unable to ascertain the cause of each case of disease. Most diseases may be caused by many factors in addition to environmental pollution. Respiratory tract infections may be caused by flu, allergies, the weather and other factors in addition to air pollution. Cancer may be caused by exposure to toxic pollutants, but it can also be linked to diet and hereditary factors. Medical officials are rarely able to estimate accurately the proportion of the total incidence of a disease that should be attributed to environmental pollution.

o Many diseases related to environmental pollution appear only long after the exposure to the pollutant. Many cancers, for example, have latency periods extending to several decades. It would thus be inappropriate to look to health statistics today to indicate the ultimate impacts of today's environmental problems. The impacts of today's problems may take many years to show up. We may not want to wait for this data; we may need to begin now in making decisions about these problems.

Commonly, the best we can do in estimating the health effects from an environmental problem is to obtain data relating to the problem at one of the earlier levels and manipulate it analytically to generate an estimate of likely health risks. Most of the work in this project has involved gathering data on pollution sources, emissions, ambient concentrations and/or exposures associated with each environmental problem, and analyzing the data so as to generate estimates of the resulting number of adverse health effects. Three examples follow showing how we undertake this analytical process for different environmental problems.

Environmental Problem: *Exposure to carbon monoxide in ambient air*
Analytical Approach: *Use data beginning with Level 3 for risk estimation*

Carbon monoxide emitted by motor vehicles, fuel combustion and other sources can reduce human capacity for strenuous exercise, can cause headaches, can reduce human ability to concentrate, and, in individuals with coronary heart disease, can increase angina pain. At extreme concentrations carbon monoxide can cause asphyxiation, but such concentrations will not occur in the general environment. Data on the actual incidence of these effects among Bangkok's population are not available. Instead, we can estimate the number of such effects that occur by:

1. Obtaining existing monitoring data on ambient outdoor concentrations of carbon monoxide in Bangkok (corresponding to Step 3 of Figure 4.1);

2. Estimating the number of people in Bangkok exposed to these concentrations and the resulting dose of carbon monoxide they receive (Step 4 of Figure 4.1); and

3. Using established epidemiological relationships to convert the dose estimate into an estimate of the expected number of resulting adverse health effects (Step 5 of Figure 4.1).

Environmental Problem: *Exposure to toxic pollutants in ambient air*
Analytical Approach: *Use data beginning with Level 1 for risk estimation*

Another environmental problem in Bangkok is exposure to ambient toxic air pollutants (e.g., benzene, metals, products of incomplete combustion, etc.). We have not found any ambient air monitoring data on such pollutants in Bangkok. In the absence of ambient data, we can estimate health risks associated with this problem by:

1. Gathering data on the level of activity by the sources of such pollutants in Bangkok (e.g., number of miles traveled by each type of motor vehicle, volume of fuels consumed by households, output of various industrial sectors) (corresponding to Step 1 of Figure 4.1);

2. Estimating emissions of toxic air pollutants from each of these sources by multiplying emission factors for each source (e.g., the number of grams of benzene emitted by a gasoline-powered automobile per mile traveled) by the amount of activity by each source (e.g., the number of vehicle miles traveled by automobiles) (Step 2 of Figure 4.1);

3. Performing simplified dispersion modeling to estimate ambient concentrations of toxic air pollutants resulting from the estimated emissions (Step 3 of Figure 4.1); and

4. Continuing, as with the carbon monoxide example, through estimating exposure, applying dose-response relationships, and estimating the number of resulting adverse health effects (Steps 4 and 5 of Figure 4.1).

Environmental Problem: *Environmentally related microbiological disease*
Analytical Approach: *Use data directly at Level 5*

For a few environmental problems, estimates of the number of resulting health effects may be

directly available. Examples include the number of cases of some sorts of water-borne diseases (typhoid, cholera, etc.) from contaminated water supplies and the number of deaths and injuries from major industrial accidents involving toxic chemicals. Risk analysis for such environmental problems will primarily involve estimating the degree of under- or over-reporting inherent in such statistics. The health statistics on incidence of microbiological disease should be adjusted upward to account for the fact that many cases of such disease are not reported. On the other hand, the health statistics should be adjusted downward to account for the fact that many cases of such disease are caused by non-environmental factors (e.g., poor health education, diet, overcrowding).

4.4 Cautionary Note on USEPA Risk Assessment Methodology

Appendix G describes in substantially more detail this process of human health risk assessment that is the basis of the work for this project. For this project, we have adopted and used the USEPA's methods. There is substantial scientific controversy about many of the technical steps in health risk assessment, and criticism of some of the choices USEPA has made in its approach. For example, in estimating the potency with which a specific chemical is likely to produce adverse health effects in humans exposed to it, researchers must typically extrapolate from the experimental results obtained by exposing animals to high doses of the chemical to situations where humans are exposed to much lower doses. Some reviewers contend that USEPA's statistical procedure for extrapolating to low doses is much too conservative; it overestimates the likelihood of adverse effects from low doses in humans.[11] Another contention is that USEPA's processes for risk assessment involving carcinogenic substances ignores natural anti-carcinogenic defense mechanisms in humans.[12] Another controversial element of USEPA's typical approach is the use of broad ambient monitoring to characterize the levels of contaminants to which humans are exposed. Some analysts believe that this seriously misrepresents the widely varying concentrations of contaminants to which humans are actually exposed in the diverse

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[11]. See the appendix "Regulatory Impact Analysis Guidance," in U.S. Office of Management and Budget, Office of Information and Regulatory Affairs: Regulatory Program of the United States, August 21, 1990.

[12]. Ames, Bruce N. et al.: "Ranking Possible Carcinogenic Hazards," in Science, Volume 236, April 17, 1987, pp 271 - 280.

microenvironments in which people spend their day.[13] These issues give rise to substantial uncertainties in EPA's risk assessment calculations. In general, these methodological uncertainties are much larger and more important than are another set of uncertainties associated with extending U.S. health effects research to Thailand. EPA's risk assessment procedures require data on several factors pertinent to how people are exposed to pollutants: how much people weigh, how long they live, how much food and water they consume, how much air they breathe, etc. Standard EPA assumptions have been developed for each of these quantities, based upon U.S. behavior. In some instances in this project we have applied the U.S.-based assumptions to Thailand, even though they are probably somewhat inaccurate (e.g., we have assumed the average Thai person lives 70 years, as is true of the U.S. population). In other instances, we have obtained data specific to Thailand and have not used the standard U.S. assumption (e.g., we assume an average Thai body weight of 54 kg rather than the typical U.S. assumption of 70 kg). Ultimately, though, inaccuracies of this sort are not very important. The uncertainties inherent in extrapolating from laboratory animals to humans are much more significant than the uncertainties from extrapolating from people in the U.S. to people in Thailand.

We do not believe it is worthwhile, in this project, to go further into a technical evaluation of alternative risk assessment procedures. We have used USEPA's methods, and our conclusions are thus subject to all of the associated uncertainties.

4.5 Analytical Ground Rules Used in Evaluating Health Risks

The major aim in this project is to compare the relative seriousness of different environmental problems in Bangkok. To compare disparate problems fairly, our methods for analyzing each problem individually must be parallel and comparable. Using standard USEPA procedures provides us with many elements of a consistent analytical approach, but several additional ground rules have been adopted. The ground rules are also critical to understanding

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[13]. Smith, Kirk R.: "Air Pollution. Assessing Total Exposure in the United States," in Environment, Volume 30, Number 8, October, 1988.

the scope of the project and how its results may be interpreted.

First, we have mentioned that problems are being compared against one another based upon the seriousness of the health risks they pose. If we were to focus on a different type of risk -- ecological or welfare, for example -- the ranking of the relative importance of the environmental problems would differ. In U.S. comparative risk studies, for example, indoor air pollution and drinking water contamination are problems that have been found consistently to pose high health risks but no ecological risks. Conversely, destruction of wetland areas and oil spills are problems that pose substantial ecological risks but minimal health risks.

Second, we focused on assessing the "residual" health risks associated with each environmental problem. By residual risks we mean the risks that will result from environmental problems as they now are. Residual risks are those remaining despite the environmental controls that are now in place, whether these controls are less than or more than is required by environmental regulations. Several alternatives to focusing on residual risks are imaginable, but we have not chosen them. We have not assessed: 1) Risks that have been abated, or risks as they would have been in the absence of control actions; or 2) Risks that will be abated, or risks as they will be after current requirements are implemented and complied with.

The reason we focused on residual risks was our interest in determining additional steps to address environmental problems. We wanted to assume current controls as the base, ask what risks remain, and what can be done to further reduce them. Our focus on residual risks has several implications for how the risk ranking results should be interpreted:

o An environmental problem might pose low residual risks now for either of two quite different reasons: a) It has always posed low risks, or b) It formerly posed higher risks, but a control program has been successful in reducing the problem to current lower levels. In the latter case, even though the problem now poses low risks, the control program addressing the problem may still be very important because it holds risks to their current low level. Without maintaining the control program, risks might increase to their former

higher level. Because a problem area is low risk does not mean that the control program to deal with that problem area is unimportant.

o In a related vein, the risk rankings can provide some guidance about environmental problems potentially worthy of enhanced control efforts, but no guidance about problems for which control efforts might be relaxed. Residual risks provide a guide to problem areas most in need of further efforts (more investment). They provide no indication of how much risks in any problem area would increase if current controls were dismantled or if current enforcement efforts were reduced.

o A problem area can appear to be high risk now, even though existing laws and regulations will reduce it to low risk when they are complied with.

A third ground rule used to facilitate comparable analyses across problems was to focus on environmental health risks to Bangkok residents, whatever the source of the risk. Many pollutants are persistent in the environment, and they can move to cause risks far from where they are originally generated. Under some meteorological conditions air pollutants emitted in Samut Prakan can cause health risks in Bangkok; under other conditions the reverse is true. Pesticides used on agricultural crops in areas outside of Bangkok cause health risks among Bangkok residents when foods are transported to the metropolis, sold and consumed. Wastewater discharged by Bangkok residents to the Chao Phraya travels downstream and contaminates fish in the Gulf of Thailand that are eaten by residents of other regions. Our approach in this project is to evaluate health risks facing the residents of Bangkok from environmental sources, wherever they are located. It is possible, therefore, that we may find some environmental problem that causes significant health risks in Bangkok, yet that is not easily controllable because it derives from elsewhere.

A final analytical ground rule that we have used in this study is to focus on aggregate health risks across Bangkok's entire population rather than on health risks to particularly severely affected sub-populations (e.g., low income groups, those living in specific geographic zones of

the city). In risk analysis jargon, we are more concerned with population risk than risks to maximally exposed individuals (MEIs). This choice derives from two factors:

o The information available to us on pollutant emissions, concentrations and exposures in Bangkok is not sufficiently disaggregated and detailed to permit a credible analysis of effects on specific geographic or socioeconomic subgroups. The resources available for this screening-level study also will not support the depth of analysis needed for evaluation of MEIs.

o In the U.S. at least, many analysts feel that broad environmental priority-setting should be based upon population risks ("the greatest good for the greatest number"), while high risks to MEIs that are not also high population risks should be taken care of by fine-tuning within individual control programs.

Choosing to focus primarily on population risks has some implications for the results of comparative ranking of environmental problems. A problem that tends to be geographically widespread and thus affects many people (e.g., air pollution) will rank high on a population risk basis. A problem that is localized and affects few people (e.g., contamination from municipal solid waste landfills), even though it may affect them severely, will rank lower on a population risk basis.

4.6 Difficulties in Estimating Health Risks for Environmental Problems

In principle, assessment of the health risks associated with an environmental problem is straightforward. We aim to estimate the levels of pollutants to which Bangkok residents are exposed as a result of the problem, typically by using data on either emissions or ambient concentrations. The number and type of health effects that will result are then projected by applying dose-response relationships to the estimated human exposures. In practice, implementing these steps is very difficult. Major uncertainties and shortcomings arise in both estimating exposures to pollutants and applying dose-response relationships. In this section, we

will discuss some of these limitations.

Perhaps the most obvious limitation involves the amount and quality of human exposure data. Most environmental problems involve numerous pollutants, whose concentration varies substantially over time and space within a city as large as Bangkok. Consider air pollution as an example. Several hundred different contaminants can be found in the air of most major cities. For Bangkok, we have ambient measurements for only six pollutants. The concentrations of the contaminants vary widely over time -- they increase during unfavorable meteorological conditions, and decrease when the wind blows them away or rain washes them out. For Bangkok, we have data that is aggregated across a year: the average, maximum and minimum concentrations observed at a station during that period. The concentrations of the air pollutants also vary by location, typically being highest near factories, streets and other sources of the pollutants. For Bangkok, we have concentration data only for the handful of locations at which air pollution monitors have been operated. The result of our limited data on air pollution in Bangkok is that we must make several tenuous assumptions in order to begin to analyze health risks:

o We must assume something about the numerous other air pollutants for which we have no data. What fraction of the total problem are we likely to have covered with the pollutants for which we do have data? Fortunately, the pollutants for which data are usually available are those that are generally thought to present the greatest risks -- those that are most common and most worrisome. We must draw on our experience from other studies in which a larger set of pollutants were evaluated, and decide how much of the air pollution problem in Bangkok we have captured with the limited set of pollutants for which we have data.

o We assume that the times and places at which the monitoring data were generated are representative; that the average of these data points accurately match the average levels of air pollution actually prevailing in the city.

o We ignore differences across individuals in how they may be exposed to these levels of pollutants. We assume that all Bangkok residents breathe the average levels of pollutants in Bangkok's air for 24 hours per day, every day. We do not take account, for example, of the fact that most people are indoors during varying fractions of the day, where air pollutant concentrations may be different than outdoor averages.

In large scale, in-depth risk analyses, steps can be taken to avoid having to make such assumptions. Additional sampling or monitoring can be conducted to fill the gaps when key data are unavailable. Additional analyses can be done to estimate risks for various specific segments of the population rather than for only the average individual. Due to limited time and resources, this study did not develop either new primary data or exhaustive analyses of any particular problem. We substituted our judgment in many cases for data or analysis that we could not pursue. Therefore, this assessment of the health risks from environmental problems in Bangkok is not completely objective. Our aims in this project have been to:

o Develop objective, quantified results to the extent the available data and time allow;

o Recognize the universal need to supplement the data with judgment;

o Be explicit and open where we are making judgments; and

o Point out how additional data acquisition or analysis in the future might help to resolve uncertainties.

Despite these goals, the fact remains that our information on exposure of people to pollutants is limited and highly uncertain.

A second major uncertainty involves the difficulty in knowing how and to what extent a substance may cause adverse health effects in humans. Even if information on exposures to pollutants were perfect, there would still be great uncertainty in evaluating the toxicological

impact of these exposures. For this project, we use standard USEPA data bases summarizing the health effects and potency of numerous chemical substances. These data have been approved for use by USEPA. However, while they appear definitive, they are far from exact. Health risk assessment for any substance depends partly on "hazard identification" (What adverse health effects does the substance cause in humans?) and "dose-response assessment" (What is the likelihood that the substance will cause the health effect, as a function of intake or exposure?). Data available to scientists for making determinations on these factors are imperfect. Difficult and debatable judgments must be made for each substance in: 1) Interpreting available epidemiological studies on humans or laboratory studies on animals; 2) Extrapolating data from laboratory animals to humans; 3) Extrapolating data on effects at high experimental doses of the substance to effects at low environmental doses; and 4) Estimating the effects of exposure to combinations of toxic substances. Each of these judgments is typically made in an intentionally conservative fashion, so that the final estimates are unlikely to underestimate the true potency of a chemical. The actual potency of a chemical is unlikely to be any higher than the value in the USEPA data base, and it is very likely to be lower.

The result is that most risk assessments represent a plausible worst case. The estimates of health risks from exposure to toxic substances thus should not be interpreted as precise or literal estimates of future health effects. The simplifying assumptions in both the toxicology and the exposure components of a risk assessment are simply too great to justify a high level of confidence in the absolute value of the results. The value of these estimates lies in their usefulness for comparing problems to one another, developing a rough idea of the magnitude of possible effects, and setting priorities for further analysis. Many of the uncertainties in risk assessment are systemic, applying equally to all substances being evaluated. If, for example, we were to conclude that exposure to chemical A in Bangkok causes about 500 cancers per year while exposure to chemical B causes 1 cancer per year, the absolute numbers would mean little. The 500 predicted cancers might in reality turn out to be 50, or even 5. We can be reasonably confident, though, in relative comparisons between chemical A and B -- A will cause a significantly greater number of cancers than B.

4.7 Difficulties in Making Comparisons Across Environmental Problems

Once the adverse health effects associated with the environmental problems have been estimated, problems may be ranked relative to each other by comparing the number and severity of the health effects estimated for each. Several factors raise complications in doing this.

1. Disparate health effects. Different environmental problems cause different health effects, ranging from serious and permanent ones (death, stroke) to mild and transient ones (headaches, restricted activity days, diarrhea). In addition to considering the number of health effects caused by each environmental problem in Bangkok, we must also consider the severity of these effects. We do this through the use of an index that describes the relative severity of different health effects. Our severity index is adapted from one developed by Thailand's National Epidemiology Board, and is shown in Table 4.2. The table lists various common diseases or health effects in Bangkok in descending order of severity. The more severe diseases have higher scores. The scoring criteria are self-explanatory, with the exception of "CFR". This is an abbreviation for the "case to fatality ratio", or the proportion of all cases of the disease that result in death. This severity index is described in more detail in Appendix H.

2. Unquantifiable health effects. For some pollutants, the available information on dose-response relationships is not sufficient to allow us to estimate an expected number of adverse health effects. For these pollutants, we have estimated only the number of people in Bangkok at risk of suffering a health effect. This is the case for all pollutants where doses have been compared with RfDs. For them, we can say that individuals receiving doses less than RfDs face virtually no risk, that individuals receiving doses exceeding RfDs face some risk, and that the more the dose exceeds the RfD the greater the likelihood of an adverse effect. But we cannot quantify the likelihood of the effect for a given level of dose. This problem also exists for carbon monoxide, where we can only calculate the number of people likely to be at risk of effects such as angina pain and headaches.

3. Differing degrees of conservatism in exposure estimates. To compensate for limited

data available on the levels of all pollutants to which residents of Bangkok are exposed from each environmental problem, we have made a great number of assumptions. We realize that these assumptions are not comparable across the problems. For one problem we may make an assumption that is highly conservative -- one that makes it likely that we will overestimate the true risks associated with the problem. For another problem, though, we may make an assumption that is not conservative; that we believe is likely to lead us to underestimate the true risks associated with the problem. It is very difficult to compare the risk estimates for two problems made under such different assumptions.

4. Differing degrees of conservatism in health effects information. We use the USEPA's dose-response information for pollutants. In compiling this information, USEPA

TABLE 4.2 SEVERITY RANKING FOR VARIOUS DISEASES

Rank	Disease	Disability (1-5)	CFR (1-5)	Av. Hosp. Stay (1-5)	Prevent-ability (1-3)	Treat-ability (1-3)	Score
1	Stroke	4	4	5	2	3	18
2	Cancer	4	4	3	3	3	17
3	Assault & Homicide	3	5	3	3	3	17
4	Encephalitis	4	3	4	2	3	16
5	Cirrhosis	3	3	3	3	3	15
6	Mental Illness	4	1	5	2	3	15
7	Drug Addiction	3	1	5	2	3	14
8	Suicide & Attempted	2	5	1	2	3	13
9	Coronary Heary Diseases	3	3	2	2	3	13
10	Diabetes mellitus	2	2	4	3	2	13
11	Traffic Accident	3	2	3	2	2	12
12	Drowning	1	5	1	2	3	12
13	Tuberculosis	2	1	5	2	1	11
14	Tetanus	2	3	3	1	2	11
15	Hypertension	2	2	2	2	2	10
16	Occupational Accident	3	1	2	2	2	10
17	Leprosy	4	1	1	2	2	10
18	Poliomyelitis	1	1	5	1	2	10
19	Diptheria	2	1	4	1	2	10
20	Rabies	1	5	1	1	2	10
21	Malaria	2	1	2	2	1	8
22	Peptic Ulcer	1	1	2	2	2	8
23	Pneumonia	1	1	2	3	1	8
24	Venereal Disease	1	1	3	2	1	8
25	Hepatitis A	1	1	2	3	1	8
26	Intest. Obstructions & Hernia	1	1	2	3	1	8
27	Cholera	1	1	2	3	1	8
28	Pertussis	1	1	2	2	2	8
29	Conjunctivitis	1	1	1	3	1	7
30	Influenza	1	1	1	3	1	7
31	Appendicitis	1	1	1	3	1	7
32	Enteric Fever	1	1	2	2	1	7
33	Leptospirosis	1	2	1	2	1	7
34	Acute Diarrhea	1	1	1	2	1	6
35	Dengue Haemorrhagic	1	1	1	2	1	6
36	Dysentery	1	1	1	2	1	6
37	Measles	1	1	2	1	1	6
38	Typhus	1	1	1	2	1	6
39	Helminthiasis	1	1	1	2	1	6
40	Rubella	1	1	1	1	1	5

Source: Adapted from National Epidemiology Board of Thailand. Review of the Health Situation in Thailand: Priority Ranking of Diseases. 1987.

has generally attempted to avoid the mistake of underestimating the health effects of a chemical. For chemicals with more uncertainty about their health effects, USEPA typically adopts a larger margin of safety. For chemicals whose effects are very well understood (e.g., from extensive epidemiological studies of effects on humans), USEPA is likely to provide a relatively small margin of safety. USEPA's health effects data base does not provide "best guesses" about the potency of each chemical included; instead it provides potency estimates that are not likely to be lower than the true potencies. Different degrees of conservatism are inherent in this data base. This has substantial implications for interpreting the results of our risk calculations for Bangkok. Nearly all of our risk calculations for this project fall into one of the following four categories:

o Cancer risk estimates. When we estimate cancer incidence resulting from pollutants with health effect evidence based on limited animal studies, our estimate is likely to be a very substantial overestimate of the true number of resulting cancers. For example, the number of cancers from pesticides in food is extremely unlikely to be as high as the 14 we estimate using EPA's cancer potency factors; it is more likely to be 1.4 or .14 or even less. The same is true, to a somewhat lesser extent, for cancer risk estimates for pollutants with evidence of carcinogenicity from studies of humans.

o Estimates of non-cancer effects that are based on epidemiological studies of humans. These provide best estimates, by contrast. They are not expected to be biased either high or low. They derive generally from estimating a relationship between a concentration of a pollutant and the incidence of a health effect in the human population exposed to it, with no conservatism intentionally built into the process. The health effects from criteria air pollutants and lead are estimated in this manner for this project.

o Estimates of non-cancer effects that are based on reported health statistics. Typically, reported health statistics should be adjusted to account for possible under or over reporting. They may then be further adjusted by apportioning the estimated cases of a disease among environmental and other causes. There is substantial uncertainty in making such adjustments, but unless an adjustment is intentionally made in a conservative or non-

conservative manner, these are also best estimates.

o Estimates of the number of individuals exposed to a pollutant at levels exceeding that pollutant's RFD. When exposure is at a level only modestly exceeding the RfD (e.g., 1-10 times the RfD), the number of people exposed will greatly overestimate the number of people likely to suffer the health effect. When exposure is much higher than the RfD, the number of people suffering the health effect will approach the number of people exposed.

These four sorts of problems and interpretive difficulties beset all comparative risk analyses. There is typically no quantitative or fully satisfactory corrective solution for them. The general approach is to rely on the judgment of those conducting the study to provide rough and appropriate adjustments to the raw calculations of health effects for each environmental problem.

4.8 Summary on Limitations of Comparative Risk Analysis

In sum, the conclusions in this study about health risks from environmental problems in Bangkok are imprecise. The assumptions and uncertainties in comparative risk analysis techniques -- in exposure assessment, in toxicology, and in comparing estimates across problems -- are substantial. Our analytical results do not constitute absolute, reliable estimates of human health risks. Instead, the results suggest likelihoods: providing rough relative comparisons between environmental problems and giving a general idea of the magnitude of possible effects.

We do not intend this to be a discouraging picture of this analysis, only a realistic one. After all, we would not be applying these methods if we did not believe in them. The USEPA's process of comparative risk analysis is the best tool we are aware of to help in setting environmental priorities. USEPA's process is imperfect, but it represents the state of the art. We believe that decision-makers usually must act promptly and cannot often wait for more scientific certainty about environmental problems. The comparative risk analysis process organizes and uses the best information available today to estimate risks so that decisions that cannot wait will be as informed as possible.

5. Risk Management Measures

In this concluding chapter, we discuss some promising measures for reducing the health risks associated with the higher and medium risk environmental problems in Bangkok. Some of these measures are control strategies, while others involve research to fill gaps in our understanding of the problems. In conformity with the priority ranking, no measures are presented to address the lower risk environmental problems.

The measures discussed in this chapter are ones that we believe show significant promise; but they are not recommendations. We have devoted all of our analytical effort in this project to understanding and assessing the environmental health risks facing the residents of Bangkok. We performed no research on management strategies. As we stated earlier, others (Thai officials and the USAID Mission) are much better qualified than we to evaluate risk management concerns and develop recommendations. We believe that the measures discussed in this chapter are worthy of further investigation by responsible officials, and we make no further claim for them.

5.1 Measures to Address Air Pollution

The Thai government already pursues several policies that, as we understand them, contribute to mitigating air pollution problems in Bangkok. Conscious efforts to direct growth of heavy industry, particularly power plants, to areas outside of the urban core limit the industrial emissions to which the urban population is exposed. Policies to encourage use of LPG and diesel fuels rather than gasoline for transportation are, on balance, probably beneficial. Their effect in reducing lead and carbon monoxide emissions probably outweighs their negative impact from increased particulate emissions.

Perhaps the most beneficial general policy to reduce air pollution while strong economic growth continues would be to encourage more efficient use of energy. Measures directed at all

sectors -- transportation (e.g., better traffic flow, more efficient vehicles), industry (fuel switching, new technologies) and even residential/commercial (more efficient appliances and lighting) -- could be appropriate. Improved energy efficiency would also have substantial economic benefits beyond environmental concerns.

Particulate Matter

The most immediate need in addressing particulate matter air pollution in Bangkok is to develop an emission inventory. Before considering specific control measures, one must know which sources are responsible for the bulk of the emissions. Major source categories include: transportation (diesel and other), power plants, other industry, construction, residential/commercial fuel use and open burning of trash. TDRI (1987) has conducted an assessment of the relative contribution of different source types to emissions for Thailand as a whole, but we expect conditions to be substantially different in Bangkok than in the rural areas of Thailand.

An emission inventory need not involve extensive and costly monitoring. Emissions from a specific source can be estimated by multiplying appropriate emission factors for the source type (several compilations of them are available from USEPA) by a measure of the activity by the source (e.g., the annual tonnage of cement produced by a cement plant, or the number of vehicle miles traveled by automobiles). In generating such an inventory, researchers would have to compile a list of major industrial facilities in and around Bangkok and data on their levels of production. This industrial data would be quite useful for estimating amounts of emissions or effluents for many substances in addition to airborne particulates.

Two additional studies relating to particulates might accompany the emission inventory. The first would be an effort to assess the size distribution of particulates. Fine, small diameter particulates are inhaled more deeply into the lungs than large particulates, and they ultimately cause greater health damage. The contribution of a source type to risk from particulates is a function of both the volume of particulates emitted and their size. Construction activities, for example, are often found responsible for a large fraction of total particulates, but a much smaller

fraction of the smaller respirable particulates. A second particulate study could involve total human exposure methods. Concentrations of particulates often vary widely across different microenvironments in a city (e.g., ambient outdoor air, in a car stuck in traffic, within the home while cooking, etc.). A thorough analysis of exposure to particulates would consider both how much time people spend in each microenvironment and typical concentrations in each location. A total human exposure approach can make a great difference in estimating the relative desirability of alternative measures for reducing risks from airborne particulates.[14]

Traditional control approaches for particulate emissions focus on encouraging industrial sources to install baghouses, cyclones, electrostatic precipitators, or scrubbers. Newer measures that may be relevant to emissions from transportation include desulfurization of diesel fuel (which can yield up to perhaps a 10% reduction in particulate emissions) and particulate traps for diesel vehicles. These two approaches have some attractive institutional features to them: progress can be achieved by focusing on a few refineries or a few owners of large diesel fleets, rather than having to deal with thousands of industrial plants or millions of homes and commercial establishments.

Carbon Monoxide

Continued efforts to fuel a large portion of the vehicle fleet with diesel and LPG rather than gasoline will help. Reducing carbon monoxide emissions can be achieved most easily by combinations of tighter auto emission standards, better enforcement of the standards, and better maintenance of autos. The U.S. has found that a very high proportion of carbon monoxide emissions are caused by a small number of older, highly polluting automobiles. Identifying these gross polluters and repairing or retiring them has substantial benefits. Policies to encourage retirement of old cars (perhaps a high annual registration fee?) and replacement with newer cars subject to much tighter emission controls may be desirable. Strategies addressing fuel

[14]. Kirk R. Smith, "Air Pollution. Assessing Total Exposure in Developing Countries," in Environment, Volume 30, Number 10, December, 1988.

composition (e.g., oxygenated fuels, reformulated gasoline) are being developed in the U.S., but they may require many years of implementation before demonstrating anything more than marginal impacts. Clean fuels technologies are being considered in the U.S. primarily because the autos themselves are already fairly well controlled. For the purposes of controlling carbon monoxide in Thailand, several further steps to improve the vehicle fleet appear sensible before considering reformulation of fuels.

Air Toxics

Although air toxics do not appear to constitute a substantial health threat in Bangkok, the data underlying this assessment are quite limited. The major limitations are: 1) no ambient air toxics sampling representative of the city as a whole seems to have been performed; and 2) no inventory of industrial activity is available that will allow rough calculation of industrial emissions of air toxics. These data gaps could be remedied at modest cost. With sufficient attention to sample design, a useful air toxics sampling effort can be undertaken for less than $100 thousand. (A broad annual monitoring program for air toxics, by contrast, might cost more than ten times this amount.) The inventory of industrial activity previously suggested for use in estimating particulate emissions would serve air toxics purposes also.

5.2 Measures to Address Lead

Residents of Thailand show very high levels of lead, which may even be increasing over time. Different studies report different results, however. A critical first step in dealing with a pollutant that appears to have such substantial likely adverse effects as lead is to understand the nature and source of the problem more fully. We suggest:

- o A substantial blood lead sampling study of Bangkok residents. Investigating the correlation between blood lead levels and socioeconomic characteristics could reveal much about groups with particularly high levels and the likely sources of much of the lead exposure. Our efforts at analyzing lead exposures from air, water and food in

Bangkok indicate that each pathway is significant, but that in total they are still not sufficient to account for average blood lead levels. What is the source of the additional exposure? Possibilities might include childhood ingestion of contaminated soil or lead-based paint, leaded cookware, or occupational exposures. A detailed sampling effort and attempts to correlate the findings with causative factors could provide important information.

o Studies of several particular exposure pathways to determine how lead is contributed in the pathway. The high lead content in Thai food is a particular mystery to us. Sampling of foodstuffs at various points in the chain from production through consumption might reveal the source of the lead. Possibilities might include crop uptake of lead from soils; air deposition of lead on food as it is transported, stored and sold; lead from canning; and lead from curbside dust at markets and vendors. Similarly, the sources of lead in drinking water could be ascertained. Possibilities here include lead leached from storage vessels at residences, air deposition of lead into storage vessels, lead leached from water distribution pipes, lead deposited into the MWA water transmission canals, or lead in the raw water from the Chao Phraya.

o Detailed investigation of industrial plants that are suspected to be major sources of lead emissions. Primary and secondary lead smelters are initial candidates. The investigation would seek to learn what fraction of lead emissions are from industrial, as opposed to transportation sources.

With the exception of lead in gasoline, which appears to be a significant source, major control efforts for lead might await these screening studies. Control measures would be directed at significant sources of human lead exposure. If, for example, lead leached from water distribution pipes was found to be important, measures could be implemented at the MWA treatment plants to reduce the corrosivity of the treated water. The U.S. has found corrosion

control measures to be highly cost-effective in water systems with "aggressive" water.[15]

Lead in gasoline presents a special case. Thailand now allows a lead content of .45 g/l, which is scheduled to be reduced to .15 g/l in 1994. By contrast, the lead level in U.S. gasoline is now .026 g/l, and most of the developed countries and several Asian nations have been at the .15 g/l level for some time. Table 5.1 provides a compilation of standards for lead in gasoline from many countries.[16] Thailand might consider accelerating its schedule for phasing down the lead content of gasoline. The cost of additional refining of crude oil necessary to raise octane levels to replace phased-out lead typically amounts to only several cents per gallon of gasoline. Reducing lead exposure by working with several Thai refineries and several gasoline importers may also be far less institutionally complex than other approaches.

5.3 Measures to Address Microbiological Diseases

Our analysis of the factors contributing to the incidence of microbiological diseases in Bangkok suggests that non-environmental factors are likely to be as or more important than environmental problems. We are not qualified to suggest appropriate measures (e.g., public

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[15]. U.S. Environmental Protection Agency. Reducing Lead in Drinking Water: a Benefit Analysis. Prepared by the Office of Policy, Planning and Evaluation, Draft Final Report, December, 1986.

[16]. Reproduced from Jerome O. Nriagu. "The Rise and Fall of Leaded Gasoline," in The Science of the Total Environment, Volume 92, 1990, p.25.

Table 5.1 Limits on Lead in Gasoline

Country	1984 Pb level ($g\,l^{-1}$)	Pb reduction schedule		Unleaded phase-in date
		Level	Date	
N. America				
U.S.A.	0.29	0.026[a]	1989	1975
Canada	< 0.77	0.29	1987	1975
Canada	< 0.77	0.026	1990	1975
CEC				
Denmark	0.15			1987
Germany	0.15	0.15		1987
Netherlands	0.40	0.15	1986	1987
U.K.	0.40	0.15	1986	1989
Belgium	0.40	0.15	1987	1989
Ireland	0.40	0.15	1989	1989
France	0.40	0.15	1989	1989
Italy	0.40	0.15	1989	1989
Greece	0.40	0.15	1989	1989
Athens	0.15			
Other European countries				
Austria	0.15			1987
Norway	0.15			1987
Sweden	0.15			1987
Switzerland	0.15			1986
East Germany	0.40	0.15	1989	1987
Czechoslovakia	0.40	0.15	1989	1987
Finland	0.40	0.15	1985	1989
Portugal	0.64	0.40	1986	1989
Spain	0.60	0.40	1986	1989
Yugoslavia	0.60	0.40	1989	1989
Asia Pacific/Latin America				
Taiwan	0.30			1985
Hong Kong	0.40	0.15	1987	
New Zealand	0.84	0.40	1987	1987
Singapore	0.40	0.15	1989	1989
Venezuela	0.84	0.29	1987	
South Africa	0.84	0.40	1986	1989
Malaysia	0.84	0.40	1989	
Argentina	0.84	0.40	1988	
Brazil	0.80	0.026	1989	
Australia				
Victoria	0.30			
New South Wales	0.40	(Australian jurisdictions are moving towards use of unleaded fuel)		
South Australia	0.65			
Western Australia, Queensland, Tasmania and Northern Territory	0.84			

[a] Concentration of $0.026\,g\,l^{-1}$ ($0.10\,g\,gal^{-1}$) is generally regarded as being "lead free".

health education, improved medical care) for dealing with the non-environmental factors, and we will not do so in this report. As to environmental measures, we have ranked the general importance of different remedial measures as follows:

Higher Importance	Providing sufficient, reliable water to the population to support bathing and other sanitary practices.
	Providing means -- toilets and sanitary sewers -- to convey human excrement away from the immediate human environment.
Medium Importance	Providing water that meets standards for microbiological contaminants at the point of use.
	Providing proper sewage treatment.
Lower Importance	More complete collection and better disposal of household refuse.
	Better drainage and reduced flooding.

Accordingly, we suggest concentrating efforts on water supply and sewer projects.

MWA should investigate various methods for increasing the amount and reliability of water supplies. An individual water connection for each residence, with reliable water pressure should be the goals. Achieving this will eliminate the need for water storage by customers; it will eliminate the practice of attaching suction pumps at residences (thereby reducing infiltration of contaminants in the distribution system); and it will ultimately improve the quality of the delivered water. For several years MWA has had programs under way to obtain additional supplies of raw water and find and repair leaks in the distribution system. In addition, MWA might consider implementing a range of other programs to encourage efficient use and conservation of water. U.S. water utilities typically find that conservation-oriented rate structures or residential fixture retrofit programs will provide water at much lower unit costs than those for construction of new water supply facilities.

Sewage conveyance and drainage projects seem more important than sewage treatment. The cost estimate for a full sewage treatment system for Bangkok exceeds $1.4 billion at 1980 prices, and it appears unlikely that such funds will be available. Of more immediate benefit might be a series of smaller, incremental projects to construct sanitary sewers for particularly poorly drained sections of the city. Effluents from these sewers might be routed to several relatively inexpensive primary treatment plants, or, in the worst case, discharged directly to the river. In any case, separating the city's sewage needs into a series of manageable small projects would seem to allow for gradual, but affordable and tangible progress.

5.4 Measures to Address Metals Other Than Lead

Much less is known about other metals (manganese and cadmium) than about lead, in terms of their concentrations in the bodies of Thai people, the sources of exposure, and the likely health effects. We are not confident in suggesting any control or remedial programs for these metals; we suggest only further exploratory studies to assess the significance of our findings that these metals occur at concentrations of concern. Studies should focus on investigating their levels in Thai body tissues and the pathways by which exposure to them occurs.

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Volume II: Technical Appendices

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41

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Volume II -- Technical Appendices

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VOLUME II: TECHNICAL APPENDICES
TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

Appendix A. Health Risks from Exposure to Air Pollution

I. Definition of the Problem

As Bangkok has grown as a center of industrial activity, and as the population of Bangkok has expanded, air pollution problems associated with industrial and nonindustrial sources has been on the rise. Air pollution results from two major categories of sources: point sources (such as industrial plants) and area sources (such as construction sites, automobiles, and boats). Of the more than 90,000 licensed factories in Thailand, 20,000 are located in the Bangkok Metropolitan Area (Industrial Works Department, Ministry of Industry, 1986). According to the Japan International Cooperation Agency (August, 1989) there were 129 motorcycles and 153 cars per 1000 persons in Bangkok, for a total of well over 1.5 million vehicles. By the year 2006, these figures are projected to increase to 150 motorcycles/1000 persons, and 200 cars/1000 persons. Both stationary and mobile sources of air pollution emit suspended particles, carbon monoxide, and oxides of nitrogen (NO_x), as well as toxic air pollutants such as metals and volatile organic chemicals (VOCs). Point sources that burn fossil fuel are also a major source of sulfur dioxide (SO_2).

Humans can be exposed to air pollutants in the obvious manner: through inhalation of the pollutants in the air. However, exposure to air pollutants can occur through less obvious routes as well. For example, air pollutants may deposit on soil. Young children may then inadvertently ingest contaminated soil through normal mouthing of objects and thumbs. Alternatively, air pollutants deposited on soil may be taken up by food crops grown in contaminated soil. Pollutants may also deposit on foods and plates at streetside stands, or in open containers for storing drinking water.

Health effects that have been related to air pollution are varied. **Suspended particulate matter** has been associated, through epidemiological studies, with restricted activity and increase in overall mortality rates. **Lead** in air has been shown to have a strong correlation with blood lead levels, which have been associated with neurological damage in children and with heart disease and stroke in adult men. A more extensive discussion of the health effects of lead in air is found in Appendix E. **Carbon monoxide** (CO) combines with hemoglobin to form carboxyhemoglobin (COHb) in blood. The presence of COHb in blood inhibits the ability of blood to carry needed oxygen to body tissues. High blood levels of COHb can lead to severe effects such as brain injury and death. At lower levels, COHb in blood has been associated with increased incidence of heart pain (angina) in persons with chronic cardiovascular disease. For the general population, low levels of COHb in blood has been associated with milder symptoms, such as inability to concentrate and headaches. Ozone causes eye

and upper respiratory irritation in the general population, and may aggravate chronic respiratory illness. **Sulfur dioxide** (SO_2) is also associated with respiratory irritation, especially in those individuals with chronic respiratory disease. **Toxic air pollutants**, such as benzene, formaldehyde, cadmium, and diesel particulates are believed to be associated with lung cancer.

Air pollution is ubiquitous and affects all segments of the population. Unlike water pollution, little can be done by individuals to avoid exposure to ambient air pollution. However, some segments of the population may be affected more than others (e.g., those who live in open air housing, those who work as street vendors, the elderly, children, etc.)

This project examined only outdoor levels of air pollution in Bangkok. The analysis did not examine the somewhat different problems that may arise from indoor air concentrations of air pollutants. In the U.S., the differences between indoor and outdoor pollution are quite important, because of the importance of indoor air pollution sources and the relatively tightly closed housing stock that limits the exchange of indoor and outdoor air. In Bangkok, however, where conservation of home heating is not an issue because of the openness of Thai housing, indoor and outdoor air concentrations may differ less sharply.

II. Data Acquired

Criteria Air Pollutants

Ambient monitoring data

Thailand has established ambient air quality standards for six air pollutants, known as "criteria pollutants," including total suspended particulates (TSP), lead, carbon monoxide (CO), ozone, sulfur dioxide (SO_2) and oxides of nitrogen (NO_x). The Office of the National Environment Board (ONEB) maintains eight permanent monitors in the city of Bangkok, in locations representing a variety of land uses: industrial, urban residential, suburban residential, and rural areas. ONEB also operates a mobile air monitoring unit. These monitors track concentrations of the criteria pollutants. CO, ozone, SO_2, and NO_x are monitored continuously, while 24-hour TSP and lead samples are taken every three days.

Summaries of the annual average concentrations for two criteria pollutants, TSP and lead, observed at seven of the eight ONEB monitoring stations for the years 1983-1986, are found in Table A.1 (data were not available for the eighth monitor). These data show an increase in ambient TSP

Table A.1 Annual Average Concentrations of TSP and Lead at Seven Monitoring Stations in Bangkok, 1983–1986(a)

Station	Land Use Type	TSP ug/m3 (b)				TSP		Lead mg/m3 (e)				Lead	
		1983	1984	1985	1986	Thai Std (c)	U.S. Std (d)	1983	1984	1985	1986	Thai Std (c)	U.S. Std (f)
ONEB	Urban Residential	100	100	90	80	100	75	0.4	0.25	0.3	0.3	10	1.5
Ban Somdet	Mixed	110	120	120	120	100	75	0.3	0.28	0.28	0.23	10	1.5
Saovabha	Commercial	90	100	90	110	100	75	0.67	0.37	0.35	0.4	10	1.5
Bangna	Industrial	120	140	100	110	100	75	0.32	0.59	0.41	0.35	10	1.5
Chankasem	Suburban residential	120	100	90	120	100	75	0.35	0.33	0.3	0.34	10	1.5
Rat Burana	Industrial	100	130	100	200	100	75	0.36	0.29	0.2	0.31	10	1.5
Sukhumvit	Urban Residential	100	100	100	120	100	75	0.42	0.39	0.45	0.44	10	1.5

Notes:
(a) Source: ONEB (1987).
(b) Annual geometric mean.
(c) Source: ONEB (1989)
(d) Source: 52 FR 24634 (July 1, 1987).
(e) Annual arithmetic mean.
(f) Source: 40 CFR 50.12., Arithmetic mean averaged over a calendar quarter.

concentrations over time, and a decrease in lead concentrations over time. In addition, at several monitors, the Thai TSP standard is exceeded. Even though the ambient concentrations reported for these two pollutants are often below the corresponding standards, some health effects are still believed to be associated with the presence of these contaminants in the ambient air. In particular, the lead values do not exceed the Thai lead ambient air standard, yet lead is believed to pose a significant health threat in Bangkok. The effects of lead on the health of the Bangkok population is discussed in Appendix E.

For carbon monoxide, the 1983-1986 data reported in ONEB (1987) show that neither the 1-hour and 8-hour ambient concentrations of CO exceeded their respective standards. However, data were also reported for curbside concentrations of CO (see Table A.8), which are substantially higher than ambient concentrations, and which may have health implications for persons who spend a significant amount of time living or working near roadways. These curbside data are discussed below.

Only summary data for ozone, SO_2, and NO_x were reported by ONEB (1987). ONEB (1987) reported that ozone levels in Bangkok were consistently well below the standard of 0.20 mg/m^3. The highest measurements reported were about 0.15 mg/m^3. These levels occurred during the hot, dry season. These data suggest little health risk associated with ozone in Bangkok. The average 24-hour concentration of SO_2 was reported to be 0.03 mg/m^3 (as measured at four stations); the average 1-hour concentration of NO_2 in Bangkok was reported to be about 0.02 mg/m^3 (as measured at five stations). These reported levels are an order of magnitude less than the Thai ambient air quality standards for these compounds. The low levels of SO_2 may be attributable to the relatively low sulfur content of fuels used (TDRI, 1987).

Ambient air quality monitoring is also performed by the Ministry of Public Health (MPH), Department of Health, Environmental Health Division (MOPH/DOH/EHD), which maintains two monitoring stations in the city. Monitors are located in Samut Prakan, an industrial area southeast of the city, and in Lad Prao, a residential area in the Northeast section of the city. EHD monitors air for concentrations of TSP, lead, SO_2 and NO_x. Data for these contaminants for the year 1989 are shown in Table A.2. These data show TSP concentrations that exceed the short-term (24-hour) and long term (annual) Thai standards for TSP. Data from the MPH monitors for the years 1979 through 1984 are reported in TDRI (1987). These data show that particulate concentrations were generally on the rise over this time frame. The 1989 data suggest that the trend has continued. The 1979-1984 data showed a decrease in mean annual lead concentrations, from 0.914 ug/m^3 in 1979 to 0.072 ug/m^3 in 1984 at Samut Prakan, and levels from 0.156 in 1982 to 0.092 in 1984 for Lad Prao. The drop is

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Table A.2. Annual Average Concentrations of TSP, Lead, NOx, and SO2 At Two Monitoring Stations Bangkok, 1989(a)

Pollutant	Units	Samut Prakan (Industrial) (a)	Lad Prao (Residential) (b)	Thai Standard (c)	U.S. Standard
TSP	ug/m3	178.4	201.5	100	75(d)
Pb	ug/m3	0.33	0.59	10	1.5(e)
NOx	ug/m3	16.3	33.8	320	100(e,f)
SO2	ug/m3	7.7	9.6	100	80(e)

Notes:
(a) Source: MOPH, DOH, EHD
(b) Source: MOPH, DOH, EHD
(c) Source: ONEB (1989)
(d) Source: 52 FR 24634 (July 1, 1987)
(e) Source: 40 CFR 50.4 through 50.12.
(f) Averaging time for U.S. std is annual; Averaging time for Thai std is 1 hour.

probably attributable to the reduction of lead in gasoline from 0.85 to 0.45 grams per liter, as well as an increase in the use of liquid propane gas and diesel fuels. The 1989 lead concentrations are above the 1984 levels, perhaps because of the increase in the number of cars since then.

To put these data in perspective, Table A.3. presents concentrations of TSP in urban areas around the world. This table shows that for 1983-1988, average annual concentrations of TSP exceeded the Thai standard for annual average TSP concentration of 100 ug/m^3. This table also shows that Bangkok's TSP problems are not unique. In fact, several cities in China and in India have more severe TSP problems than Bangkok. However, the levels observed in Bangkok exceed levels observed in Malaysia and Hong Kong, and in many European and U.S. cities.

Curbside monitoring data

ONEB has performed curbside monitoring of air quality for short periods of time at various points throughout the city in the last six years. These monitoring efforts have measured TSP, lead, CO, SO_2, and NOx. Data summarizing the average results found at various monitoring points along roads in Bangkok for the years 1984, 1985, 1986, 1988 and 1989 are found in Table A.4. As these data demonstrate, concentrations of the contaminants tend to be much higher than the concentrations detected at ambient monitors (shown in Table A.2). How these curbside concentrations relate to exposure depends on the dispersion of the contaminants from the roadways. Some contaminants may not disperse farther than a few meters from the roadways, suggesting that the impact on general air concentrations is not large: however, since many people work, eat, reside, drive and walk along the roadways, their exposure to these concentrations may be substantial.

Toxic Air Pollutants

There is ample ambient monitoring data for conventional air pollutants such as TSP, CO, and lead that can be used to assess health risks from these pollutants. Ambient monitoring has the advantage of providing actual measurements of pollutants in the air over time. By using monitoring data, we avoid having to make assumptions regarding emissions quantities and atmospheric dispersion of pollutants in the air. No such ambient data appears to exist for toxic air pollutants such as benzene, formaldehyde, and asbestos. For toxic air pollutants, we can only estimate ambient concentrations using emissions of these compounds and applying dispersion modelling to them.

Mobile sources

Emissions estimates are derived by using emissions factors that relate emissions to readily

Table A.3. Mean Daily Concentrations of TSP in Urban Areas in Various Countries(a)
(ug/m3)

	1973-75	1976-78	1979-81	1983-88
Asia				
China				
Shenyang	X (b)	X	225-523	258-529
Beijing	X	X	252-479	268-462
Giuangzhou	X	X	96-375	179-248
Shanghai	X	X	235-330	152-285
Xian	X	X	235-463	328-471
India				
Delhi	X	326-432	325-445	291-453
Calcutta	353-477	324-389	428-498	333-426
Bombay	X	154-243	154-243	140-267
Malaysia				
Kuala Lumpur	X	90-153	92-247	96-112
Hong Kong	28-93	29-114	25-83	21-48
BANGKOK	X	137-281	195-243	198-243
Europe				
Brussels	X	29	25	21
Prague	137-251	121-146	X	X
Copenhagen	X	33-43	31-48	44-58
Helsinki	X	57-144	65-136	62-118
Athens	X	206-259	211-235	179-188
London	33-40	25-35	18-46	15-30
North America				
United States				
Chicago	X	74-161	56-134	100
New York City	X	57-74	51-65	44-62

Notes:
(a) Source: WRI, 1989.
(b) X = not available.

Table A.4. Average Concentrations of CO, TSP, Lead ,& Oxides of Nitrogen Found at Curbside Monitors for Years 1984–1986, 1988, and 1989(a)

Year	1984			1985				1986				1987				1988				1988				1989				1989				Thai	US
Sampling Description	Mean of Ten Sampling Locations			Mean of Four Roads				Mean of Two Roads				Mean of Twelve Sampling Points				Mean of Two Roads				Mean of Twelve Sampling Points				Monitoring at Rachitree Hospital				Mean of Five Sampling Points				Std(e)	Std
Pollutant:	1(b)	2(c)	3(d)	avg	max av	min	max	avg	max av	min	max	avg	max av	min	max	avg	max av	min	max	avg	max av	min	max	avg	max av	min	max	avg	max av	min	max		
CO 1 hour (mg/m3)	22.3	24.6	19.1	11.2	23.5	1.8	33.0	4.0	10.0	0.0	16.0	9.5	19.7	1.1	31.8	4.0	8.5	1.0	13.5	5.8	12.8	0.7	21.1	2.0	6.0	1.0	14.0	6.0	12.6	0.7	25.5	50.00	40(f)
CO 8 hour (mg/m3)	12.6	15.0	11.2	12.8	15.8	6.3	20.8	5.0	6.5	1.5	8.5	9.8	14.1	3.2	21.1	3.5	4.5	2.5	5.5	5.8	8.6	1.3	12.5	2.0	4.0	1.0	5.0	6.2	9.0	1.3	14.4	20.00	10(f)
TSP 24 hour (ug/m3)	400	500	458	337.	-	297.	380	285	0	210	365	300	-	300	500	255	0	215	305	362	-	243.	522.	150	-	100	210	445	-	301.6	686.6	100	75(g)
Lead 24 hour (ug/m3)	1.9	2.1	1.8	1.2	-	1.1	1.4	1.2	0.0	1.0	1.4	1.6	-	1.1	2.2	1.3	0.0	1.0	1.9	1.6	-	1.0	2.5	0.7	-	0.3	0.9	1.9	-	1.3	3.0	10	1.5(f,h)
NOx 1 hour (ug/m3)	-	-	-	-	-	-	-	40	105	10	145	-	-	-	-	100	295	15	390	-	-	-	-	-	-	-	-	-	-	-	-	320	100(f,i)

Notes:

(a) Source: ONEB Monitoring Data

(b) Round one of 1984 monitoring, performed during transition to one-way.

(c) Round two of 1984 monitoring, first record after transition to one-way.

(d) Round three of 1984 monitoring, second record after transition to one-way.

(e) Source: ONEB (1989).

(f) Source: 40 CFR 50.4 thru 50.12

(g) Source: 52 FR 24364 (July 1, 1987)

(h) U.S. Lead Standard. Arithmetic mean of 24 hour data averaged over calendar quarter.

(i) U.S. averaging time for NOx is annual.

available measures, such as the number of kilometers travelled by vehicles. To obtain emissions estimates, we combined U.S. mobile source emission factors with data on transportation in Bangkok. A study conducted by (Japanese International Cooperation Agency) JICA in 1989 to develop options for improvement in the transportation system in Bangkok provided much of the information needed to estimate emissions in conjunction with emissions factors, including Vehicle Kilometers Travelled (VKT), composition of Bangkok traffic by vehicle type, and the type of fuels used by different vehicle types. The specific methods used to estimate the ambient toxic air pollutant concentrations from mobile sources and their associated health risks are discussed in Section III below.

Stationary sources

Toxic air pollution emission data for stationary sources were unavailable. From what we were able to determine, it does not appear that an emissions inventory is maintained by the Thai government. In the absence of direct emissions inventories, it would still be possible to estimate emissions using emission factors that relate average emissions to measures associated with the release of that pollutant, such as the amount of fuel combusted, the volume and type of feedstock, etc. For example, emissions factors from printing ink manufacturing are expressed as kilograms of nonmethane volatile organic chemicals emitted per megagram (metric ton) of ink product produced. However, due to time constraints, we were unable to obtain information that could be used to develop estimates of emissions, such as production data for various industries in Bangkok.

III. Analytical Methodology Used to Develop Risk Estimates

Criteria Air Pollutants

For this analysis, we have estimated health effects resulting from exposure to the following "criteria" air pollutants: TSP, lead and CO. The methods used to estimate risks from TSP and CO are discussed below. The risks from lead are discussed separately in Appendix E. Health effects from the other three criteria pollutants are not considered. Ozone was not considered in our analysis, because we do not have on average 1-hour ozone levels in Bangkok needed to perform such an analysis of health effects; furthermore, as discussed above, ONEB (1987) reported that ozone levels in Bangkok are quite low. The maximum reported concentration of ozone measured in Bangkok from 1983 to 1986 was 0.15 mg/m^3, below the 1-hour standard of 0.20 mg/m^3, suggesting that the health effects from this pollutant are minimal. Health effects associated with SO_2 and NO_x are also expected to be minimal because the ambient concentrations are an order of magnitude below the standards. Furthermore, many epidemiological studies relating morbidity and mortality to air pollution have been

unable to distinguish the morbidity and mortality effects of TSP and SO_2 (and the acid aerosols it forms). Therefore, to avoid double counting, we have performed our evaluation using only TSP and have ignored the other effects of sulfates that may occur when SO_2 levels are below the standard.

Total suspended particulates

A simple comparison of incidence of respiratory illness in Bangkok to incidence in other parts of Thailand suggests that ill health among the Bangkok population may be linked to air pollution. The incidence of respiratory disease in the Central Region (including Bangkok) was 136.9 per 1000 persons while the rate of respiratory disease for Thailand overall was 107.4 per 1000 (ONEB, 1987). Furthermore, respiratory disease increased 3 percent during the period 1982 to 1985. A possible contribution to these rates is air pollution in Bangkok. These statistics are difficult to compare, however, without knowing regional differences in rates of cigarette smoking, also a major cause of respiratory illness (as well as heart disease and cerebrovascular disease), and other factors that may influence differences in the disease rates.

Several U.S. researchers have attempted to relate more rigorously the incidence of respiratory and other diseases to air pollution. There are several epidemiological methods used to estimate health risks from air pollutants. One method is to use longitudinal studies, which relate changes in health effects to changes in air pollutant levels over time in a single location. Another method is cross-sectional studies, which relate differences in morbidity and mortality among several locations to differences in the air pollution levels in these locations. These studies attempt to relate the concentration of air pollutants to measures of health such as restricted activity, mortality, and specific minor health effects such as headaches and nausea. Typically, health effects are measured using available mortality data, hospital admissions data, or survey data where respondents recall health ailments over a specified period. Many of the relationships are derived using multiple regression techniques which control for other variables that may affect health such as age, gender, income and other variables. Although these studies have been criticized on methodological grounds, we will use these relationships to derive upper-bound estimates on possible health effects from total suspended particulates. Limitations of these studies are discussed in greater detail in Section V.

From the available literature, we selected four U.S. studies that relate total suspended particulates (TSP) to morbidity and mortality as the basis for estimating health risks. The equations developed in these studies were the same used in Oates et al. (1989) to estimate benefits of environmental standard setting. These studies, and the equations used, are described below.

Morbidity from TSP

Ostro (1983a) related TSP levels to work loss days, while Ostro (1983b) related total TSP to restricted activity days and work loss days. Both of these studies were based on data obtained from the Health Interview Survey conducted by the National Center for Health Statistics. This scientific survey consisted of 50,000 interviews. Individuals interviewed were questioned about their health and that of household members. Respondents were questioned about illnesses that resulted in work loss or reduced activity days over the previous two weeks. Questions regarding socioeconomic and demographic factors, smoking habits, and occupation were also included. These data were compared with air pollution data for the city in which the respondents lived. To control for possibly confounding factors, the regression equations included variables for age, sex, existence of a chronic health condition, race, marital status, annual family income, annual mean temperature, annual precipitation, population density, occupational status (i.e., whether person is a blue collar worker or not), and the number of cigarettes smoked per day. The results of the ordinary least squares regression will be used in this analysis. However, Ostro (1983b) also used other statistical techniques and still found significant relationships between air pollution and health.

The relationship between work loss days and TSP is:

RECEPTWLDPT = 0.00145 * 26 [(delta TSP) * POP]

where:

RECEPTWLDPT	=	excess work loss days in receptor population
26	=	adjustment from 2 week recall period to full year
delta TSP	=	change in TSP concentration, and
POP	=	size of the receptor population.

For restricted activity days, the relationship is:

RECEPTRADPT = 0.00282 * 26 [(delta TSP) * POP]

where:

RECEPTRADPT	=	excess restricted activity days in receptor population
26	=	adjustment from 2 week recall period to full year
delta TSP	=	change in TSP concentration, and
POP	=	size of the receptor population.

Mortality from TSP

Oskaynak et al. (1986) related suspended particle concentrations to excess mortality using cross-sectional mortality data. City-specific daily mortality data were related to daily air pollution data using multiple regression techniques. The multiple regressions included variables for percentage of population over 65, the median age of the population, the percentage of the population that is nonwhite, the density of the population, the percentage of the population with a college education, and the percentage of poor in the population. The form of the function is assumed to be linear. The equation is:

RECEPTMORT = [0.2 / 100000] *[(delta TSP)*POP]

where:

RECEPTMORT	=	estimated excess mortality in receptor population per year
delta TSP	=	change in TSP concentration, and
POP	=	size of the receptor population.

The level of TSP in the air will never reach zero, even in the absence of human activity, because of naturally occurring particulate matter (sea salt, suspended soil particles, etc.). Therefore, we must consider the morbidity and mortality related to TSP above some arbitrary minimum level. We used the US standard for annual average particulate concentration, 75 ug/m^3 for this minimum level. The input to the equations was the difference between this level and the measured TSP value.

For measured TSP concentrations, we used the ONEB data from seven monitors for 1983 through 1986, and data from two MPH monitors for 1989. Rather than average concentrations at the monitors, we assumed that each of the seven urban monitors for which we had data represented air quality in about one-seventh of the city. We then assumed that the population density of the city was

approximately uniform, so that each measurement corresponded to the exposure level of one-seventh (14%) of the city's population. For 1989, we have data for two monitors, one in Samut Prakan, an industrial area southeast of Bangkok, and one in Lad Prao, a residential area in Northeastern Bangkok. Two of the seven monitors for the 1984-1988 data were in industrial areas, while the other five were in residential or commercial areas. To make the 1989 analysis consistent with the earlier years, we assumed two-sevenths (about 29 percent) of the city's population lived in industrial areas, and five-sevenths (about 71 percent) in commercial or residential areas. The Samut Prakan data were then used to estimate the exposures for those in industrial areas, while the Lad Prao data were used to estimate exposures for those in other areas of the city.

Carbon monoxide

Studies for CO have been conducted relating it to increased incidence of heart pain (angina) for individuals having heart disease. From these studies, the U.S. EPA derived a standard below which increase in onset of angina attacks is not expected to occur. Standards were set for both long-term averages and for the short-term one hour exposures; however, only the long-term average will be considered here. Data relating health effects to low ambient concentrations of CO do not permit estimation of the number of persons actually experiencing a particular health effect at a given ambient concentration. Instead, this analysis will follow the technique used by U.S. EPA Region III that estimates the number of persons at risk for health effects from CO exposure. If the 8-hour standard is exceeded, US EPA Region III (1988) assumed that those members of the sensitive population with existing heart disease will be at moderate risk for onset of angina pain. Those without existing heart disease may be at low risk for mild health effects such as headaches. The severity of the effects that may be experienced is not evaluated.

In the U.S., about 10 percent of the population is assumed to have chronic heart disease and thus may be affected by elevated CO levels. To estimate the percent of the Bangkok population that has heart disease, we compared the mortality due to heart disease in the U.S. (from U.S. Statistical Abstracts) with the mortality due to heart disease in Bangkok (obtained from BMA health statistics). The mortality rate due to heart disease in Bangkok is only 20 percent of that in the U.S.; therefore, we will assume that the fraction of Bangkok residents afflicted with chronic heart disease is only 20 percent of the fraction of afflicted U.S. citizens. Therefore, this study assumes that 2 percent (20 percent of 10 percent) of the Bangkok population suffers from chronic heart disease.

For carbon monoxide, we used the 8-hour average data from the curbside monitoring performed by ONEB. This approach has two potential flaws: the concentrations of CO at the curbside

may overestimate 8 hour average exposure to someone who does not live or work near a road. Furthermore, ONEB sampling was short-term (usually only a few weeks at a time) and therefore may not represent year-round concentrations.

As with TSP, rather than average concentrations at all of the monitors from which data are available, we assumed that each of the twelve curbside monitors for which we had data represented air quality in about one-twelfth of the city. We then assumed that the population density of the city was approximately uniform, so that each measurement corresponded to the exposure level of one-twelfth of the city's population. We did not know the CO concentration for each day during monitoring; therefore, we assumed that the mean concentration is the typical curbside CO concentration on each day of the year for that area of the city. As a result, the population at risk is at risk every day of the year. We also looked at the maximum concentration observed for one day, and estimated populations at risk due to these concentrations. Estimates from these data posit that the population at risk will be at risk at least one day per year.

Toxic Air Pollutants

As discussed above, because monitoring data for toxic air pollutants were not available, we instead derived estimates of their ambient concentrations. To obtain estimates of the concentrations of these pollutants, we examined only emissions from mobile sources. Toxics from area sources, such as dry cleaners, gas stations, etc., and from industrial sources were not considered. We cannot quantify how much of the air toxics problem in Bangkok may be left out because of these omissions.

To estimate risk from air toxics, first we estimated emissions of toxic air pollutants from mobile sources. Next, we used these estimates to derive ambient concentrations. The methods used for each of these steps is described below.

Emissions estimates

U.S. EPA estimates toxic air emissions from mobile sources in two steps. The first step is to estimate a contaminant-specific emissions factor (in grams per mile) for each model year for four basic classes of vehicles: light and heavy duty gasoline-powered vehicles, and light and heavy duty diesel-powered vehicles. Separate emissions factors may be estimated considering the pollution control device in use on each vehicle class during each model year. The second step is to estimate the number of miles travelled by each type of vehicle. To do so for any particular year, EPA must consider not only the number of miles travelled for each type of vehicle, but also the age distribution within each vehicle class. This is an important point to consider, since older vehicles tend to have

higher emissions; therefore, a vehicle fleet for a particular calendar year with a greater percentage of older cars would have higher total emissions than a fleet with newer cars.

For this analysis, the emission factors for various toxic air pollutants from mobile sources were extracted from U.S. EPA (1987a) and from U.S. EPA (1985a). Where possible, we used emission factors for vehicles without catalytic converters, since these devices, to the best of our knowledge, are not in common use in Bangkok. When separate emission factors were not given for vehicles without pollution control devices, we used emission factors for the U.S. 1974 model year, since this was the last year before catalytic converters were required in the U.S. If this information was not available, we used the emissions factors for the overall 1974 U.S. fleet. These latter emission factors contain implicit assumptions regarding the percentage of miles travelled by each vehicle class and the age distribution within each vehicle class for the US in 1974. The contaminants considered, the vehicle class associated with the contaminant emissions, the emission factors used for each contaminant, and the method used to derive the emission factors is found in Table A.5.

Most emission factors are expressed in units of grams per mile. For certain organic contaminants, emissions factors are expressed as a percentage of the total hydrocarbon (HC) emissions for the vehicle. We obtained an estimate of total hydrocarbon emission factor, in grams per mile, for the 1974 US fleet to represent the HC emissions for the Bangkok fleet. The HC emission factor is then multiplied by the appropriate percentage for each contaminant.

By using the emission factors presented in U.S. EPA (1987a), we assume that the speed and fuel efficiency assumptions used to derive these emission factors are also valid for Bangkok vehicles. In reality, the assumption for average vehicle speed may be inaccurate because of the severe traffic congestion that afflicts Bangkok.

To obtain total emissions, the emissions factors are multiplied by the number of vehicle miles travelled per day to obtain the daily emissions estimate for each contaminant. Certain contaminant emissions are associated with gas-fueled vehicles only, some are emitted from diesel-fueled vehicles only, and others are emitted from both kinds of vehicles. In addition, emission factors are different for light duty and heavy duty vehicles using each fuel type. To match the emission factors with the appropriate measure of miles travelled, we needed an estimate of the number of miles travelled by each vehicle category. JICA (1989) conducted a study to assess needed improvements in the Bangkok road transportation system. This report provided data on the number of vehicle kilometers travelled (VKT) each day by the Bangkok fleet. It also provided, for certain major roads, the traffic

Table A.5. Emission Factors (a)

Contaminant	Emission Factor (g/mile)	Source and Nature of Estimate
1,3 Butadiene	9.59E-02 o	Gasoline Vehicles Only Expressed as % of total Hyrdrocarbon emissions Estimated using 1974 Hydrocarbon emission level (b)
Asbestos(l) Asbestos(h)	4.00E-03 o 2.80E-02	Both Gasoline and Diesel Vehicles Consistent emission levels expected from all types of vehicles Estimated using 1984 EPA data
Benzene(l) Benzene(h)	1.48E-01 o 6.78E-01	Gasoline Vehicles Only Expressed as % of total exhaust and total evaporative HC emissions Estimated using 1974 model year levels of HC emissions (b)
Cadmium	1.60E-05 o	Leaded Gasoline Vehicles Only Higher emission levels expected with non-cat. equipped vehicles Estimated using 1980 and 1981 EPA non-catalyst vehicle data
Diesel Particulate(ld, l) Diesel Particulate(ld, h) Diesel Particulate(hd, l) Diesel Particulate(hd,h)	7.00E-01 o 9.00E-01 1.89E-01 3.36E-01	Diesel Vehicles Only Higher emission levels expected in heavy duty vehicles Estimated using 1974 EPA model year data
Ethylene(l) Ethylene(h)	9.18E-01 o 1.33E+00	Gas Vehicles Only Expressed as % of total Hydrocarbon emissions Estimated using 1986 EPA light and heavy duty fleet data and 1974 Hydrocarbon emission Level (b)
Ethylene Dibromide	5.92E-04 o	Lead Gas Vehicles Only Higher emission levels expected from non-cat. equipped vehicles Estimated using 1986 EPA light duty fleet data
Formaldehyde(g,l) Formaldehyde(g,h) Formaldehyde(d,l) Formaldehyde(d,h)	1.02E-01 o 3.06E-01 3.06E-01 1.02E-01	Both Gasoline and Diesel Vehicles Expressed as % of total Hydrocarbon emissions Estimated using 1985 EPA gas and diesel fleet data and 1974 Hydrocarbon emission level (b)
Organics associated with non-diesel particulates (ld) Organics associated with non-diesel particulates (hd)	2.30E-02 o 7.20E-02	Gasoline Vehicles Only Higher emission levels expected from heavy duty vehicles Estimated using 1970-1974 EPA light duty model year data and Pre-1987 EPA heavy duty model year data

Notes:
(a) Source: U.S. EPA, 1987a.
(b) Source: U.S. EPA, 1985a.

composition, that is, the percentage of traffic that is composed of personal cars, motorcycles, trucks and buses. For each of these vehicle types, the report also presented the fraction of vehicles that use diesel fuel and the fraction that use gasoline. Combining these data enabled us to derive the number of kilometers travelled for each of the vehicle classes for which distinct emission factors are available[1]. Kilometers were, of course, converted to miles for use in this assessment.

Dispersion model

To estimate dispersion of the toxic emissions over the city of Bangkok, we used a simple box model derived from Sullivan (1988a,b). This model assumes that emissions are uniformly emitted across the area of the box. The model also assumes complete mixing of the emissions over the area of the city and over a given mixing height. The box model calculation is (Sullivan, 1988a):

$$C = Q\,(x/uz) \times 10^6$$

where:

C	=	concentration of the contaminant (ug/m^3),
Q	=	emission of the contaminant (grams per m^2 per second),
x	=	square root of the area of the city, (meters),
u	=	wind speed, (m/s),
z	=	vertical dispersion term (meters), and
10^6	=	factor to convert g to ug.

The vertical dispersion term estimates the vertical extent of the mixing of pollutants. It is calculated as (Sullivan, 1988b):

$$z = (0.06)\,(x/2)\,[(\,1 + (x/2)(0.0015)]^{.05}$$

where:

z	=	vertical dispersion coefficient, (meters), and
x	=	square root of the area of the city, (meters).

For this assessment, we ignored the effects of atmospheric decay of the contaminants. Ignoring this

[1] We assume that the cars and motorcycles represent "light duty" vehicles while trucks and buses represent "heavy duty" vehicles. This is accurate for trucks, since the JICA traffic composition data gives values specifically for "heavy" trucks. However, it may be inaccurate for buses, since the category "buses" may include both light and heavy duty buses.

phenomenon will tend to over estimate air concentrations. We also ignored wet and dry deposition of contaminants. Wet and dry deposition would tend to lower the air concentration of the contaminant, but would transfer the contaminant to another medium, such as water or soil, to which human exposure may also occur. Wind speed estimates for this model were obtained from JICA (1990). The area over which emissions disperse was set equal to the study area used in JICA (1989) in order to be consistent with the VKT estimates derived in that report.

Risk assessment

All toxic air contaminants for which emissions are estimated are carcinogens by inhalation. U.S. EPA (1990) provides unit risk estimates for all of the toxic contaminants considered in this analysis. Unit risk factors for these contaminants are found in Table A.6. Unit risk factors represent the individual lifetime (70 year) risk per ug/m^3 of contaminant in the air. These factors incorporate standard EPA assumptions regarding human body weight and ventilation rate (the amount of air people breathe. The standard assumption for ventilation rate is 20 m^3 per day, while human body weight is assumed to be 70 kg. Since we know that the average body weight of Thai people is only 54 kg (MOPH, Dept of Nutrition, 1989), the unit risk factors presented in EPA (1990) were adjusted by a factor of 70/54, or 1.3, for use in this analysis. The risk is the product of the contaminant-specific unit risk factor and the ambient concentration of the contaminant.

IV. Discussion of Findings

Risks from Criteria Pollutants

Risk estimates for TSP are found in Table A.7. Estimated restricted activity days (RAD) have risen from about 11 million per year in 1983 to about 19 million in 1986 (Table A.7.a), while work loss days (WLD) have risen from about 6 million in 1983 to about 10 million in 1986 (Table A.7.b). Based on 1989 data, RAD may be as high as 51 million per year and WLD may be as high as 26 million; however, these high estimates may be a result of assigning about 70 percent of the Bangkok population to a monitor with a relatively high TSP concentration. Excess mortality was estimated to be about 300 in 1983, 500 in 1986, and about 1400 in 1989 (Table A.7.c.). Note that risks are estimated even for those areas which are currently below the standard for this pollutant. The rise in the number of health effects predicted is due to both increasing population, and the apparent rise in ambient TSP concentrations at certain monitors in recent years.

Risk estimates for CO are found in Table A.8. These results show an estimated 20,000 persons

Table A.6. Unit Risk Estimates for Carcinogenic Emissions From Mobile Sources (a)

Contaminant	Unit Risk(b)	Adjusted Unit Risk(c)
1,3 Butadiene	2.80E-04	3.63E-04
Asbestos(h)	2.60E-02	3.37E-02
Asbestos(l)	6.60E-04	8.56E-04
Benzene	8.00E-06	1.04E-05
Cadmium	1.80E-03	2.33E-03
Diesel Particulate(h)	1.00E-04	1.30E-04
Diesel Particulate(l)	2.00E-05	2.59E-05
Ethylene	2.70E-06	3.50E-06
Ethylene Dibromide	5.10E-04	6.61E-04
Formaldehyde	1.30E-05	1.69E-05
Organics associated with non-diesel particulates	2.50E-04	3.24E-04

Notes:

(a) Source: EPA, 1987.

(b) Unit risk equals risk per lifetime exposure to 1 ug/m3; Lifetime based on U.S. standard assumption of 70 years.

(c) Adjusted for Thai body weight. Source: MOPH, Department of Nutrition.

Table A.7. Estimated Health Risks From Ambient TSP

A.7.A. Restricted Activity Days

Station	Land Use Type	Estimated Population in the Area of the Monitor (a)					Estimated Restricted Activity Days (No restricted activity below 75 ug/m3) (b)				
		1983	1984	1985	1986	1989	1983	1984	1985	1986	1989
ONEB	Urban Residential	717000	739000	766000	781000		1309000	1349000	839000	285000	
Ban Somdet	Mixed	717000	739000	766000	781000		1832000	2428000	2516000	2566000	
Saovabha	Commercial	717000	739000	766000	781000		785000	1349000	839000	1995000	
Bangna	Industrial	717000	739000	766000	781000		2355000	3507000	1398000	1995000	
Chenkasem	Suburban Residential	717000	739000	766000	781000		2355000	1349000	839000	2566000	
Rat Burana	Industrial	717000	739000	766000	781000		1309000	2967000	1398000	7127000	
Sukhumvit	Urban Residential	717000	739000	766000	781000		1309000	1349000	1398000	2566000	
Samut Prakan	Industrial					1673000					12628000
Lad Phrao	Residential					4182000					38619000
Total		5019000	5173000	5362000	5467000	5855000	11254000	14298000	9227000	19100000	51247000

Notes:

(a) Source: Data from ONEB (1987)
Data from MOPH (1989)

(b) From Ostro, 1983(b)

(c) From Ostro, 1983(b)

(d) From Oskynak et al., 1986.

Table A.7. Estimated Health Risks From Ambient TSP

A.7.B. Work Loss Days

		Estimated Population in the Area of the Monitor (a)					Estimated Work Loss Days (c)				
Station	Land Use Type	1983	1984	1985	1986	1989	1983	1984	1985	1986	1989
ONEB	Urban Residential	717000	739000	766000	781000		676000	697000	433000	147000	
Ban Somdet	Mixed	717000	739000	766000	781000		946000	1254000	1300000	1325000	
Saovabha	Commercial	717000	739000	766000	781000		405000	697000	433000	1031000	
Bangna	Industrial	717000	739000	766000	781000		1216000	1811000	722000	1031000	
Chankasem	Suburban Residential	717000	739000	766000	781000		1216000	697000	433000	1325000	
Rat Burana	Industrial	717000	739000	766000	781000		676000	1532000	722000	3680000	
Sukhumvit	Urban Residential	717000	739000	766000	781000		676000	697000	722000	1325000	
Samut Prakan	Industrial					1673000					6522000
Lad Phrao	Residential					4182000					19944000
Total		5019000	5173000	5362000	5467000	5855000	5811000	7385000	4765000	9864000	26466000

Notes:
(a) Source: Data from ONEB (1987)
Data from MOPH (1989)
(b) From Ostro, 1983(b)
(c) From Ostro, 1983(b)
(d) From Oskynak et al., 1986.

Table A.7. Estimated Health Risks From Ambient TSP

A.7.C. Mortality

Station	Land Use Type	Estimated Population in the Area of the Monitor (a)					Estimated Mortality Based on TSP (d)				
		1983	1984	1985	1986	1989	1983	1984	1985	1986	1989
ONEB	Urban Residential	717000	739000	766000	781000		36	37	23	8	
Ban Somdet	Mixed	717000	739000	766000	781000		50	67	69	70	
Saovabha	Commercial	717000	739000	766000	781000		22	37	23	55	
Bangna	Industrial	717000	739000	766000	781000		65	96	38	55	
Chankasem	Suburban Residential	717000	739000	766000	781000		65	37	23	70	
Rat Burana	Industrial	717000	739000	766000	781000		36	81	38	195	
Sukhumvit	Urban Residential	717000	739000	766000	781000		36	37	38	70	
Samut Prakan						1673000					346
Lad Phrao						4182000					1058
Total		5019000	5173000	5362000	5417000	5855000	308	392	253	523	1404

Notes:
(a) Source: Data from ONEB (1987)
Data from MOPH (1989)
(b) From Ostro, 1983(b)
(c) From Ostro, 1983(b)
(d) From Oskynak et al., 1986.

Table A.8. 1988 Estimated Risks From Curbside Concentrations of Carbon Monoxide

Sample Area	Estimated Population	Data Type	CO 8 hour (mg/m3)(a)	Comparison to U.S. Standard(b)	Persons at Risk(c) Mod(d)	Low(e)
Rajaprarop Rd.	477,000	avg	8.61	Below	0	0
		max	12.5	Above	10,000	467,000
Yaoyarat Rd.	477,000	avg	8.11	Below	0	0
		max	10.08	Above	10,000	467,000
Hluang Rd.	477,000	avg	3.01	Below	0	0
		max	4.69	Below	0	0
Bamrung Mueng Rd	477,000	avg	7.22	Below	0	0
		max	11.83	Above	10,000	467,000
Sukumvit Rd.	477,000	avg	4.48	Below	0	0
		max	7.22	Below	0	0
Bang Lum Poo	477,000	avg	3.65	Below	0	0
		max	5.62	Below	0	0
Praknong	477,000	avg	2.51	Below	0	0
		max	3.91	Below	0	0
Paholyotin Rd.	477,000	avg	3.61	Below	0	0
		max	5.18	Below	0	0
Silom Rd.	477,000	avg	10.17	Above	10,000	467,000
		max	15.63	Above	10,000	467,000
Wong Wien Yai	477,000	avg	12.44	Above	10,000	467,000
		max	18.33	Above	10,000	467,000
Chaisamorapoom	477,000	avg	2	Below	0	0
		max	4	Below	0	0
Seepraya	477,000	avg	3.64	Below	0	0
		max	5.42	Below	0	0
Total:	5,724,000			Average	20,000	934,000
				Worst Day	50,000	2,335,000

Notes:

(a) Source: ONEB, Monitoring Data 1988

(b) Source: EPA Region III, 1988. See text for explanation of risk assessment.

(c) Population is at risk when CO 8 hour exceeds 10 mg/m3.

(d) Mod: Persons with chronic heart disease assumed to be at mod risk when CO exceeds 10mg/m3.

(e) Low: Persons in general population assumed to be at low risk when CO exceeds 10 mg/m3.

with heart disease at moderate risk for angina pain from CO exposure. This figure represents about 0.3 percent of the total Bangkok population, but about 15 percent of the population assumed to have cardiac disease. About 934,000 persons (or about 15 percent of the Bangkok population) are estimated to be at low risk of mild effects (such as headaches) from CO exposure. Based on the maximum observed CO curbside concentrations, about 50,000 persons with cardiovascular disease are at moderate risk of angina pain at least one day per year, and over 2 million are at low risk of moderate effects at least one day per year.

It is useful to get an idea of the contribution of various sources to ambient concentrations of criteria air pollutants. TDRI (1987) estimated the relative contribution of transportation, power generation, industry, services, fisheries, agriculture, and households to emissions of TSP, SO_2, NO_2, total hydrocarbons, and CO for Thailand as a whole, assuming no pollution control devices were in place. These estimates, displayed in Table A.9., were derived by TDRI using energy demand data and WHO and EPA emissions factors for these sectors. Two factors are important to keep in mind when examining Table A.9. First, these estimates were made for Thailand as a whole; these proportions are probably not directly applicable to Bangkok. In Bangkok, we might expect the relative contribution from transportation to be higher than its contribution in the rest of the country; while fisheries, agriculture and household fuel may be less important for Bangkok than for the rest of the country. Second, contribution to emissions is not the same as contribution to ambient concentrations at human receptor points, since this will depend on dispersion patterns and proximity of the source to human population centers. Nonetheless, this table does give a rough idea of relative importance of the different sectors to the pollution problem. The importance of various sectors differs by pollutant. Transportation plays a major role in total hydrocarbon and CO emissions, with power, industrial and other fuel combustion sources contributing most of the SO_2 emissions.

These results suggest that serious health problems are associated with current levels of air pollution in Bangkok. Furthermore, preliminary projections of industrial emissions for the years 1986 through 2011, provided by TDRI, imply that without improved air quality management, the situation may worsen in the next 20 years. Table A.10. summarizes TDRI's projections for five major air pollutants. According to these projections, industrial emissions of NO_x, SO_2, carbon dioxide and TSP are projected to increase about two- to six-fold, depending on the pollutant. With an annual increase in vehicles expected to be several percent, mobile source emissions in Bangkok will also rise over time.

Table A.9 Estimated Emission from Combustion Sources (1982) (a)

(tons/year and percent)

Sources	TSP	SO2	NOx	HC	CO
Transport	7,515	47,339	35,390	17,952	406,570
%	3%	15%	23%	46%	60%
Power generation	96,300	153,087	43,027	1,054	2,143
%	33%	48%	28%	3%	0%
Industry	62,701	106,735	23,970	6,569	110,212
%	21%	34%	16%	17%	16%
Service	4,221	2,145	5,114	1,525	108,937
%	1%	1%	3%	12%	16%
Fisheries	0	1,220	12,204	2,305	4,972
%	0%	0%	8%	6%	1%
Agriculture	54,022	3,607	8,166	1,882	34,666
%	19%	1%	5%	5%	5%
Household	67,109	2,997	24,843	4,942	4,941
%	23%	1%	16%	13%	1%
Total	291,868	317,130	152,714	39,229	672,441

Notes:

(a) Table reproduced from TDRI, 1987.

Table A.10. Estimated Industrial Emissions of 6 Pollutants, 1986–2011.

(tons per year)

Pollutant	1986	1988	1991	1996	2001	2006	2011	Industry With Largest Contribution	Percentage Increase in Emissions (1986-2011)
HC	12,467	12,035	12,356	13,223	13,635	14,689	16,124	Food	29.33%
NOx	33,396	43,348	52,660	76,485	101,682	139,640	193,347	Nonmetal manuf.	478.95%
SO2	106,594	146,036	170,171	241,702	350,380	505,609	725,314	Food, and Paper Nonmetal manuf.	580.45%
CO	51,080	51,811	54,856	62,127	68,421	78,780	93,341	Textiles, and Paper Nonmetal manuf.	82.73%
CO2	16,366,258	18,223,168	20,929,889	27,224,793	34,130,686	44,111,912	58,193,758	and Food Paper and	255.57%
TSP	159,293	207,420	247,974	342,113	445,735	593,684	804,635	Nonmetal manuf. Food, Paper,	405.13%
								and Nonmetal manuf.	
Total	16,729,088	18,683,818	21,467,906	27,960,443	35,110,539	45,444,314	60,026,519		

Notes:

(a) Source: TDRI, preliminary estimates.

Risks from Toxic Air Pollutants

Estimated cancer risks from toxic air pollutants from mobile sources are found in Table A.11. This table shows that about 70 to 80 cancer cases per year over the entire Bangkok population may result from exposure to mobile source emissions of toxic air pollutants. Including toxic air pollutants from industrial and area sources would increase these estimates. The range was calculated using low and high estimates of emission factors and unit risk estimates for each toxic contaminant, where available. These estimates should be considered upper-bound estimates, since they are based on 95th percentile of the unit risk estimate and on a conservative box model of pollutant mixing that ignores atmospheric decay and deposition. These results suggest that the cancer risk from toxic air pollutants may be significant and deserves closer investigation.

V. Limitations

Using morbidity and mortality relationships for TSP derived from cross-sectional mortality studies is controversial, even for applications within the U.S. Applying these relationships to another country is even more tenuous. Problems with cross-sectional mortality studies are discussed in detail in Evans et al. (1984). Reviewers have criticized the databases used in these studies as inadequate. Some criticisms are based on the measures of exposure used in these studies, such as the use of central city monitors as estimators of exposure and the use of a single year of pollution data. The measures of mortality used and the inadequate control for age in the regression relationships have also been criticized. Many other critics have questioned these studies with the regression analysis because of the potential for omission of confounding variables, such as smoking, diet, occupational exposures, migration, and other factors. However, Evans et al. (1984) point out that these confounding variables must be correlated with air pollution levels in order to be effective confounders. Furthermore, many of these variables were included in the derivation of regression relationships used in this analysis.

Table A.11. Risk Calculations for Air Toxics From Mobile Sources

Contaminant	Emission Factor(a)	Total Emission (g/m2/sec)	Concentration(c) (ug/m3)	Unit Risk(d) (ug/m3)	Indiv Risk(e)	Pop Risk Case/yr (f)
1,3 Butadiene	9.59E-02	2.6E-08	2.0E+00	2.8E-04	5.5E-04	4.6E+01
Asbestos(l)	4.00E-06	1.1E-12	8.2E-05	6.6E-04	5.4E-08	4.5E-03
Asbestos(h)	2.80E-05	7.7E-12	5.7E-04	2.6E-02	1.5E-05	1.2E+00
Benzene(l)	1.48E-01	4.0E-08	3.0E+00	8.0E-06	2.4E-05	2.0E+00
Benzene(h)	6.78E-01	1.9E-07	1.4E+01	8.0E-06	1.1E-04	9.3E+00
Cadmium	1.60E-05	3.0E-12	2.2E-04	1.8E-03	4.0E-07	3.4E-02
Diesel Particulate(ld, l)	7.00E-01	8.3E-09	6.2E-01	1.0E-04	6.2E-05	5.2E+00
Diesel Particulate(ld, h)	9.00E-01	1.1E-08	8.0E-01	1.0E-04	8.0E-05	6.7E+00
Diesel Particulate(hd, l)	1.89E-01	3.6E-09	2.7E-01	1.0E-04	2.7E-05	2.3E+00
Diesel Particulate(hd,h)	3.36E-01	6.4E-09	4.8E-01	1.0E-04	4.8E-05	4.0E+00
Ethylene(l)	9.18E-01	2.5E-07	1.9E+01	2.7E-06	5.1E-05	4.2E+00
Ethylene(h)	1.33E+00	3.6E-07	2.7E+01	2.7E-06	7.3E-05	6.1E+00
Ethylene Dibromide	5.92E-04	1.1E-10	8.2E-03	5.1E-04	4.2E-06	3.5E-01
Formaldehyde(g,l)	1.02E-01	1.9E-08	1.4E+00	1.3E-05	1.8E-05	1.5E+00
Formaldehyde(g,h)	3.06E-01	5.7E-08	4.3E+00	1.3E-05	5.5E-05	4.6E+00
Formaldehyde(d,l)	3.06E-01	9.5E-09	7.1E-01	1.3E-05	9.2E-06	7.7E-01
Formaldehyde(d,h)	1.02E+00	3.2E-08	2.4E+00	1.3E-05	3.1E-05	2.6E+00
Organics associated with non-diesel particulates (ld)	2.30E-02	4.3E-09	3.2E-01	2.5E-04	8.0E-05	6.7E+00
						0.0E+00
Organics associated with non-diesel particulates (hd)	7.20E-02	0.0E+00	0.0E+00	2.5E-04	0.0E+00	0.0E+00
Totals:						
Low					8.2E-04	6.9E+01
High					9.7E-04	8.1E+01

Notes:

(a) Source: EPA 1990.

(b) Total emissions= (Emission Factor x Miles Traveled)

(c) Source: Sullivan, 1988. See text for explanation of box model.

(d) Source: EPA 1987.

(e) Individual risk= Contaminant concentration x unit risk

(f) Population risk= (Individual lifetime risk x Population)/70 days in average lifetime

l= low estimate

h= high estimate

d= diesel

g= gas

Other criticisms have been leveled against the statistical methods used to estimate the relationship between air pollution and mortality. Importantly, the likely shape of the dose-response function remains unclear. Some studies have argued for nonlinear relationships. That is, with increasing pollution concentration, the slope of the relationship between mortality and air pollution concentrations has been shown to decrease (Schwartz and Marcus, 1990). If the relationship between air pollution and mortality is in fact nonlinear, then these relationships may overestimate health effects at higher pollutant concentrations.

Some reviewers believe that TSP in and of itself may not be the pollutant of concern: instead, the pollutant of concern may be SO_2, or associated acid aerosols formed by SO_2 or nitric oxides, or an unknown pollutant that is correlated with levels of these pollutants. If the sources of TSP in Bangkok are different than in the US upon which mortality studies are based, then the associated pollutants that may be the underlying cause of the mortality and morbidity may also be different, and may make the relationships found in the U.S. invalid for Bangkok.

Finally, the size of the particles are important. Those particles less than 10 microns in diameter are of most concern because of their ability to penetrate deeply within the respiratory system. Mr. Jumpol Siriswasdi, Chief of air inspection of the ONEB, recently stated that at least half of the particles in Bangkok dust are less than 10 microns ("Bangkok's Killing Dust", The Nation, Dec. 1989). If the particle size distribution of TSP in Bangkok air is significantly different than the distribution in cities in which cross-sectional mortality studies were conducted, then the relationships may be inappropriate in this setting.

As discussed above, the estimation of risks from air toxics does not include stationary sources or small area sources such as dry cleaners and gas stations. The magnitude of the underestimation of risk is unknown. However, a U.S. study of the air toxics problem in the U.S. estimated that up to 60 percent of the cancer incidence estimated from toxic air pollutants is attributable to road vehicles (Haemisegger et al., 1985). Thus, if stationary and small area sources contribute about the same percentage to air toxics in Bangkok as they do in the US, then the omission may only underestimate risk by about 40%. However, the degree of control of air toxic emissions from both stationary and mobile sources in the U.S. probably differs from that in Thailand. In the U.S., air toxics are controlled to some extent by devices installed to reduce criteria air pollution emissions from stationary sources. Also, some co-control occurs from criteria pollution control equipment and engine modifications applied to motor vehicles, but perhaps to a lesser degree. On balance, we might guess that the percent contribution of stationary sources to air toxics in Thailand may be somewhat higher

than in the US.

The estimation of risks from air toxics is also limited by the use of U.S. emission factors to estimate emissions from the Bangkok vehicle fleet. First, we assumed that Bangkok's fleet has 1974 era emission controls. This probably overestimates emissions, since many Bangkok vehicles are newer than 1974 with engines and tuning (independent of catalytic converters) that control emissions. Second, we assumed that the average speed used to develop U.S. emission factors is representative of Bangkok traffic. In fact, Bangkok's severe traffic congestion slows traffic considerably. This assumption would tend to underestimate emissions.

Appendix B. Health Risks from Exposure to Water Pollution

I. Definition of the Problem

The Chao Phraya River flows south from the northern valleys of Thailand, through the Bangkok Metropolitan Area, to the Gulf of Thailand. The average annual flow of the Chao Phraya from 1982-1988 was 9.5 billion cubic meters (MOPH/DOH/EHD, 1989). There is a dramatic fluctuation in the flow rate of the river between the rainy season, which lasts from about May to October, and the dry season, which lasts from about mid-October to mid-May. Bangkok is located on the estuary of the Chao Phraya, about 50 kilometers from the river mouth; therefore, the segment of the river that flows through the city is under tidal influences.

The Chao Phraya and associated canals (called klongs) have played a significant role in Bangkok history and tradition. Communities along the banks of the river and the klongs have historically used the waters for domestic purposes (bathing, swimming, washing clothes), as well as for navigation and commerce. However, many of these uses are declining due to severe deterioration of water quality in both the river and the klongs. Both rapid population and industrial growth have contributed to this decline. In addition, many of the klongs have been filled in over the past few decades, causing drainage problems.

The Chao Phraya continues to serve as water supply to almost all of Bangkok's population. The Metropolitan Waterworks Authority (MWA), which provides water to nearly 75% of Bangkok's population, draws nearly all of its water from the Chao Phraya. Water is drawn from an intake located approximately 90 kilometers from the mouth of the river (that is, about 40 kilometers north of Bangkok). MWA also draws some of its water supply from groundwater, but is phasing out this practice due to subsidence problems caused by overextraction of groundwater.

This Appendix discusses human health risks that may be associated with the pollution of the Chao Phraya River and the klongs. Other appendices (Appendices C, E, and F) will address health risks from specific pollutants or specific exposure pathways associated with water pollution.

The Nature of Water Pollution

Measures of water quality can be classified into three categories. First, there are physical/chemical characteristics of water that act as indicators of water quality. These include

temperature and pH (a measure of acidity or alkalinity), as well as levels of biochemical oxygen demand (BOD), dissolved oxygen (DO), and inorganic nutrients such as phosphorus and nitrogen compounds. Two of these indicators, BOD and DO, are related to the oxygen content of water necessary to support aquatic life. Aerobic microorganisms in water require oxygen in order to break down organic matter; an excess of organic matter can severely deplete the oxygen in the water. BOD is a measure of the amount of oxygen depleted from water by microorganisms due to degradation of organic matter. Organic material can be naturally occurring or can originate from human activity. Dissolved oxygen is a direct measure of the quantity of oxygen present in the water, and is affected by the quantity of organic matter in the water, by the quantity of inorganic chemicals which react with oxygen, and by temperature (since solubility of gases is temperature-dependent). In temperate climates, 5 to 7 mg/l DO is typically required to support healthy natural aquatic communities, but in tropical waters, where fish are adapted to lower levels of dissolved gases that are the natural result of higher water temperatures, a level of 2 to 4 mg/l is suggested (Onodera, 1985). Inorganic nutrients such as phosphorus and nitrogen fuel the growth of aquatic microorganisms and plants. Excessive nutrients can cause exploding populations of microorganisms and blooms of aquatic plants, which can choke the water body, interfering with navigation and recreational uses of the water, and eventually add to BOD levels once the plants die and decompose. Therefore, the oxygen content of the water can be affected by nutrients as well as by BOD.

Microbiological contamination is a second measure of water quality. Measured as the presence of coliform bacteria, microbiological contamination is related to the transmission of infectious diseases, including pathogenic bacteria and viruses. Coliform bacteria themselves are intestinal bacteria that occur in healthy persons and generally are not thought to cause illness except at high concentrations. However, their presence in a water body suggests the presence of human (or other warm-blooded animal) waste in the water. Such wastes may transmit pathogenic enteric bacteria and viruses, such as those which cause hepatitis, typhoid, dysentery, and cholera. The exact kind of pathogens and concentrations of pathogens present in water containing fecal coliforms will depend on the rate of illness in the population from which the wastes are generated; there is no constant ratio between the presence of coliforms and the presence of pathogens. However, in general, the percentage of water samples that contain pathogens does appear to increase as the fecal coliform concentration of the water increases (Waite, 1984). In the U.S., which has a comparatively low incidence of infectious disease, *Salmonella typhi* are found at a rate of less than 1 per million coliforms; measured virus densities are 10 to 200 enteric viruses per million coliforms (Hammer, 1986). Typical microbiological standards for drinking water in the U.S. are set at less than 1 coliform per 100 ml water, since at this concentration, ingestion of pathogens is highly improbable. The Thai standard is similar, at less than 2.2 per 100 ml.

In the U.S., the allowable concentration of fecal coliforms in untreated surface waters depends on the use of the water body. Water quality standards for the protection of public water supplies are more strict than those for waters used for agricultural or industrial purposes. The standards for direct contact waters may be stricter than water supply standards, and those for protection of shellfish harvesting are stricter still (Hammer, 1986).

The third category of water quality measures includes concentrations of toxic pollutants. Toxic pollutants, such as synthetic organic chemicals and heavy metals, may pose chronic health risks to users of the water body. Some metallic contaminants, such as iron and manganese, may originate from natural sources. Other contamination can occur through direct discharge of industrial and domestic wastewaters, surface runoff, or deposition of air pollutants.

Pathways of Exposure

One direct pathway of exposure to contaminated surface water and groundwater is through drinking. In addition to drinking contaminated water, however, the other primary pathways of exposure that can affect health are direct contact with contaminated surface water, such as swimming, bathing, washing clothes, etc. Such direct exposure leads to the transmission of water borne diseases, or can cause infections in open wounds. Washing food, implements, or dishes with contaminated water may also transmit pathogenic microorganisms.

Indirect pathways of exposure include ingestion of contaminated foods harvested from polluted water bodies. For example, "red tide" toxins, produced by excessive growth of microorganisms, and in part attributable to loading of nutrients to surface water, can cause contamination of shellfish and subsequent illness in humans that ingest the shellfish (see Paphavasit et al.(1985)). In addition, organochlorine and heavy metal water pollutants that bioconcentrate in organisms living in contaminated surface waters also create health risks to Bangkok residents that eat the affected organisms. Another indirect pathway of exposure results from irrigation of fields with polluted surface water or groundwater which can also lead to exposure to contaminants if pollutants are taken up from the water and soil by crops that are subsequently consumed by Bangkok residents. (Risks from pesticides and metals in food are discussed in Appendix C.)

II. Data Acquired

Water Supply Data

In 1989, the Government of Thailand produced a country profile detailing the drinking water supply and sanitation situation in the country. According to this report, the 6th National Plan (1987-1991) set a national goal for water supply of 5 liters per capita per day (lpcd) of drinking water (including drinking, tooth brushing, cooking), and 45 lpcd for domestic use (including bathing, housecleaning, latrine, etc.). In Bangkok, the overall goal for provision of domestic water is 210 lpcd.

The Metropolitan Waterworks Authority (MWA), a state enterprise under the Ministry of the Interior, provides water to the Bangkok Metropolitan area. The MWA operates a Central System and seven separate systems. The Central System is the primary source of water for Bangkok. According to Dhamasiri (1989), MWA water production from surface water sources in 1989 was 2.7 million m^3/day. Water is extracted from the Chao Phraya at Sam Lae, about 91 km north of the Gulf of Thailand (about 40 km north of Bangkok). The intake location was chosen to avoid pollution from Bangkok and salinity from high tides. Water flows through Klong Prapa Canal, and then is diverted to one of three treatment plants: Sam Sen, Thonburi, and Bang Khen. Of these, Bang Khen is the largest, producing 2 million m^3/day of water. In addition to surface water sources, MWA pumped about 120,000 m^3/day of groundwater from 41 deep public wells. This represents a significant drop in the daily pumping rate, which was 464,000 m^3/day (from 110 public wells) in 1980. This source of water will be abandoned altogether in the near future.

There are 7.2 million people in the MWA service area, which includes Bangkok and surrounding areas. Of these, 5.4 million people are served, a service rate of 75 percent (Government of Thailand, 1989). Dhamasiri (1989) reports that about 900,000 m^3/day are extracted from licensed private wells in Bangkok and surrounding areas. Unlicensed extraction of groundwater may be as much as another 50 percent (TDRI, 1987). These groundwater withdrawals far exceed the estimated safe yield of 0.8 million m^3/day. Overpumping has led to subsidence and to salt water intrusion into aquifers underlying Bangkok.

As the population of Bangkok grows, so does the demand for water. MWA expects that demand will approximately triple in the next 30 years, requiring 7.78 million m^3/day (Dhamasiri, 1989). MWA is pursuing several strategies to meet this demand. First, MWA planned an expansion of the Bang Khen treatment plant of 0.5 million m^3 of water per day for 1989. The goal is to increase production by 0.9

million m^3 by the year 1991, while simultaneously phasing out reliance on groundwater to prevent further subsidence (Gov't of Thailand, 1989). In addition, MWA is trying to reduce water loss from the distribution system. The loss rate has been reduced from 63% in 1971 to only 34% in 1988 (Govt of Thailand, 1989). The Water Loss Reduction Office of the MWA is responsible for detection of leaks, monitoring of operations and other functions to promote the reduction of water loss through the distribution system. The goal is to reduce distribution losses to 30% by 1991 (Govt of Thailand, 1989). Even if these measures are successful, there is a limit to the amount of water that can be extracted from the Chao Phraya each day because of competing uses. Dhamasiri (1989) estimates that a maximum of 5.18 million m^3/day can be taken from Chao Phraya for water supply uses. To compensate, plans are being made to build an interbasin canal to bring water from the Mae Klong River to Bangkok. Bangkok will also need surface storage after 2017, in order to serve an estimated population of 15 million people (Dhamasiri, 1989).

While Thailand has been nearing its water supply goals for various segments of the population, water quality goals have not been met. The Government of Thailand (1989) provides figures on water quality compliance for various water systems in Thailand. Although compliance for the MWA was not specifically discussed, for all large piped water systems in Thailand, the compliance with physical/chemical water quality parameters is 84%, while compliance with bacteria goals is only 51%. The MWA may have better quality water than this. MWA sampling of 7 areas in Bangkok shows that all but one of the areas are in compliance with bacteriological goals. For water provided by deep wells in Thailand, physical/chemical parameter compliance is 56%, while compliance with bacteriological goals is 51%. For all water types of water supply systems in Thailand, drinking water that satisfies quantity requirements is 73 percent country wide, but only 26 percent based on bacteriological quality standards.

Domestic Waste Treatment Data

The Bangkok Metropolitan Authority (BMA) is responsible for sewage and solid waste disposal. Only about 2 percent of Bangkok residents are hooked up to sewage treatment plants (TDRI, 1987). Seventy-three hospitals are required by the MOPH to have wastewater treatment facilities for their own sewage, while 15 private hospitals are required by the BMA to have wastewater treatment facilities (Government of Thailand, 1989). Some large establishments such as hotels have their own wastewater treatment facilities. However, most residences and businesses have septic tanks or cess pools. These are intended to allow liquids to seep into the ground with soils acting as filters for the microbiological contaminants. Solids are intended to be collected periodically. However, because of underlying clay,

127

and the density of septic tanks and cess pools, seepage is retarded, and liquids often run off into klongs or storm drains. Others illegally bypass treatment altogether, connecting septic tanks directly to storm drains, klongs, and the river (ONEB, 1988). Bangkok has over 1500 primary canals through which waste can travel to the Chao Phraya (Gov't of Thailand, 1989).

Since 1960, several studies have been conducted to estimate the cost of a centralized sewage treatment facility for Bangkok. A 1960 proposal estimated costs at 1460 million baht ($58.4 million). By 1968, cost estimates had soared to about 13,000 million baht ($520 million). A 1981 JICA proposal suggested a system whose total costs were estimated to be over 36,000 million baht ($1.4 billion) (ONEB, 1988). None of these proposals was implemented because of the prohibitive costs. ONEB (1988) recently proposed a decentralized intercepting sewer system which would be much lower in cost than the JICA proposal. Cost estimates for this system were approximately 11,000 million baht ($440 million) for construction costs and 328 million baht ($13 million) for annual operating costs.

Drinking Water Data

Tap water data and raw water data from MWA are shown in Table B.1. These tap water data are from ten samples taken from a single area of Bangkok. We were able to obtain chemical-specific concentration data for this area only. However, MWA data on drinking water quality in seven areas of the city show that all areas are comparable in terms of compliance with bacteriological and turbidity standards. We will assume that they are also comparable in terms of chemical-specific concentrations. This may be a reasonable assumption for contaminants that originate from the water supply source, since nearly all of the water supply for the parts of Bangkok served by the MWA comes from the same source. However, this assumption is unlikely to be valid for contaminants that get into the water supply during distribution and storage.

River and Klong Water Quality Data

Water quality at several sampling stations in the vicinity of Bangkok is monitored by the Ministry of Public Health, Department of Health, Environmental Health Division (MOPH/DOH/EHD). The results of 1989 monitoring are shown in Table B.2. These data also show the Class 3 and Class 4 standards that apply to the parts of the Chao Phraya for which monitoring data are reported. The river is classified according to the beneficial uses for which the reach of the river

Table B.1. 1989 Drinking Water Quality Data For Bangkok

Parameter	Units	MWA Raw Water(a)	Tap Water Average(b)	Thai DW Standard(c)
Turbidity	NTU	70	4	5
pH		7	7	6.5-8.5
Colour	Pt-Co	less than 5	5	5
Conductivity	uohms/cm	300		
Total Solids	mg/l	200		500
Total Alk.	mg/l	90		
Carb. hardness	mg/l	90	90	
non-carb. hard.	mg/l	6		
Chloride	mg/l	10	20	250
Sulfate	mg/l	20		200
Oxygen consume	mg/l	5		
Nitrogen	mg/l		0.07	
Ammonia, free	mg/l	0.2		
Nitrate as N	mg/l	0.1		45
Nitrite as N	mg/l	0.006		
Calcium	mg/l	30		75
Iron	mg/l	0.7	0.1	0.5
Fluoride	mg/l	0.2	0.3	0.7
Lead	mg/l		0.02	0.05
Tin	mg/l		0.03	
Sulfate	mg/l		4	200
Manganese	mg/l	0.03		0.3
Magnesium	mg/l	7		50
DO	mg/l	4		
BOD	mg/l	2		
Bacteria	MPN/100 ml	50000		
Coliform Bact.	MPN/100 ml	200000	less than 2.2	less than 2.2
Faecal Coliform	MPN/100 ml	900	none found	none

Notes:

(a) Source: MWA 1989 (provided by MOPH).

(b) MWA 1989 (provided by MOPH). Data for Area 7, assumed to be typical of drinking water in Bangkok

(c) From ONEB (1989).

Table B.2. Water Quality Data for the Chao Phraya River, 1989(a)

Station No., Name, and Km from River Mouth		DO mg/l	Turbidity NTU	Alk. PP mg/l	Alk. MO mg/l	Cl mg/l	NH3 mg/l	NO3 mg/l	PO4 mg/l	BOD mg/l	Fe mg/l	Mn mg/l	Cu mg/l	Zn mg/l	Pb mg/l	Cr mg/l	Cd mg/l	Hg ug/l	Coliform MPN/ 100ml	Fecal Coliform MPN/ 100ml	Hardness mg/l
Station 3	ave	1.7	49	0	103.6	245.7	0.5	0.7	0.4	5.7	1.57	0.17	0.01	0.09	0.02	0.01	0	0.14	561569	107753	122.9
Bangkok Port	min	0	2.8	0	68	13	0	0	0	0.1	0	0.07	0	0.01	0	0	0	0	1600	540	78
30 km	max	5.5	108	0	140	1900	2.1	6.6	1.1	33	3	0.32	0.03	0.19	0.08	0.01	0.01	0.42	3500000	330000	160
	STD(d)	1.6	33.9	0	22.5	435.6	0.6	1.3	0.4	6.3	0.85	0.07	0.01	0.05	0.02	0	0	0.17	840805	96010	26
Station 6	ave	nm(e)	44.5	0	84	5.8	0.2	0.5	0.7	1.5	1.52	0.06	0	0.08	0.02	0.01	0.01	0	24000	13000	94
Rama VI Bridge	min	nm	17	0	68	2.6	0.1	0.4	0.4	1.3	0.94	0.03	0	0.06	0	0.01	0.01	0	24000	13000	94
57 km	max	nm	72	0	100	9	0.4	0.6	0.9	1.6	2.1	0.08	0	0.09	0.03	0.01	0.01	0	24000	13000	94
	STD	nm	27.5	0	16	3.2	0.2	0.1	0.3	0.1	0.58	0.03	0	0.01	0.02	0	0	0	0	0	0
Station 7	ave	nm	72.3	0	88	15.3	0.1	0.5	0.6	1.1	1.8	0.05	0.01	0.08	0.02	0.01	0	0	160000	35000	90
Nonthonburi	min	nm	26	0	68	8	0.1	0.3	0.2	0.5	1.3	0.03	0	0.07	0	0.01	0	0	160000	35000	90
67 km	max	nm	140	0	98	21	0.3	0.6	1	1.5	2.1	0.07	0.01	0.09	0.03	0.01	0.01	0	160000	35000	90
	STD	nm	48.9	0	14.1	5.4	0.1	0.1	0.3	0.4	0.36	0.02	0	0.01	0.01	0	0	0	0	0	0
Station 8	ave	3.4	53.8	0	88.2	15.2	0.1	0.1	0.3	1.3	1.73	0.09	0.01	0.08	0.01	0.01	0	0.008	92658	51311	100
Parkred	min	2	17	0	64	7	0	0	0	0.5	0.51	0.01	0	0.03	0	0	0	0	2400	700	78
78 km	max	5.1	120	0	108	22	0.4	0.4	0.9	4.3	2.9	0.46	0.02	0.22	0.03	0.01	0	0.45	540000	350000	122
	STD	7.3	32	0	14.1	4.4	0.1	0.1	0.3	0.8	0.65	0.09	0	0.04	0.01	0	0	0.16	138534	100801	13.4
Station 9	ave	nm	56	0	80	12	0.3	0.3	0.6	1.2	1.87	0.06	0	0.06	0.01	0	0	0.3	134000	87667	86
Pathumtani	min	nm	39	0	72	6	0.1	0.1	0.2	0.5	1.6	0.04	0	0.05	0	0	0	0	24000	7000	78
85 km	max	nm	72	0	88	16	0.7	0.7	1	2.1	2.1	0.09	0	0.08	0.03	0.01	0	0.59	350000	240000	94
	STD	nm	13.5	0	6.5	4.3	0.3	0.3	0.3	0.7	0.21	0.02	0	0.01	0.01	0	0	0.3	152744	107779	8
Average	ave	2	55	0	88	59	0.2	0.42	0.5	2.2	1.7	0.09	0.006	0.008	0.02	0.008	0.002	0.09	194000	59000	99
Over All	min	1	20	0	68	7	0.06	0.2	0.2	0.58	0.87	0.04	0	0.04	0	0.004	0.002	0	42000	11000	84
Stations	max	5	102	0	107	390	0.8	2	1	8.5	2.4	0.2	0.01	0.1	0.04	0.01	0.005	0.3	915000	194000	110
	STD	4	31	0	15	91	0.3	0.4	0.3	1.7	0.53	0.05	0.002	0.02	0.01	0	0	0.1	226000	61000	9.5
Class 3 Std (b)		4	-	-	-	-	0.5	5	-	2	-	1	0.1	1	0.05	0.05	0.005	0.002	20000	4000	
Class 4 Std (c)		2	-	-	-	-	0.5	5	-	4	-	1	0.1	1	0.05	0.05	0.005	0.002	-	-	-

Notes:

(a) Source: MOPH, Department of Health, Division of Environmental Health

(b) Chao Praya is Class 3 from 62-142 km. Standards from ONEB(1989).

(c) Chao Praya is Class 4 from 7-62km. Standards from ONEB(1989).

(d) STD = standard deviation.

(e) nm = not measured.

is intended. The ONEB classification scheme is shown in Figure B.1. The 1989 monitoring data show that DO and coliform bacteria standards are frequently not met. In addition, the Hg concentration exceeds the standard at Station 3, and the cadmium standard is exceeded at Station 6.

Data for 1989 from two klongs were also provided by MPH/DOH/EHD. Results from these sampling efforts are shown in Table B.3. Sampling was conducted at these two klongs (Sam Sen and Rajburana) because of complaints and therefore data may not be representative of all klongs. However, the levels of BOD reported for these two klongs are comparable to the levels for over 25 klongs throughout the city reported in ONEB (1988). Coliform levels are very high at two of the sampling stations on Klong Rajburana.

Onodera (1985) conducted an extensive investigation of the extent and distribution of pollution in the sediment and water of the Chao Phraya river and associated klongs. He found that contamination began at the Nonthaburi Bridge (82 km from the river mouth) and continued to the river mouth. Low dissolved oxygen (below 2 mg/l), high BOD, high nitrates, phosphorus and coliform bacteria were all observed during the dry season. Coliform bacteria increased during the rainy season. It was observed to be as high as 2,400,000 coliforms per 100 ml, and as high as 350,000 coliforms per 100 ml in the klongs (probably indicating runoff from septic systems due to overflooding of septic tanks and other catchments). Data for Onodera (1985) are shown in Table B.4. Data from the 1984 sampling are shown along with data from earlier studies conducted in 1979 and 1982[1]. For comparison purposes, average water quality at low flow periods in the reach between 7 and 62 kilometers, which has been designated class 4 by the NEB, from 1981 to 1984 are shown in Table B.5.

Organochlorine pesticides were also investigated by Onodera (1985). Pesticide data for river and klong water and sediments are shown in Tables B.6a and B.6b. The organochlorine pesticides with the highest frequencies of occurrence were: alpha BHC, heptachlor, aldrin, and dieldrin. These compounds occurred in more than 70% of water samples in October 1984. The maximum observed concentration of alpha BHC exceeded the Class 3 and Class 4 water quality standard by an order of magnitude in September 1982 sampling, while the maximum concentration of lindane exceeded its standard by a factor of four. The maximum observed value for heptachlor in September 1983 sampling was almost twice the standard for this compound. The 1984 sampling showed maximum

[1]These data cannot be assumed to reflect trends in river quality over time, since the methods and sampling locations used may differ among the studies.

Figure B.1

Classifications	Objectives/Condition & Benificial usages
Class 1	Extra clean tresh surface water resources using for : (1) conservation. not necessary pass through water treatment processes require only ordinary process for pathogenic destruction (2) ecosystem conservation which basic living organisms can spread breeding naturally
Class 2	Very clean tresh surface water resources using for : (1) consumption which require the ordinary water treatment process before uses (2) aquatic organism conservation for living and assisting for fishery (3) fishery (4) recreation
Class 3	Medium clean tresh surface water resources using for (1) consumption but have to pass through an ordinary treatment process before uses (2) agriculture
Class 4	Fairly clean tresh surface water resources using for (1) consumption but require special water treatment process before uses. (2) industry (3) other activities
Class 5	The resources which are not classified in class 1–4 and using for (1) navigation

Source : Notification of the Ministry of Science. Technology and Energy (B.E. 2528 (1985.)). published in the Royal Government Gazette. Vol. 103. Part 60. dated April 15. B.E. 2529 (1986)

Chao Phraya River Water Quality Standards

Control areas (Km. from River mouth)	Water Quality Standards (Same as Standard of Water Classification)
7–62	Class 4
62–142	Class 3
142–379	Class 2

Source : Notification of the Office of the National Environment Board (January 17. B.E. 2529 (1986)). published in the Royal Government Gazette. vol. 103. No. 60. dated April 15. B.E. 2529 (1986)

Reproduced from: ONEB (1989b)

128

Table B.3. Water Monitoring Data for Two Klongs in Bangkok, Thailand, 1989(a)

Parameter	Units	Klong Rajburana									Klong San Sab			
		Station 1	Station 2	Station 3	Station 4	Station 5	Station 6	Station 7	Station 8	Average	Station 1	Station 2	Station 3	Average
Temperature	C	31	30	31	31	31	31	30	31	31	31	31	31	30
pH	pH units	7.5	7.6	7.5	7.5	7.5	7.5	7.4	7.4	7.5	8.1	8	8.2	8.1
Conductivity	umhoms/cm	-	-	-	-	-	-	-	-	-	-	-	-	-
DO	mg/l	-	-	-	-	-	-	-	-	-	2.9	1.9	4.3	3
Turbidity	NTU	3.1	6.4	6.2	4.6	2.3	4.4	4.6	2.7	4.3	12	13	16	14
Alkalinity, PP	mg/l	nil	nil	nil	nil	nil	nil	nil	nil	nil	nil	nil	nil	nil
Alkalinity, MO	mg/l	140	140	140	150	140	140	140	150	143	100	90	130	110
Cl	mg/l	-	-	-	-	-	-	-	-	-	-	-	-	-
Total N	mg/l	-	-	-	-	-	-	-	-	-	-	-	-	-
NH3 (Ammonia as N)	mg/l	0.52	0.85	1.3	1.3	1.6	1.6	1.8	1.7	1.3	0.27	0.16	0.36	0.26
NO3 (Nitrate as N)	mg/l	0.4	1	1	0.9	1.2	0.8	1.2	1.3	0.9	0.6	0.5	0.7	0.6
PO4	mg/l	0.6	0.7	0.5	0.3	0.6	0.7	0.6	0.7	0.6	0.3	0.4	0.4	0.4
BOD5	mg/l	9.1	8.9	8.3	8.6	8.4	8.6	8.5	8.5	8.6	6.5	3.5	5.7	5.2
Fe	mg/l	1.2	1.2	0.85	0.62	0.52	0.66	0.79	0.89	0.84	1.2	2.1	1.4	1.5
Mn	mg/l	0.1	0.13	0.13	0.12	0.12	0.13	0.14	0.14	0.13	0.27	0.3	0.23	0.27
Cu	mg/l	0.01	0.01	0.01	0.009	0.01	0.01	0.01	0.01	0.01	0.009	0.01	0.009	0.009
Zn	mg/l	0.11	0.09	0.08	0.09	0.1	0.1	0.1	0.08	0.09	0.09	0.08	0.08	0.08
Pb	mg/l	0.02	0.02	0.02	0.02	0.01	0.03	0.03	0.02	0.02	0.03	0.02	0.01	0.02
Cr	mg/l	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.01	0.009	0.01	0.02	0.01	0.01
Cd	mg/l	0.004	0.004	0.004	0.004	0.003	0.003	0.004	0.003	0.004	0.003	0.004	0.005	0.004
As	mg/l	-	-	-	-	-	-	-	-	-	-	-	-	-
Hg	ug/l	nil	nil	nil	nil	nil	nil	nil	nil	nil	0.07	0.07	nil	0.05
Aldrin	ug/l	nil	nil	nil	nil	nil	nil	-	-	nil				
Dieldrin	ug/l	nil	nil	nil	nil	nil	nil	-	-	nil				
Endrin	ug/l	-	-	-	-	-	-	-	-	-				
Total DDD	ug/l	-	-	-	-	-	-	-	-	-				
o,p-DDD	ug/l	nil	nil	nil	nil	nil	nil	-	-	nil				

Table B.3. Water Monitoring Data for Two Klongs in Bangkok, Thailand, 1989(a)

Parameter	Units	Klong Rajburana									Klong San Sab			
		Station 1	Station 2	Station 3	Station 4	Station 5	Station 6	Station 7	Station 8	Average	Station 1	Station 2	Station 3	Average
p,p-DDD	ug/l	-	-	-	-	-	-	-	-	-				
Total DDE	ug/l	-	-	-	-	-	-	-	-	-				
o,p-DDE	ug/l	0.019	0.038	0.023	0.027	nil	nil	-	-	0.018				
p,p-DDE	ug/l	nil	nil	nil	nil	nil	nil	-	-	nil				
Total DDT	ug/l	-	-	-	-	-	-	-	-	-				
o,p-DDT	ug/l	nil	nil	nil	nil	nil	nil	-	-	nil				
p,p-DDT	ug/l	nil	nil	nil	nil	nil	nil	-	-	nil				
Endrosulfan	ug/l	-	-	-	-	-	-	-	-	-				
Heptachlor	ug/l	nil	nil	nil	nil	nil	nil	-	-	nil				
Heptachlor Epoxide	ug/l	nil	nil	0.068	nil	nil	nil	-	-	0.011				
Lindane	ug/l	nil	nil	nil	nil	nil	nil	-	-	nil				
Mirex	ug/l	-	-	-	-	-	-	-	-	-				
alpha BHC	ug/l	nil	nil	nil	nil	nil	nil	-	-	nil				
Beta BHC	ug/l	nil	-	-	-	-	-	-	-	-				
TDE	ug/l	-	-	-	-	-	-	-	-	-				
m,p-DDD	ug/l	nil	nil	nil	nil	nil	nil	-	-	nil				
Coliform	MPN/100 ml	54000	160000	54000	54000	160000	3500000	160000	110000000	14000000	24000	35000	35000	31000
Fecal Coliform	MPN/100 ml	13000	35000	35000	35000	35000	120000	160000	4000000	550000	4900	24000	13000	14000
Cyanide	ug/l	0.04	0.06	0.07	0.07	0.08	0.08	0.08	0.08	0.07				
Hardness	mg/l										230	200	200	210

Notes:

(a) Source: MOPH, Department of Health, Division of Environmental Health (1989)

Table B.4. Summary of Physical/Chemical Characteristics of Chao Phraya River and Klong Water, 1984 (a)

Parameter	ONEB Class 4 Standard (b)	River			Klongs
		April (c) (range)	September (d) (range)	October (d) (range)	October (d) (range)
pH	5-9	7.36-7.7	7.09-7.33	7.4-7.7	7.05-7.5
Temperature		30-32.5	29-31	29-29.5	29.7-30.8
Chloride		15.9-1980	14.6-28.5	9.4-19.4	14.1-317.5
DO	2.0 or greater	0.6-2.05	0.2-3.84	2.05-4.15	0-2.65
BOD(e)	4.0 or less	0.6-2.5	0.88-4.09	1.15-3.25	1.02-3.83
COD		11.04-68.8	12.31-28.19	12.94-33.83	10.65-58.79
N03-N	5	0.01-0.477		0.18-0.252	0.012-0.269
N02-N		ND-0.225		0.01-0.078	0.003-0.06
NH3-N	0.5	0.43-8.58		0.48-2.39	0.95-10.59
Kjeldahl N		0.67-2.44		0.63-6.18	
Total Phosphorus		0.04-0.21			0.043-0.59
Phenol	0.005	2.9-5.2	ND		
Coliform Bacteria	20,000	13,000-170,000	28,000-2,400,000	49,000-540,000	20,000-350,000
Suspended Solids			35-192		23.2-86
Detergent (water)			ND-.152		0.024-0.138
Detergent (sediment)			2.33-18.84		1.79-28.50

Notes:
(a) From Onodera (1985). Range of measurements from 20 km to 58 km for Chao Phraya river.
(b) ONEB has designated Chao Phraya from 7 - 62 km as Class 4.
(c) Dry season.
(d) Rainy season.
(e) Length of BOD, i.e. 5 or 7 days, not reported.

Table B.5. Low Flow Conditions in the Chao Phraya River (Km 7–62), 1981–1984(a)

Parameter	Class 4 Standard(b)	1981	1982	1983	1984
DO, mg/l	2	1.1	1.5	1.2	1.3
BOD(c), mg/l	4	2.2	2.2	2.2	1.9
Coliform Bacteria, MPN/100 ml	20,000	63,000	>6700	209,300	163,000
Cadmium, mg/l	0.005	-	-	<.0005	0.009
Lead, mg/l	0.05	-	ND	0.012	0.008
Mercury, mg/l	0.002	-	-	0.005	0.0003
Salinity, mg/l	-	-	2.9	2.8	2.6

Notes:

(a) Source: ONEB (1988). Data reported over entire reach. No smaller intervals monitored.

(b) The Chao Phraya River from 7 - 62 km is designated Class 4 by the ONEB

(c) Length of BOD test, i.e. 5 or 7 days, not reported.

Table B.6A. Measurements of Organochlorine Pesticides in Chao Phraya and Klong Water from River Mouth to 142 Km

Pesticide	Chao Phraya River 1979 (a) Freq(c) (%)	Max (ppb)	Med (ppb)	Chao Phraya River September, 1982 (b) Freq(c) (%)	Max (ppb)	Med (ppb)	Chao Phraya River September, 1983 (b) Freq(c) (%)	Max (ppb)	Med (ppb)	Chao Phraya River April, 1984 (b) Freq(c) (%)	Max (ppb)	Med (ppb)	Chao Phraya River October, 1984 (b) Freq(c) (%)	Max (ppb)	Med (ppb)	Klongs Along the Chao Phraya October, 1984 (b) Freq(c) (%)	Max (ppb)	Med (ppb)	Class 3 Std(d) (ug/l)	Class 4 Std(e) (ug/l)
alpha BHC	0%			100%	0.3	0.003	100%	0.061	0.038	95%	0.056	0.002	100%	0.035	0.007	100%	0.072	0.055	0.02	0.02
beta BHC	0%			18%	0.008		0%			5%	0.024		0%			0%			0.02	0.02
gamma BHC (Lindane)	0%			6%	0.028		58%	0.082		27%	0.021		72%	0.032	0.003	91%	trace	0.001	0.02	0.02
Heptachlor	0%			0%			92%	0.377	0.152	41%	0.015		27%	0.166		82%	trace	0.001	0.2	0.2
Heptachlor epoxide	0%			0%			0%			32%			18%			0%			0.2	0.2
Chlordane	-			0%			0%			0%			0%			0%			nd	nd
Aldrin	0%			94%	0.092	0.025	83%	0.064	0.027	100%	0.288	0.126	100%	0.751	0.008	100%	0.028	0.018	0.1	0.1
Dieldrin	100%	0.117	0.09	21%	0.003		18%	0.13		100%	0.289	0.08	88%	0.442	0.029	100%	0.082	0.046	0.1	0.1
Endrin	8%	0.313		0%			0%			0%			0%			0%			nd	nd
p,p'-DDE	8%	0.034		0%			0%			36%	0.031		18%	0.03		45%	trace		nd	nd
o,p'-DDT	0%			18%	0.034		0%			0%			0%			0%			1	1
p,p'-DDD	8%	trace		0%			0%			9%	0.025		18%	0.06		18%	0.04		nd	nd
p,p'-DDT	25%	trace		0%			0%			13%	0.015		9%	0.271		0%			nd	nd
o,p'-DDE	0%			0%			0%						0			0%			nd	nd
o,p'-DDD	0%			0%			0%						0			0%			nd	nd

Notes:

(a) From Polprasert (1979), as cited in Onodera (1985).

(b) From Onodera (1985).

(c) Frequency of detection.

(d) ONEB has designated the Chao Phraya from 62-142 as Class 3.

(e) ONEB has designated the Chao Phraya from 7-62 as Class 4.

Table B.6B. Measurements of Organochlorine Pesticides in Chao Phraya Sediment (Up to 142 Km) and Klong Sediment

Pesticide	Chao Phraya River 1984 (a)			Klongs Along the Chao Phraya River 1984 (a)		
	Frequency (b) (percent)	Maximum (ppb)	Median (ppb)	Frequency (b) (percent)	Maximum (ppb)	Median (ppb)
alpha BHC	92%	3.3	1.32	100%	0.7	0.29
beta BHC	0%			0%		
gamma BHC (Lindane	92%	15.9	8.27	0%		
Heptachlor	0%			89%	100	3.5
Heptachlor epoxide	17%	1		0%		
Chlordane	0%			0%		
Aldrin	53%	2.8	1.2	100%	16.6	2.78
Dieldrin	94%	6.1	2.03	100%	15.4	4.2
Endrin	6%	3		0%		
o,p'-DDE	0%			0%		
p,p'-DDE	62%	5.6	3.18	100%	1.54	0.71
o,p'-DDD	35%	1.5		0.7	2	0.72
p,p'-DDD	87%	18	3.43	1	15	4.6
o,p'-DDT	0%			0%		
p,p'-DDT	37%	19.8		50%	58	

Notes:

(a) From Onodera (1985).

(b)Frequency of detection.

concentrations of aldrin that exceeded the standard by three to seven times. DDT was also detected during 1984 sampling.

Higher mean concentrations of pesticides were observed in klong water than in the river, indicating that polluted klongs are a possible source of contamination of the river. Mean concentrations of BHC in the klongs in October 1984 would have exceeded the Class 3 and 4 water quality standard by about a factor of two, if these standards were applied to the klongs. Notably, concentrations were 10-100 times higher in sediments than in water for both the klongs and the river, indicating an accumulation of pesticides that can serve as a reservoir of contamination. The highest water concentration of pesticides was found near the river mouth, while the highest concentration in the sediments was found 40-60 km upstream, indicating that the sediment is possibly acting as a reservoir for downstream transport of pesticides.

Heavy metal data for river and klong water and sediment are shown in Tables B.7a and B.7b. Maximum observed concentrations for cadmium, chromium, lead, and mercury all exceeded their respective water quality standards in 1977 sampling. These metals did not exceed the standards in 1984 sampling; however, this may reflect a change in techniques used to analyze the water samples, rather than an improvement in water quality. Onodera found that heavy metal concentrations were higher downstream than upstream. As with pesticides, metals were found to accumulate in sediments. In klong waters, high concentrations of copper, lead, cadmium and mercury were found in two or more klongs. The most contaminated klongs were Bang-Nangkreng, Samrong, and Phra Khanong. As with pesticides, the maximum concentration in sediments is upstream of the maximum concentration in water, supporting the idea that sediment acts as a reservoir of contamination. Higher concentration of metals in klongs than in river indicate that polluted klongs are a source of river contamination.

Groundwater Data

We obtained data on groundwater quality for six provinces surrounding Bangkok. Results from sampling in these regions are shown in Table B.8. We were unable to obtain groundwater quality data for aquifers underlying Bangkok; therefore, for the purposes of this analysis, we assumed that contaminants in Bangkok wells could be represented by the average contaminant concentrations for wells less than 100 kilometers from Bangkok. Importantly, this method ignores local sources of groundwater contaminations, such as open dumps or spills, which could dramatically affect groundwater concentrations.

Table B.7A. Measurements of Heavy Metals in Chao Phraya and Klong Water from River Mouth to 142 Km

	Chao Phraya River 1977 (a)			Chao Phraya River September, 1984 (b)			Klongs Along the Chao Phraya Rive October, 1984 (b)			Class 3 Standard (c)	Class 4 Standard (d)
Metal	Minimum (ppb)	Mean (ppb)	Maximum (ppb)	Minimum (ppb)	Mean (ppb)	Maximum (ppb)	Minimum (ppb)	Mean (ppb)	Maximum (ppb)	(ppb)	(ppb)
Iron				2043	4117	6881	1356.16	2709	2866		
Manganese				10	124	330	97	315	1187	1000	1000
Cadmium	ND(e)	8.4	121.1	ND	2.52	4.29	0.24	0.65	1.46	5	5
Copper	ND	19.1	68.7	4.81	16	66.9	ND	17.2	48.2	100	100
Chromium	ND	18.8	123.3							50	50
Lead	ND	42.8	242.6	ND	2.07	4.05	ND	4.54	8.86	50	50
Zinc	11.2	38.7	177.8	8	65.6	162	32.8	56.1	91.9	1000	1000
Mercury	0.94	2.3	8.2	ND	0.2	0.43	ND	0.2	0.43	2	2
Nickel				2.75	18.6	137	2.73	10.3	27.3		

Notes:

(a) From Polprasert (1979), as cited in Onodera (1985).

(b) From Onodera (1985).

(c) ONEB has designated the Chao Phraya from 62-142 as Class 3.

(d) ONEB has designated the Chao Phraya from 7-62 as Class 4.

(e) ND = not detected.

Table B.7B. Heavy Metals in Upper Chao Phraya Sediment (Up to 142 Km) and Klong Sediment

Metal	Chao Phraya River 1979 (a)			Chao Phraya River September, 1984 (b)			Klongs Along the Chao Phraya River October, 1984 (b)		
	Min (ppm)	Mean (ppm)	Max (ppm)	Min (ppm)	Mean (ppm)	Max (ppm)	Min (ppm)	Mean (ppm)	Max (ppm)
Iron				187	695	1406	281	1308	2437
Manganese				340	771	1119	262	572	924
Cadmium	0.5	2.74	5.47	ND	0.4	0.97	0.32	0.54	0.97
Copper	3.34	13.4	37.5	10.4	22.2	41.7	14.5	44.9	118
Chromium	ND(d)	17.86	47.5						
Lead	50	81	195	8.9	22.6	92	11.8	65.6	356
Zinc	20.08	57.05	170.5	41.4	121	218	111	275	614
Mercury (c)	0.08	0.279	1.86	0.2	1.16	2.81	0.2	0.94	2.81
Nickel				13.8	21.8	32.9	17.3	35.1	103

Notes:

(a) From Polprasert (1979), as cited in Onodera (1985).

(b) From Onodera (1985).

(c) Measurement in ppb.

(d) ND = not detected.

Table B.8. Deepwell Groundwater Water Quality Data for Several Provinces Near Bangkok, Thailand, 1989(a)

Parameter	Units	Province (km from Bangkok) Lopburi (153)	Ayuthaya (76)	Singburi (142)	Samutsakorn (36)	Ratchaburi (Well 1) (100)	Ratchaburi (Well 2) (100)	Chai Nat (194)	Average (b)	Thai Groundwater Standards (c)
pH	pH units	-	-	8.2	7.5	7	6.5	7	7.00	7.0-8.5
Color	Color units	25	-	10	15		15	15	15.00	5
Turbidity	JTU	17	-	5.9	2.6	12	60	23	25.00	5
Hardness	mg/l	308	-	100	196	956	280	162	480.00	300
Fe	mg/l		-	0.12	0.05	0.72	2.7	1.9	1.20	0.5
Mn	mg/l		-	0.02	0.04	0.28	1.1	0.73	0.47	0.3
Cu	mg/l	0.0045	0.0045	0.0045	0.0045	0.01	0	0.009	0.00	1
Zn	mg/l	0.09	0.19	0.42	0.08	0.49	0.51	0.44	0.32	5
Pb	mg/l	0	0	0.05	0.01	0.05	0	0	0.02	none
Cr	mg/l	0.005	0	0	0	0.01	0	0.01	0.00	
Cd	mg/l	0	0	0.004	0	0.003	0	0.003	0.00	none
As	mg/l	0	0							none
Sulphate	mg/l	-		20.1	5.2	12.1	29.2	16.5	16.00	200
Cl	mg/l	7	39	15	30	38.5	40.3	19	37.00	200
NH3	mg/l	0.05								
NO3	mg/l			0.2	0.4	3.2	0.3	0.3	1.30	45
Fl	mg/l	0.22	0.14	0.29	0.78	0.05	0.2	0.21	0.29	1

Notes:

(a) Source: MOPH, Department of Health, Division of Environmental Health

(b) Average of wells 100 kilometers or fewer from Bangkok (Ayuthaya, Samutsakorn, Ratchaburi)

(c) Groundwater Standards for Drinking Purposes (from (ONEB, 1989))

According to ONEB (1988), in 1987, domestic, agricultural and industrial wells extracted an estimated 500,000 m^3 per day of groundwater in Bangkok. Of these, 2970 were domestic wells with a flow of about 260,000 m^3/day. Assuming a domestic demand of 210 lpcd, this implies that the population served by private domestic wells in Bangkok is about 1.2 million persons, or about 22% of the population of Bangkok. This corresponds well with the estimated 25% of the population not served by the MWA water supply.

III. Analytical Methodology Used to Estimate Risk

Several approaches were used to analyze the risks from water pollution in Bangkok. We evaluated risks from drinking water, direct water contact, and irrigation water. Methods used for each assessment are described below. Methods for estimating risks from consumption of contaminated fish are discussed in Appendix C.

Risks from Drinking Water

The first step to estimate risks from drinking water was to obtain estimates of water contamination levels. We examined risks from drinking both surface water and groundwater.

Drinking water from surface water supplies

When available, we used average tap water concentrations from MWA sampling to characterize concentrations of contaminants in drinking water. However, the tap water data did not contain information on a number of metals and pesticide contaminants for which surface water data are available. One approach to estimate tap water concentrations would be to apply an estimated treatment removal efficiency to raw water data for these contaminants. To do so, we must know the type of treatment system used, and its effectiveness in removing contaminants of interest. According to MWA (1989), the treatment train for the Bangkok water supply consists of screening, coagulation, chlorine treatment, and sand and gravel filtration. Sources disagree about the extent of removal of contaminants achieved by this kind of treatment system. According to Hammer (1986), many water contaminants are not significantly affected by this type of conventional chemical water treatment (these are termed "refractory" compounds). These contaminants include metals such as chromium and cadmium, as well as organics such as pesticides. Therefore, to be conservative, we assumed no removal and used raw water data from water sampled at 91 km (the location of the MWA water

intake) in order to estimate drinking water concentrations of these refractory contaminants. The 1989 MPH Chao Phraya data printout did not have any pesticides data, so we used pesticides water quality data from the 91 km reach in the 1985 Onodera study (or from the 82 km reach if 91 km data were not reported) to estimate the concentration of pesticides in the Chao Phraya at the point of water intake. Average concentrations were used to represent typical exposure for calculation of population risks.

Water concentration estimates can be compared to the various environmental standards; however, compliance with standards does not necessarily mean that there is no health risk associated with drinking the water, since standards are often not strictly health-based (that is, economic and technical considerations are often taken into account when they are established). Even if the water concentration of a particular contaminant does not exceed its standard, it may still contribute to risk. Consequently, this analysis estimates risk based on estimated exposure, even when concentrations are below the standards.

To estimate exposure, we calculated contaminant intake from water in terms of dose per kilogram (kg) body weight per day. This was achieved by multiplying the water concentration by the assumed water consumption rate. We used two liters as the amount of water consumed per person per day. This value does not seem unreasonable in light of the fact that the national water supply goal for drinking water is 5 lpcd, which includes drinking water, washing, toothbrushing, and cooking. Furthermore, this value is used by the U.S. EPA for U.S. consumption, and was used by Fingleton et al. (1989) in a paper on a risk-based approach for the selection of hazardous waste disposal sites in Thailand. Finally, we divided by the average Thai body weight to obtain the dose in mg/kg/day.

Individual risk estimates for noncarcinogens were estimated by comparing the dose to RfDs, using methods described in Appendix G. A dose in exceedance of the RfD suggests the possibility of an adverse health effect occurring, but does not predict the actual probability of the health effect occurring, or the severity of the effect. Cancer risk estimates were derived for the carcinogenic pesticides using methods described in Appendix G. Special methods were used for calculating the risks from ingestion of lead in drinking water. These methods, and the results, are found in Appendix E.

Risks from microbiological contaminants in drinking water were estimated through different procedures, described in Appendix F. The approach used for other contaminants (i.e., applying dose

-response relationships to estimated concentrations of chemicals in tap water) is not applicable for microbiological contaminants, for two reasons. First, there are no satisfactory dose-response relationships allowing estimation of likely disease incidence. Second, infiltration into the distribution system is the primary means by which these contaminants enter tap water. This process is highly variable throughout the distribution system, and the resulting concentrations of microbiological agents at the tap will vary substantially across the city. Extensive at-the-tap monitoring data would be needed to characterize this variation, and such data were not available. Instead, in Appendix F, the risk assessment procedure for microbiological contaminants involves evaluating the overall incidence of microbiological disease in Bangkok.

For population risk estimates, we assumed that 75 percent of the Bangkok population drinks surface-supplied drinking water. This may be an conservative assumption, since many people drink bottled water rather than tap water, and many boil tap water that they do drink. (Boiling would have no effect on metals content of the water, and would probably have little effect on the pesticide content of the water). Population risk estimates are equal to typical individual lifetime risk times the size of the Bangkok population. This figure is annualized by dividing by 70 years, assumed to be the length of the average lifespan.

Drinking water from groundwater supplies

The average groundwater contaminant levels for wells less than 100 kilometers from Bangkok (presented in Table B.8.) were assumed to be equal to the concentrations in drinking water from groundwater supplies in Bangkok. Exposure was then estimated using the technique described above for surface water supplies. However, for population risk estimates, we assumed that only 25 percent of the Bangkok population uses groundwater for drinking and cooking, and is thus exposed to these concentrations in drinking water. Lead (Appendix E) and microbiological contaminants (Appendix F) were handled separately.

Risks from Direct Contact

Surface water concentrations (Chao Phraya and klong waters) can be compared to typical water quality standards for protection of persons in direct contact with surface water. These standards are set to protect against health effects that may result from ingestion or dermal contact with water during bathing or swimming (or other casual/incidental contact).

Hammer (1986) presents typical standards set by states in the U.S. for water contact recreation.

The coliform bacteria standards suggest a mean of 1000 (200 fecal) per 100 ml with not more than 10 samples exceeding 2000 (400 fecal). Recommended dissolved oxygen levels are 4 to 5 ppm (higher levels of DO tend to enhance water quality due to increased degradation of organic contaminants, and decreased odors). Direct water contact standards based on coliform are controversial, since direct contact can lead to eye, skin and respiratory diseases, as well as enteric diseases. Studies have shown that the vast majority of diseases among recreational water users in the U.S. were eye, nose, throat, ear, and skin infections, not gastrointestinal disorders (Waite, 1984). The relationship between coliform bacteria, which indicate the possible presence of enteric pathogens that cause gastrointestinal disorders, and the onset of these other illnesses, is unclear. However, Waite (1984) describes studies that show that the rates of various illness among swimmers is related to water quality, and discusses correlations that indicate that the total incidence of illness decreases significantly when total coliforms are below 1000 per 100 ml. Despite the evidence that a relationship between illness rate and the presence of coliform bacteria exists, there are no dose-response functions from which numerical risk estimates can be made. Therefore, this analysis compares water concentrations to typical standards for water contact.

We do not know the size of the population that uses the Chao Phraya or klongs for these purposes. However, according to ONEB (1988), there were 21,592 canal houses in 1984. Assuming an average household size of 5.6 persons, this leads to a canal population of about 121,000 persons. This does not include those persons who live along the river. In addition, there are many people who use the river for commerce and for commuting. These persons may also be exposed to surface water contaminants, especially through inhalation of contaminants that volatilize from the river surface.

Risks from Irrigation

Using contaminated klong or river water to irrigate crops may result in uptake of contaminants by the crops, which are subsequently consumed by humans or livestock. A sophisticated analysis, which models exposure by considering the specific contaminant concentrations in water used for irrigation, contaminant-specific crop uptake rates from irrigation water and the quantity of contaminated crops consumed by Bangkok residents, is not possible due to data constraints. However, some general limits on metal concentrations and fecal coliform levels in irrigation water are available (Eckenfelder, 1980). For this analysis, we compare river water, klong water, and groundwater contaminant concentrations to these guidelines to qualitatively estimate possible risks from agricultural use of these waters. In addition, Appendix C discusses risks from pesticides and heavy metals in food.

Risks from Fish Consumption

Fish and other aquatic life may bioconcentrate contaminants from the water they live in. Human health risks can result when contaminated fish and shellfish are consumed. The analysis of food contamination in Appendix C estimates these risks by combining information on contaminant levels in marine animals with data on average Thai dietary consumption of fish and shellfish.

Risks to Aquatic Life

This analysis did not compare Chao Phraya and klong water quality to standards for protection of aquatic life, but they would be exceeded in many cases. Although assessment of ecological effects is beyond the scope of this work, it is important to note that the most severe effects of water pollution may, in fact, be the effects on the aquatic ecosystem.

IV. Discussion of Results

Risks from Drinking Water

We compared the estimated drinking water concentrations to Thai drinking water standards and to U.S. Maximum Concentration Limits (ONEB, 1989). The results are shown in Table B.9. All average concentrations are below the Thai drinking water standards; however, the maximum value for lead exceeds the Thai drinking water standard. Health effects from lead exposure are addressed specifically in Appendix E.

Calculated risks from ingestion of surface supplied water are found in Table B.10. This table shows that the highest population cancer risk is associated with the ingestion of aldrin and heptachlor. None of the estimated exposures to metals exceeds the Reference Dose (RfD). In fact, none exceeds 10 percent of the RfD. Calculated risks from groundwater are found in Table B.11. We had no data on the concentrations of pesticides or other carcinogens in water; therefore, we cannot estimate cancer risks from ingestion of groundwater. The exposure from ingestion of groundwater does not exceed the RfD for any of the contaminants for which data were available. These results suggest that, with the exception of lead and microbiological contaminants, there is little risk associated with the drinking water supply in Bangkok. For a discussion of risks from lead in drinking water,

Table B.9. Comparison of Estimated Tap Water Concentrations to Drinking Water Standards

Parameter	Estimated Tap Water Concentration (mg/l) Surface Water Source (1) Average	Data Source (1)	Estimated Tap Water Concentration (mg/l) Ground Water Source (2) Average	U.S. MCLs	Thai Drinking Water Standards	Thai Groundwater Standards (3)	Notes
Aldrin	0.000027	(c)			0.03		(4), (5)
Cadmium	0	(b)	0.001	0.01	0.01	none	(6)
Chloride	17	(a)	37	250		200	
Chromium VI	0	(b)	0.003	0.05	0.05		
Copper	0	(b)	0.005	1	1	1	
p,p'-DDD	0.00002	(c)					
Dieldrin	0.000057	(c)			0.03		(4), (5)
Fluoride	0.31	(a)	0.29	4	0.7	1	
Heptachlor	0.00012	(c)			0.1		(4), (7)
Iron	0.14	(a)	1.2	0.3	0.5	0.5	
Lead	0.02	(a)	0.02	0.005	0.05	none	(8)
alpha BHC	0.000042	(c)			3		(4)
Lindane (gammaBHC)	0.000027	(c)		0.004	3		(4)
Manganese	0.06	(b)	0.47	0.05	0.3	0.3	
Mercury (ug/l)	0.0003	(b)		0.002	0.001		
Nickel	0.011	(d)					
Sulfate	3.9	(a)	16	250	200	200	
Tin	0.03	(a)					
Zinc	0.06	(b)	0.32	5	5	5	

Notes:

(1) Data sources:

(a) From MWA tap water data, 1989.

(b) Derived from concentration of metal in surface water at 91 km (1989 MOPH data); assuming no removal by treatment system.

(c) Pesticide concentration from Onodera (1985). Avg of detected values, 1982-1984. Concentration measured at 82 or 91 km; assuming no removal by treatment system.

(d) Metal concentration from Onodera (1985). Concentration measured at 91 km; assuming no removal treatment system.

(2) Groundwater values are average values from Table B.8.

(3) Groundwater standards for drinking purposes (from ONEB (1989)).

(4) New parameter being considered by the technical committee Group 5 of Thai Industrial Standards Institute.

(5) Standard is for aldrin and dieldrin combined.

(6) Standard for Cd being revised to 0.005 mg/l by the technical committee Group 5 of Thai Industrial Standards Institute.

(7) Standard is for heptachlor and heptachlor epoxide.

(8) Proposed US MCL for lead is 0.005 mg/l (53FR31516).

Table B.10. Estimated Risk from Ingestion of Surface Water Supply

Parameter	Average (mg/l)	Maximum (mg/l)	Data Source (1)	CAG Class (2)	Cancer Potency Estimate (mg/kg/day)-1	RfD (mg/kg/day)	Average Exposure Surface Water (mg/kg/day)	Population Cancer Risk Surface Water (cases/yr)(3)	Average Exposure as % RfD (percent) (4)
Aldrin	2.70E-05	4.90E-05	(c)	B2	17	3.00E-05	1.00E-06	1.30E+00	3.3%
Chloride	1.70E+01	1.70E+01	(a)				6.20E+01		
Chromium VI	0.00E+00	1.00E-02	(b)			5.00E-03	0.00E+00		0.0%
p,p'-DDD	2.00E-05	2.00E-05	(c)	B2	0.24	5.00E-04	7.40E-07	1.40E-02	0.1%
Dieldrin	5.70E+00	8.20E-05	(c)	B2	1.6	5.00E-05	2.10E-06	2.60E-01	4.2%
Fluoride	3.10E-01	5.00E-01	(a)				1.20E-02		
Heptachlor	1.20E-04	1.20E-04	(c)	B2	4.5	5.00E-04	4.40E-06	1.50E+00	0.9%
Iron	1.40E-01	1.50E-01	(a)				5.20E-03		
Lead(5)	2.00E-02	1.00E-01	(a)				7.40E-04		
alpha BHC	4.20E-05	1.30E+00	(c)	B2/C	1.3	3.00E-04	1.60E-06	1.60E-01	0.5%
Lindane	2.70E-05	2.70E-05	(c)	B2/C	1.3	3.00E-04	1.00E-06	1.00E-01	0.3%
Manganese	6.00E-02	9.00E-02	(b)				2.20E-03		
Mercury(ug/l)	3.00E-04	5.90E-04	(b)			3.00E-04	1.10E-05		3.7%
Nickel	1.10E-02	-	(d)			2.00E-02	4.10E-04		2.1%
Sulfate	3.85E+00	5.50E+00	(a)				1.40E-01		
Tin	3.00E-02	1.10E-01	(a)				1.10E-03		
Zinc	6.00E-02	8.00E-02	(b)			4.70E-01	2.20E-03		0.5%

Notes:

(1) Data sources:

(a) From MWA tap water data, 1989.

(b) Derived from concentration of metal in surface water at 91 km(1989 MOPH Data), assuming no removal by treatment system(Hammer,1986).

(c) Pesticide concentration from Onodera(1985). Avg of detected values,1982-1984.Concentration measured at 82 or 91 km; assuming no removal by treatment system.

(d) Metal concentration from Onodera (1985). Concentration measured at 91 km; assuming no removal by treatment system.

(2) EPA's Cancer Assessment Group Weight of Evidence Classification. See Appendix H for details.

(3) See Appendix H for the calculations for population and maximum individual cancer risk.

(4) Calculated as: (Exposure/RfD) x 100

(5) See Appendix F.

Table B.11. Estimated Risk From Ingestion of Groundwater

Parameter	Tap Water Concentration (mg/l) Groundwater Source (a) Average	RfD	Average Exposure Groundwater (mg/kg/day)	Average Exposure as % RfD (percent) (b)
Cadmium	1.00E-03	5.00E-04	3.70E-05	7.4%
Chloride	3.70E+01		1.40E+00	
Chromium VI	3.00E-03	5.00E-03	1.10E-04	2.2%
Copper	5.00E-03	4.00E-02	1.90E-04	0.5%
Dieldrin		5.00E-05	0.00E+00	0.0%
Fluoride	2.90E-01		1.10E-02	
Iron	1.10E+00		4.07E-02	
Lead	2.00E-02		7.40E-04	
Manganese	4.70E-01		1.70E-02	
Nitrate	1.30E+00		4.80E-02	
Sulfate	1.60E+00		5.70E-01	
Zinc	3.20E-01	4.70E-01	1.20E-02	2.6%

Notes:
(a) Groundwater values are average values from Table B.8.
(b) Calculated as: (Exposure/RfD) x 100

see Appendix E. Risks from microbiological disease are discussed in Appendix F.

Risks from Direct Contact

Data from Tables B.2 and B.3 show that the river water and klong water are well above the 200 fecal coliforms per 100 ml suggested by Hammer (1986) for direct contact with water. The average concentration of fecal coliform bacteria in the river exceeded 100,000 MPN/100 ml at one station; average coliform concentrations in one of the two klongs for which data were reported exceeded 500,000 MPN/100 ml. Those living along canals (perhaps 121,000 persons) and the river, and any others in contact with the river or klongs, may be at risk from swimming, bathing, or otherwise using Bangkok's surface water. More persons may be exposed when Bangkok floods. Gastrointestinal disease or skin, eye or respiratory tract illnesses may be the result. In fact, it has been noted that persons with open wounds who come into contact with klong water often suffer from infections of those wounds (personal communication, Dr. Samporn Surarith).

Risks from Fish Consumption

Cancer risks from ingestion of marine organisms are calculated in Appendix C. Using information presented in that Appendix, it is estimated that risks from consumption of contaminated fish are minimal, contributing only about two cancer cases every ten years. With the exception of mercury, contamination of fish by heavy metals also appears to contribute little to risk. For mercury, fish consumption accounts for over half of the exposure to mercury through the diet. It should be noted that we do not know the origin of the fish included in Bangkok residents' diets; therefore, it is uncertain if the contamination found in these organisms is attributable to pollution from Bangkok, or to pollution originating elsewhere.

Risks from Irrigation

Average concentrations from all stations monitored were compared with irrigation guidelines discussed by Eckenfelder (1980). The comparison is found in Table B.12. The average surface water concentrations do not exceed these guidelines. Groundwater and klong water are also well within these guidelines.

Table B.12. Comparison of Surface and Groundwater Quality to Irrigation Criteria

Parameter	Suggested Value for Irrigation (a)	Average Concentration In Chao Phraya 1989 (b)	Average Concentration In Groundwater 1989 (b)	Average Concentration in Klong Ratchburana 1989 (b)	Average Concentration in Klong San Seb 1989 (b)
Cadmium	1.00E-02	2.00E-03	1.00E-03	3.00E-03	4.00E-03
Chromium VI	1.00E-01	8.00E-03	3.00E-03	1.00E-02	1.00E-02
Copper	2.00E-01	6.00E-03	5.00E-03	1.00E-02	1.00E-02
Iron	5.00E+00	1.70E+00	1.16E+00	8.40E-01	1.53E+00
Lead	5.00E+00	1.60E-02	2.00E-02	2.00E-02	2.00E-02
Zinc	2.00E+00	7.80E-02	3.20E-01	9.00E-02	8.00E-02
Fecal Coliform	2.00E+03	5.89E+04	NA(c)	5.50E+05	1.40E+04
Total Coliform	2.00E+04	1.94E+05	NA	1.40E+07	3.10E+04

Notes:
(a) From Eckenfelder (1980).
(b) From MOPH 1989 Data.
(c) NA = not applicable.

Sources of Contamination

The MOPH/DOH/EHD conducted a study of the incoming wasteloads of 22 rivers to the Gulf of Thailand (MPH, 1989). Table B.13. shows the estimated input of the Chao Phraya compared to the total input to the Gulf of Thailand. The Chao Phraya was found to contribute 25% of the total phosphorus and 41% of the nitrogen. The Chao Phraya also contributed about 22% of the heavy metals. Based on population, the estimated load to the Gulf of Thailand from different cities was estimated. Bangkok contributed an estimated 67% of the BOD and 67% of the suspended solids.

According to an ONEB summary of water quality in the Chao Phraya (ONEB, 1990), the percentage contributed by various activities to waste in the Chao Phraya is 25% for industry, 27% for restaurants, 40% for domestic sources, and 8% from other sources. An earlier ONEB study (ONEB, 1988) also estimated that about 75% of the waste was from nonindustrial sources, but estimated that 54% was from houses. According to a 1968 estimate cited by ONEB (1988), the industrial contribution was only about 5 percent at that time; industrial contribution appears to be on the rise.

V. Limitations

This analysis is subject to important limitations. The most critical shortcoming may be the lack of data on several types of contaminants of concern. Data on pesticides in groundwater and data on organics (other than pesticides) and radionuclides in surface water and groundwater were not available. Furthermore, data were not available regarding the presence or concentrations of disinfection byproducts in treated water supplies. However, based on information regarding the behavior of these contaminants in the environment, and the experience with these contaminants in the U.S., we can speculate about the type and, to some extent, the degree of risk that might exist in Bangkok from these pollutants:

- Pesticides. Contamination of groundwater by pesticides is usually a localized phenomenon, and thus does not generally pose widescale population risks. Furthermore, many organochlorine pesticides are relatively insoluble in water; their concentrations in water usually result in low estimated risks. Pesticide contamination of groundwater will be found in agricultural areas with highly transmissive soils or where improper well construction provides a conduit to the surface. The geology under Bangkok consists of clay layers with at least 6 underlying aquifers; the thickness

Table B.13. Contribution of the Chao Phraya to Incoming Waste Loads to the Gulf of Thailand, 1982–1988 (a)

Pollutant	Average Annual Waste from Chao Phraya (tons/year) 1982-1988	Total Waste Load to the Gulf of Thailand (tons/year) 1982-1988	Contribution of the Chao Phraya (percent)
Suspended solids	1.04E+06	3.29E+06	32%
Phosphate	5.42E+03	2.17E+04	25%
Nitrate	2.74E+04	5.27E+04	52%
Total Nitrogen	1.45E+04	4.88E+04	30%
BOD(c)	2.78E+04	8.93E+04	31%
Coliform Bacteria (b)	1.04E+04	1.70E+04	61%
Faecal Coliform (b)	3.29E+03	5.40E+03	61%
Cadmium	4.13E+01	2.97E+02	14%
Lead	3.93E+02	2.87E+03	14%
Magnesium	4.80E+00	4.49E+01	11%
Manganese	3.14E+03	9.57E+03	33%
Chromium	1.12E+02	7.75E+02	14%
Copper	1.15E+02	7.26E+02	16%
Iron	2.03E+04	9.30E+04	22%
Zinc	1.83E+03	7.50E+03	24%

Notes:

(a) Source: MOPH, DOH, EHD (1989). Figures derived from river quality monitoring and river flow data.

(b) Units are 1.00E+14 MPN/year

(c) Length of BOD test, i.e. 5 or 7 days, not reported.

of the clay layer averages 10-30 meters in depth. The uppermost stratum is 25 meters (ONEB, 1988). The thick clay layer would tend to isolate groundwater from local surface sources of contamination. Therefore, given the protective geology underlying the city, and considering the largely urban nature of Bangkok, it is unlikely that pesticides will present a substantial population risk problem in Bangkok groundwater.

Other organics. Volatile organic compounds (VOCs), PCBs, dioxin, and other synthetic organic compounds could represent a significant source of risk in surface waters. VOCs readily volatilize from surface waters, and may serve as a source of risk through inhalation for persons residing on or near the river, close to a source of discharge. Synthetic organic compounds are often relatively insoluble in water, but tend to bioaccumulate in fish and other aquatic organisms, and may pose risks to humans through fish comsumption. Given the amount of waste discharged to the Chao Phraya (and ultimately, to the Gulf of Thailand), we can guess that these chemicals may be a potentially significant source of risk.

Synthetic organic chemicals are found with moderate frequency in groundwater in the U.S., adding up to modest risk nationwide. Sources include leaking from municipal or industrial waste dumps, or leaking underground chemical storage tanks. Again, Bangkok's geology is probably protective, and thus these chemicals should not constitute a substantial population risk through groundwater contamination. There are data, however, to suggest substantial organic contamination in Bangkok's dumps, and groundwater contamination around these point sources could be a problem if wells are improperly constructed so as to provide a conduit from the surface through protective clay layers.

Disinfection byproducts. The disinfection of public water supplies with chlorine can lead to the formation of carcinogenic byproducts, such as chloroform. Although the concentrations of these byproducts will vary, typical individual risks from drinking publicly supplied water in the U.S. are in the range of 1×10^{-4}. If this individual risk is also representative of the Bangkok water supply, over a population of 4.4 million (or about 75 percent of the population of Bangkok), exposure to these byproducts could lead to about 400 cancer cases per year.

- Radionuclides. Radionuclides are frequently found in U.S. groundwater. However, the presence of radionuclides is wholly dependent upon the particular geology of the aquifer. We have no information regarding the presence or absence of radionuclides in Bangkok aquifers.

This analysis is also limited by the type and quality of the data we were able to obtain on surface and groundwater quality. For example, the results of the analysis are dependent on the accuracy of the estimates of the contaminant concentrations in drinking water. Furthermore, we assume that 75 percent of the Bangkok population drinks water containing these levels of contaminants. In fact, many people use bottled water or boil tap water before use, but we do not know extent of this practice, or its effect on concentrations of contaminants, except that it is unlikely to affect concentrations of metals and many pesticides.

We have no data about how the distribution system affects concentrations for either surface supplied water or groundwater. Contamination of the water supply could vary depending on the area of the city because of the distribution system. Some points in the system have low pressure, so citizens use pumps to draw water. This creates negative pressure in the pipes. Leaks in pipes with negative pressure could cause contamination from surrounding soils, contaminated shallow groundwater, of klongs.

Finally, we have little information about the size of the population exposed through direct contact, nor do we know the extent to which direct contact may actually lead to illnesses. We have no socioeconomic/demographic information for the exposed population, nor do we know the proportion of the exposed population that would actually contract an illness or the nature of the illnesses that might be contracted.

Appendix C. Health Risks from Ingestion of Contaminated Food

I. Definition of the Problem

A variety of chemical and microbiological contaminants may find their way onto or into food consumed by Bangkok residents. In this project, some sources of food contamination considered include residues of pesticides applied to vegetables when they were grown, toxic pollutants in water that bioconcentrate in fish, or bacteria carried by flies breeding in untreated waste that find their way into meats. Other sources of food contaminants may be manmade, but are not within the scope of environmental problems we consider in this project. Examples include contamination of food during packaging or handling (e.g., metal residues from canning) and organic carcinogens formed when meats are grilled. Finally, we also do not consider natural contaminants in food, such as the carcinogen aflatoxin, which is found in peanuts and some other commodities. The three classes of contaminants in food that we will examine in this project include: microbiological agents whose prevalence in Bangkok is a result of environmental conditions (discussed in Appendix F); pesticides; and metals.

Pesticides

When pesticides are applied to crops, residues of the pesticides may remain on the crop surface, or may be incorporated systemically into the plant. Pesticides may also be applied to crops after harvest to prevent spoiling during transport and storage. Livestock may ingest pesticides through grazing on treated land, or by ingested feed that has been treated with pesticides; therefore, meat products and milk may also contain pesticide residues. In fact, certain lipophilic pesticides have the tendency to bioconcentrate in the fatty tissues of animals, resulting in concentrations in animal products that are higher than feed concentrations. In addition, fish can bioconcentrate pesticides to levels several orders of magnitude greater than the concentrations of the pesticide in the water in which they reside. Pesticide residues remaining in and on food will be ingested by the consumer. The magnitude of exposure to these pesticides will depend on the quantity of food ingested and the residues remaining on each food after transport, handling and preparation.

Metals

Metal contaminants may also be detected in food. Metal contamination may result from the plant uptake of metals from the environment during transport and marketing, from the application of pesticides containing metals (e.g., certain fungicides contain zinc), from deposition of airborne metals on foods, or from processing and/or canning of foods.

II. Data Acquired

Three categories of information are needed to characterize risk from ingestion of contaminated food: (1) dose-response information that projects the likelihood of adverse health effects occurring as a function of the dose of each chemical ingested; (2) the daily consumption rate of dietary items; and (3) the concentration of contaminants in each dietary item.

For this assessment, cancer potencies and reference doses (RfDs) were obtained from the U.S. Environmental Protection Agency for as many of the compounds of interest as possible. These values are displayed in Table C.1. Derivation and use of these values is discussed in detail in Appendix G.

Data on daily consumption of various commodities were obtained from the Nutrition Division, Department of Health, Ministry of Public Health. Average consumption values for the year 1976 were provided for twenty-two food categories, in units of grams per person per day. These data are displayed in Table C.2. While the consumption data reflects dietary patterns for urban Thais, these data are not specific to Bangkok. For use in this assessment, the consumption data were converted to the units of grams per kg body weight per day (see Section III. for a detailed explanation). Data on average body weight of Thai people were also provided by the Nutrition Division.

Concentrations of pesticide residues and metals concentrations in food were provided by the Thailand Ministry of the Public Health, Food and Drug Administration (Thai FDA). The Thai FDA provided average pesticide residue concentrations in domestic samples analyzed between 1982 and 1985. Results were available for 25 pesticides in nine commodity categories. The range of results, the average and the percent of samples in which the pesticide was detected were provided. These data, displayed in Table C.3, reflect the pesticide residues for the country as a whole. Residues for

Table C.1. US EPA Cancer Potency Estimates and RfDs for Various Pesticides and Metals

Compound	Weight of Evidence Group	Human Cancer Potency (mg/kg/day)-1	RfD (mg/kg/day)	Notes
Aldrin	B2	1.70E+01	3.00E-05	
Arsenic	A	0.00E+00	1.00E-03	(1)
Azinphos-ethyl	D	1.50E-07	1.30E-03	
Cadmium	B1	0.00E+00	5.00E-04	(1)
Captan	B2	2.30E-03	1.30E-01	
Chlordane	B2	1.30E+00	6.00E-05	
Chlorpyrifos		0.00E+00	3.00E-03	(2)
Chromium VI	A	0.00E+00	5.00E-03	
Copper		0.00E+00	4.00E-02	(2)
DDD	B2	2.40E-01	5.00E-04	
DDE	B2	3.40E-01	5.00E-04	
DDT	B2	3.40E-01	5.00E-04	
Diazinon		0.00E+00	9.00E-05	(2)
Dicofol		0.00E+00	1.00E-03	(2)
Dieldrin	B2	1.60E+00	5.00E-05	
Dimethoate	D	0.00E+00	2.00E-04	(3)
Endosulfan	D	0.00E+00	5.00E-05	(3)
Endosulfan sulfate		0.00E+00	none yet	(2)
Endrin	D	0.00E+00	3.00E-04	(3)
EPN		0.00E+00	1.00E-05	(2)
Fenitrothion		0.00E+00	4.00E-03	(2)
Heptachlor	B2	4.50E+00	5.00E-04	
Lead	B2	0.00E+00	1.00E-07	(1)
Lindane	B2/C	1.30E+00	3.00E-04	
Lindane (BHC)	B2/C	1.30E+00	3.00E-04	
Malathion		0.00E+00	2.00E-02	(2)
Mercury	D	0.00E+00	3.00E-04	(3)
Nickel	D	0.00E+00	2.00E-02	(3)
Parathion		1.80E-03	3.30E-04	
Parathion-methyl	D	0.00E+00	2.50E-04	(3)
Phosdrin		0.00E+00	2.50E-04	(2)
Pirimiphos methyl		0.00E+00	1.00E-02	(2)
Prothiofos		0.00E+00	none yet	(2)
Toxaphene	B2	1.13E+00	2.50E-04	
Zinc		0.00E+00	4.70E-01	(2)

Notes:

(1) Carcinogenic by inhalation only.

(2) No human cancer potency measure has been developed yet.

(3) No evidence of carcinogenicity.

Table C.2. Daily Consumption Data for Thai People

Food Item	Consumption Rate (a) (g/pers/dy)	Consumption Rate (b) (g/kg/dy)	Category Assigned For Pesticides Data	Category Assigned For Metals Data
Rice, flour, potatoes, including products:				
Rice	252.74	4.68	Cereal	Flour
Others	45.88	0.85	Cereal	Flour
Meat and meat products:				
Meat	90.57	1.68	Meat	
Fish	33.31	0.62	Marine animal	Fish
Other marine animals	6	0.11	Marine animal	Fish
Eggs	20.02	0.37	Eggs	
Fresh Milk	8.44	0.16	Fresh milk	Milk
Powdered Milk	1.87	0.03		Milk
Condensed Milk	2	0.04		Milk
Bean and bean products:				
Dry beans	1.66	0.03	Dry legumes	
Products	7.23	0.13		
Vegetables:				
Green/yellow vegetables	45.36	0.84	Vegetables	Vegetables
Other vegetables	62.17	1.15	Vegetables	Vegetables
Fruits	107.98	2.00	Fruits	Fruits
Fats:				
Animal fats	8.42	0.16	Animal and	
Plant fats	6.47	0.12	veg oils	
Coconut	14.75	0.27	and fats	
Sugar	12.91	0.24		Sugar
Spices	25.2	0.47		
Instant foods	19.88	0.37		
Drinks	16.29	0.30		Fuit juice and drinks
Others	0.82	0.02		
Total	789.97	14.63		

Notes:

(a) Source: Nutrition Division, Department of Health, Ministry of Public Health (1976).

(b) Assumes average body weight is 54 kg (source: MOPH, Nutrition Division (1989)).

Table C.3. Concentration Data for Pesticides
Residue concentrations (mg/kg)(a)

Pesticide	Cereals	Meats	Marine Animals	Eggs	Fresh Milk	Dry legumes	Vegetables	Fruits	Animal/Veg Fats and Oils
Total Number of Samples	10	66	120	34	26	14	251	123	19
Aldrin									
min	ND	ND	ND	ND	ND	ND	ND	ND	ND
max	ND	ND	ND	ND	ND	ND	<0.01	ND	ND
avg	-	-	-	-	-	-	<0.01	-	-
percent	-	-	-	-	-	-	0.8%	-	-
BHC									
min	0	0	0	0	0	0	0	ND	0
max	0.02	0.08	<0.01	<0.01	<0.01	0.1	0.39	ND	0.03
avg	<0.01	<0.01	<0.01	-	<0.01	<0.01	<0.01	-	<0.01
percent	11.1%	33.3%	5.8%	-	7.7%	14.3%	4.4%	-	57.9%
DDT									
min	0	0	0	0	0	0	0	0	0
max	<0.01	1.15	0.06	0.49	0.06	0.04	3.3	0.14	0.17
avg	<0.01	0.055	<0.01	0.062	<0.01	<0.01	0.023	<0.01	0.073
percent	20.0%	86.4%	42.5%	100.0%	84.6%	35.7%	23.1%	4.9%	100.0%
Dieldrin									
min	ND	0	0	0	0	0	0	ND	0
max	ND	0.03	<0.01	<0.01	0.12	<0.01	1.81	ND	0.06
avg	-	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	-	<0.01
percent	-	39.9%	5.8%	67.6%	84.6%	7.1%	6.4%	-	36.8%
Endrin									
min	ND	0	ND	0	ND	0	0	ND	ND
max	ND	<0.01	ND	<0.01	ND	0.05	0.11	ND	ND
avg	-	<0.01	-	<0.01	-	<0.01	<0.01	-	-
percent	-	7.6%	-	3.8%	-	35.7%	4.4%	-	-
Heptachlor									
min	ND	0	ND	0	0	ND	0	0	ND
max	ND	<0.01	ND	<0.01	0.35	ND	2.08	0.04	ND
avg	-	<0.01	-	<0.01	0.024	-	<0.01	<0.01	-
percent	-	12.1%	-	41.2%	30.8%	-	2.8%	0.8%	-

Notes:

(a) Source: Report on Study and Surveillance on pesticide residue in food 1982-1985, Thailand Food and Drug Administration.

Table C.3. Concentration Data for Pesticides
Residue concentrations (mg/kg)(a)

Pesticide	Cereals	Meats	Marine Animals	Eggs	Fresh Milk	Dry legumes	Vegetables	Fruits	Animal/Veg Fats and Oils
Total Number of Samples	10	66	120	34	26	14	251	123	19
Lindane									
min	ND	0	ND	ND	0	ND	0	ND	ND
max	ND	0.03	ND	ND	<0.01	ND	<0.01	ND	ND
avg	-	<0.01	-	-	<0.01	-	<0.01	-	-
percent	-	3.0%	-	-	7.4%	-	1.2%	-	-
Malathion									
min	ND	ND	ND	ND	ND	ND	ND	0	ND
max	ND	ND	ND	ND	ND	ND	ND	0.1	ND
avg	-	-	-	-	-	-	-	<0.01	-
percent	-	-	-	-	-	-	-	1.6%	-
Parathion									
min	ND	ND	ND	ND	ND	ND	0	0	ND
max	ND	ND	ND	ND	ND	ND	0.29	0.29	ND
avg	-	-	-	-	-	-	<0.01	<0.01	-
percent	-	-	-	-	-	-	2.0%	8.9%	-
Captan									
min	ND	ND	ND	ND	ND	ND	ND	ND	ND
max	ND	ND	ND	ND	ND	ND	ND	ND	ND
avg	-	-	-	-	-	-	-	-	-
percent	-	-	-	-	-	-	-	-	-
Diazinon									
min	ND	ND	ND	ND	ND	ND	0	ND	ND
max	ND	ND	ND	ND	ND	ND	0.57	ND	ND
avg	-	-	-	-	-	-	<0.01	-	-
percent	-	-	-	-	-	-	1.6%	-	-
Chlorpyrifos									
min	ND	ND	ND	ND	ND	ND	0	0	ND
max	ND	ND	ND	ND	ND	ND	0.02	<0.01	ND
avg	-	-	-	-	-	-	<0.01	<0.01	-
percent	-	-	-	-	-	-	0.8%	0.8%	-

Notes:

(a) Source: Report on Study and Surveillance on pesticide residue in food 1982-1985, Thailand Food and Drug Administration.

(continued)

Table C.3. Concentration Data for Pesticides
Residue concentrations (mg/kg)(a)

Pesticide	Cereals	Meats	Marine Animals	Eggs	Fresh Milk	Dry legumes	Vegetables	Fruits	Animal/Veg Fats and Oils
Total Number of Samples	10	66	120	34	26	14	251	123	19
Chlordane									
min	ND	0	ND	ND	0	ND	0	0	0
max	ND	<0.01	ND	ND	<0.01	ND	<0.01	<0.01	<0.01
avg	-	<0.01	-	-	<0.01	-	<0.01	<0.01	<0.01
percent	-	1.5%	-	-	3.8%	-	0.4%	0.8%	5.3%
Azinphos-ethyl									
min	ND	ND	ND	ND	ND	ND	0	ND	ND
max	ND	ND	ND	ND	ND	ND	0.19	ND	ND
avg	-	-	-	-	-	-	<0.01	-	-
percent	-	-	-	-	-	-	0.8%	-	-
Endosulfan									
min	ND	ND	ND	ND	ND	ND	0	ND	ND
max	ND	ND	ND	ND	ND	ND	0.13	ND	ND
avg	-	-	-	-	-	-	<0.01	-	-
percent	-	-	-	-	-	-	0.8%	-	-
Endosulfan sulfate									
min	ND	ND	ND	ND	ND	ND	0	0	ND
max	ND	ND	ND	ND	ND	ND	0.18	0.3	ND
avg	-	-	-	-	-	-	<0.01	<0.01	-
percent	-	-	-	-	-	-	0.4%	0.8%	-
Parathion-methyl									
min	ND	ND	ND	ND	ND	0	0	0	ND
max	ND	ND	ND	ND	ND	0.03	0.07	0.46	ND
avg	-	-	-	-	-	<0.01	<0.01	<0.01	-
percent	-	-	-	-	-	7.1%	2.4%	8.9%	-
Pirimiphos methyl									
min	ND	ND	ND	ND	ND	ND	0	ND	ND
max	ND	ND	ND	ND	ND	ND	0.07	ND	ND
avg	-	-	-	-	-	-	<0.01	-	-
percent	-	-	-	-	-	-	1.2%	-	-

Notes:

(a) Source: Report on Study and Surveillance on pesticide residue in food 1982-1985, Thailand Food and Drug Administration.

(continued)

Table C.3. Concentration Data for Pesticides
Residue concentrations (mg/kg)(a)

Pesticide	Cereals	Meats	Marine Animals	Eggs	Fresh Milk	Dry legumes	Vegetables	Fruits	Animal/Veg Fats and Oils
Total Number of Samples	10	66	120	34	26	14	251	123	19
Dimethoate									
min	ND	ND	ND	ND	ND	ND	0	0	ND
max	ND	ND	ND	ND	ND	ND	0.36	3.84	ND
avg	-	-	-	-	-	-	<0.01	0.04	-
percent	-	-	-	-	-	-	4.0%	12.2%	-
Dicofol									
min	ND	ND	ND	ND	ND	ND	0	0	ND
max	ND	ND	ND	ND	ND	ND	7.61	1.9	ND
avg	-	-	-	-	-	-	0.03	0.072	-
percent	-	-	-	-	-	-	4.4%	13.8%	-
Fenitrothion									
min	ND	ND	ND	ND	ND	ND	0	ND	ND
max	ND	ND	ND	ND	ND	ND	<0.01	ND	ND
avg	-	-	-	-	-	-	<0.01	-	-
percent	-	-	-	-	-	-	0.4%	-	-
EPN									
min	ND	ND	ND	ND	ND	ND	0	ND	ND
max	ND	ND	ND	ND	ND	ND	0.16	ND	ND
avg	-	-	-	-	-	-	<0.01	-	-
percent	-	-	-	-	-	-	0.4%	-	-
Phosdrin									
min	ND	ND	ND	ND	ND	ND	0	ND	ND
max	ND	ND	ND	ND	ND	ND	1.29	ND	ND
avg	-	-	-	-	-	-	<0.01	-	-
percent	-	-	-	-	-	-	1.2%	-	-

Notes:

(a) Source: Report on Study and Surveillance on pesticide residue in food 1982-1985, Thailand Food and Drug Administration.

(continued)

Table C.3. Concentration Data for Pesticides
Residue concentrations (mg/kg)(a)

Pesticide	Cereals	Meats	Marine Animals	Eggs	Fresh Milk	Dry legumes	Vegetables	Fruits	Animal/Veg Fats and Oils
Total Number of Samples	10	66	120	34	26	14	251	123	19
Toxaphene									
min	ND	ND	ND	ND	ND	ND	0	ND	ND
max	ND	ND	ND	ND	ND	ND	0.47	ND	ND
avg	-	-	-	-	-	-	<0.01	-	-
percent	-	-	-	-	-	-	0.4%	-	-
Prothiofos									
min	ND	ND	ND	ND	ND	ND	0	ND	ND
max	ND	ND	ND	ND	ND	ND	0.57	ND	ND
avg	-	-	-	-	-	-	<0.01	-	-
percent	-	-	-	-	-	-	2.0%	-	-

Notes:

(a) Source: Report on Study and Surveillance on pesticide residue in food 1982-1985, Thailand Food and Drug Administration.

Bangkok may be higher or lower than these values. Thus, the analysis based on these data may be termed an investigation of health risks for the average urban Thai citizen rather than for the average resident of Bangkok.

Data on metals in food was obtained from a study of contaminants in food, 1984-1986, performed by the Food Control Division of the Thai FDA. The average contaminant values were provided for eight metals in seven commodity categories. Unlike the pesticides residue data, the metals data, shown in Table C.4, are from samples collected in Bangkok. The commodity categories for which data were reported were somewhat different for pesticide residues and metals.

III. Analytical Methodology Used to Develop Risk Estimates

Description of the Calculations

Calculation of risks from food contamination proceeded in four steps. In the first step, the quantity of contaminant ingested through consumption of each contaminated commodity was calculated as:

$$DOSE = C \times CONC \times P$$

where:

- DOSE = dose, (mg/kg/day);
- C = daily consumption of the commodity (kg food per kg body weight per day);
- CONC = concentration of the contaminant in the commodity (mg/kg) in those samples of the commodity in which the contaminant is present; and
- P = percent of the commodities in which commodity is present.

The third factor is important to note. For most of the commodity samples tested, no pesticides were found. This is expected: since not all crops are treated with pesticides, only a portion of the commodity supply is likely to be contaminated. This means that, for the average person, only a fraction of the food he or she ingests will be contaminated. The data obtained from Thai FDA (Table C.3) indicate the percent of samples in which the pesticides of interest was detected at all, and also the levels at which it was present in the cases when it was detected.

Table C.4. Concentration Data for Metals (a),(b)

Metal	Flour	Meat	Fish	Milk Products	Vegetable	Fruit	Sugar	Fruit juice and drink products
Mercury (mg/kg)	0.007	0.01	0.2	0.01	0.01	0.009	0.01	0.01
Lead (mg/kg)	0.33	0.31	0.55	0.14	0.45	0.24	0.26	0.05
Iron (mg/kg)	18.9	13.21	24.62	0.85	33.5	13.95	4.36	0.49
Copper (mg/kg)	1.63	0.98	0.61	0.13	0.94	1.62	0.21	0.14
Cadmium (mg/kg	0.11	0.16	0.05	0.03	0.07	0.73	0.08	0.02
Zinc (mg/kg)	7.4	13.68	15.53	3.75	4.87	1.43	0.24	0.26
Tin (mg/kg)	13.94	28.52	32.14	5.19	11.67	11.74	18.13	1.56
Arsenic (mg/kg)	0.1	0.04	0.23	0.07	0.13	0.14	0.53	0.02

Notes:
(a) Source: Study of Contaminants in Foods. (1984-1986), FDA, Food Control Division. Samples from Bangkok onl
(b) No. of samples not given.

In the second step, the exposure from all commodities was summed to obtain the total exposure to each contaminant, in mg contaminant per kg body weight per day. Next, if the contaminant was a carcinogen, the third step involved multiplying the daily exposure by a cancer potency estimate, whose units are risk per mg/kg/day exposure. The result was an individual cancer risk over the lifetime of the individual. To obtain the annual cancer cases estimated to occur as a result of pesticide exposure, the individual risk was multiplied by the size of the exposed population and divided by the length of the average lifespan, assumed to be 70 years.

Generally, dose-response data, which allow the estimation of the magnitude of a health effect response to exposure, are not available for noncarcinogens. Therefore, if the contaminant was not a carcinogen, the daily exposure was simply compared to a Reference Dose (RfD), the daily exposure below which no adverse effects are expected to occur. In this analysis, exposure is presented as a percentage of the Reference Dose. Exposures which exceed the Reference Dose pose a risk that adverse health effects will occur, although the extent of the risk posed and the severity of the response are not estimated. Exposures that approach the reference dose may also be of concern. Food is only one source of exposure to environmental contaminants; exposure may also occur through water, air and soil, and/or occupationally. If the dose from food alone is close to the RfD, chances are increased that the RfD will be exceeded when food exposures are combined with exposures from other sources.

Description of Use of Data Sources

The contaminant exposure calculations required data on daily consumption of commodities and on contaminant concentrations in the commodities. Consumption data provided by the Nutrition Division were presented in units of grams per person per day. To convert this value into kilograms of food per kilogram body weight per day, the values were divided by a factor of 1000. Values were then divided by average body weight of a Thai adult. The average body weight for adult men is 58 kilograms, while average body weight for adult women is 50 kilograms (Nutrition Division, DOH, MOPH, 1989). Assuming that the population is evenly divided between the sexes, the average adult body weight is assumed to be 54 kilograms.

The data provided by the Thai FDA for both pesticide residues and for metal contamination were already provided in unit of milligrams of contaminant per kilograms of food, so no unit conversion was required. However, to perform the calculations, we had to match the twenty-one commodity categories presented in the consumption data with the nine commodity categories for which pesticide residue data

are presented, and with seven commodity categories for which metals data are presented. Table C.2. shows how the consumption categories were fitted to the pesticide and metals data commodities categories. Note that data were not available for all commodity categories; therefore, some possible sources of dietary exposure to pesticides and metals were not included in this analysis.

In the U.S., dietary consumption data are generally expressed in terms of wet weight of food consumed per day, while contaminant concentration data generally reported in mass of contaminant per mass dry weight of the commodity. When the data are used together, the consumption values must be converted to dry weight equivalent values. We assumed these U.S. data reporting conventions also exist in the reporting of Thai data, and converted the Thai dietary consumption data to dry weight values, using typical wet:dry weight ratios derived from a U.S. EPA dietary database (the Tolerance Assessment System, or TAS).

To calculate annual cancer cases over the entire population, the average residue concentrations (rather than maximum or minimum) were used. For many of the pesticides, average residue values were presented as "< 0.01 mg/kg." To use these data, we conservatively assumed that the value 0.01 represents an upper bound on the average concentration of the pesticide residue. The actual average value may be much lower than this, and may approach zero. Therefore, the estimated risks from pesticides for which data are reported as "< 0.01 mg/kg" probably overestimate true risk.

IV. Discussion of Findings

Estimated risks from the ingestion of pesticide-contaminated food are found in Table C.5. The findings show that consumption of pesticide residues in food result in an estimated 14 cases of cancer per year. About 70 percent of this risk is attributable to dieldrin residues, primarily from residues in meat, milk, and fats/oils. This result is not surprising, given the lipophilic nature of this chemical. It should be noted that the average concentrations for dieldrin were reported as <0.01 mg/kg for all commodities. As discussed above, for the purposes of this analysis, we conservatively assumed that the value 0.01 mg/kg represented the average concentration of dieldrin in the commodities; if the actual average concentration is much lower than this, the risks from dieldrin may be substantially less than those estimated here. Other pesticides which contribute to the risk include BHC (about 1 case per year) and DDT (about 2 cases per year). The remaining pesticides result in less than one case of cancer per year.

Table C.5. Risks from Pesticides in Food

Pesticide	Exposure (mg/kg/day)									Total Exposure From All Foods (mg/kg/day)	Population Risk From Pesticide Exposure (cases/yr)
	Cereal	Meat	Marine Animal	Eggs	Fresh Milk	Dry Legumes	Vegetables	Fruit	Animal And Vegetable Oils		
Aldrin											
min	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0E+00	
max	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.11E-06	0.00E+00	0.00E+00	1.1E-06	
avg	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.85E-09	0.00E+00	0.00E+00	8.9E-09	1.3E-02
BHC											
min	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0E+00	
max	9.70E-05	4.47E-05	2.43E-06	9.27E-07	1.56E-06	8.78E-07	4.31E-05	0.00E+00	1.37E-05	2.0E-04	
avg	5.38E-06	1.86E-06	1.41E-07	0.00E+00	1.20E-07	1.26E-08	4.87E-08	0.00E+00	2.65E-06	1.0E-05	1.1E+00
DDT											
min	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0E+00	
max	4.85E-05	6.43E-04	1.46E-05	4.54E-05	9.38E-06	3.51E-07	3.65E-04	1.56E-05	7.78E-05	1.2E-03	
avg	9.70E-06	2.66E-05	1.03E-06	5.75E-06	1.32E-06	3.14E-08	5.88E-07	5.44E-08	3.34E-05	7.8E-05	2.2E+00
Dieldrin											
min	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0E+00	
max	0.00E+00	1.68E-05	2.43E-06	9.27E-07	1.88E-05	8.78E-08	2.00E-04	0.00E+00	2.74E-05	2.7E-04	
avg	0.00E+00	2.23E-06	1.41E-07	6.27E-07	1.32E-06	6.24E-09	7.08E-08	0.00E+00	1.68E-06	6.1E-06	1.0E+01

Note:

(a) Calculated from residue concentration and dietary consumption data. See text for calculation.

Table C.5. Risks from Pesticides in Food

Pesticide	Exposure (mg/kg/day) Cereal	Meat	Marine Animal	Eggs	Fresh Milk	Dry Legumes	Vegetables	Fruit	Animal And Vegetable Oils	Total Exposure From All Foods (mg/kg/day)	Population Risk From Pesticide Exposure (caser'r)
Chlordane											
min	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0E+00	
max	0.00E+00	5.59E-06	0.00E+00	0.00E+00	1.56E-06	0.00E+00	1.11E-06	1.11E-06	0.00E+00	9.4E-06	
avg	0.00E+00	8.39E-08	0.00E+00	0.00E+00	5.94E-08	0.00E+00	4.43E-09	8.89E-09	0.00E+00	1.6E-07	1.7E-02
Azinphos-ethyl											
min	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0E+00	
max	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.10E-05	0.00E+00	0.00E+00	2.1E-05	
avg	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.85E-09	0.00E+00	0.00E+00	8.9E-09	1.1E-10
Endosulfan											
min	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0E+00	
max	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.44E-05	0.00E+00	0.00E+00	1.4E-05	
avg	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.85E-09	0.00E+00	0.00E+00	8.9E-09	0.0E+00
Endosulfan sulfate											
min	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0E+00	
max	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.99E-05	3.33E-05	0.00E+00	5.3E-05	
avg	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.43E-09	8.89E-09	0.00E+00	1.3E-08	0.0E+00

Note:

(a) Calculated from residue concentration and dietary consumption data. See text for calculation.

(continued)

Table C.5. Risks from Pesticides in Food

Pesticide	Exposure (mg/kg/day) Cereal	Meat	Marine Animal	Eggs	Fresh Milk	Dry Legumes	Vegetables	Fruit	Animal And Vegetable Oils	Total Exposure From All Foods (mg/kg/day)	Population Risk From Pesticide Exposure (cases/yr)
Endrin											
min	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0E+00	
max	0.00E+00	5.59E-06	0.00E+00	9.27E-07	0.00E+00	4.39E-07	1.22E-05	0.00E+00	0.00E+00	1.9E-05	
avg	0.00E+00	4.25E-07	0.00E+00	3.52E-08	0.00E+00	3.14E-08	4.87E-08	0.00E+00	0.00E+00	5.4E-07	0.0E+00
Heptachlor											
min	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0E+00	
max	0.00E+00	5.59E-06	0.00E+00	9.27E-07	5.47E-05	0.00E+00	2.30E-04	4.44E-06	0.00E+00	3.0E-04	
avg	0.00E+00	6.76E-07	0.00E+00	3.82E-07	1.16E-06	0.00E+00	3.10E-08	8.89E-09	0.00E+00	2.3E-06	8.5E-01
Lindane											
min	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0E+00	
max	0.00E+00	1.68E-05	0.00E+00	0.00E+00	1.56E-06	0.00E+00	1.11E-06	0.00E+00	0.00E+00	1.9E-05	
avg	0.00E+00	1.68E-07	0.00E+00	0.00E+00	1.16E-07	0.00E+00	1.33E-08	0.00E+00	0.00E+00	3.0E-07	3.2E-02
Malathion											
min	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0E+00	
max	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.11E-05	0.00E+00	1.1E-05	
avg	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.78E-08	0.00E+00	1.8E-08	0.0E+00

Note:

(a) Calculated from residue concentration and dietary consumption data. See text for calculation.

(continued)

Table C.5. Risks from Pesticides in Food

Pesticide	Exposure (mg/kg/day) Cereal	Meat	Marine Animal	Eggs	Fresh Milk	Dry Legumes	Vegetables	Fruit	Animal And Vegetable Oils	Total Exposure From All Foods (mg/kg/day)	Population Risk From Pesticide Exposure (cases/yr)
Parathion											
min	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0E+00	
max	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.21E-05	3.22E-05	0.00E+00	6.4E-05	
avg	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.21E-08	9.89E-08	0.00E+00	1.2E-07	1.8E-05
Captan											
min	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0E+00	
max	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0E+00	
avg	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0E+00	0.0E+00
Diazinon											
min	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0E+00	
max	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.31E-05	0.00E+00	0.00E+00	6.3E-05	
avg	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.77E-08	0.00E+00	0.00E+00	1.8E-08	0.0E+00
Chlorpyrifos											
min	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0E+00	
max	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.21E-06	1.11E-06	0.00E+00	3.3E-06	
avg	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.85E-09	8.89E-09	0.00E+00	1.8E-08	0.0E+00

Note:

(a) Calculated from residue concentration and dietary consumption data. See text for calculation.

(continued)

Table C.5. Risks from Pesticides in Food

Pesticide	Exposure (mg/kg/day) Cereal	Meat	Marine Animal	Eggs	Fresh Milk	Dry Legumes	Vegetables	Fruit	Animal And Vegetable Oils	Total Exposure From All Foods (mg/kg/day)	Population Risk From Pesticide Exposure (cases/yr)
Parathion-methyl											
min	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0E+00	
max	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.63E-07	7.74E-06	5.11E-05	0.00E+00	5.9E-05	
avg	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.24E-09	2.66E-08	9.89E-08	0.00E+00	1.3E-07	0.0E+00
Pirimiphos methyl											
min	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0E+00	
max	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	7.74E-06	0.00E+00	0.00E+00	7.7E-06	
avg	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.33E-08	0.00E+00	0.00E+00	1.3E-08	0.0E+00
Dimethoate											
min	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0E+00	
max	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	3.98E-05	4.27E-04	0.00E+00	4.7E-04	
avg	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.43E-08	5.42E-07	0.00E+00	5.9E-07	0.0E+00
Dicofol											
min	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0E+00	
max	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	8.42E-04	2.11E-04	0.00E+00	1.1E-03	
avg	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.46E-07	1.10E-06	0.00E+00	1.2E-06	0.0E+00

Note:

(a) Calculated from residue concentration and dietary consumption data. See text for calculation.

(continued)

Table C.5. Risks from Pesticides in Food

Pesticide	Exposure (mg/kg/day) Cereal	Meat	Marine Animal	Eggs	Fresh Milk	Dry Legumes	Vegetables	Fruit	Animal And Vegetable Oils	Total Exposure From All Foods (mg/kg/day)	Population Risk From Pesticide Exposure (cases/yr)
Fenitrothion											
min	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0E+00	
max	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.11E-06	0.00E+00	0.00E+00	1.1E-06	
avg	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.43E-09	0.00E+00	0.00E+00	4.4E-09	0.0E+00
EPN											
min	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0E+00	
max	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.77E-05	0.00E+00	0.00E+00	1.8E-05	
avg	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.43E-09	0.00E+00	0.00E+00	4.4E-09	0.0E+00
Phosdrin											
min	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0E+00	
max	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.43E-04	0.00E+00	0.00E+00	1.4E-04	
avg	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	1.33E-08	0.00E+00	0.00E+00	1.3E-08	0.0E+00

Note:

(a) Calculated from residue concentration and dietary consumption data. See text for calculation.

(continued)

Table C.5. Risks from Pesticides in Food

Pesticide	Exposure (mg/kg/day) Cereal	Meat	Marine Animal	Eggs	Fresh Milk	Dry Legumes	Vegetables	Fruit	Animal And Vegetable Oils	Total Exposure From All Foods (mg/kg/day)	Population Risk From Pesticide Exposure (cases/yr)
Toxaphene											
min	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0E+00	
max	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	5.20E-05	0.00E+00	0.00E+00	5.2E-05	
avg	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	4.43E-09	0.00E+00	0.00E+00	4.4E-09	4.2E-04
Prothiofos											
min	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.0E+00	
max	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	6.31E-05	0.00E+00	0.00E+00	6.3E-05	
avg	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	0.00E+00	2.21E-08	0.00E+00	0.00E+00	2.2E-08	0.0E+00
Total											1.4E+01

Note:

(a) Calculated from residue concentration and dietary consumption data. See text for calculation.

Risks from ingestion of metals in food are shown in Table C.6. This table shows that arsenic exposure from food consumption may exceed the reference dose by 20 percent. Most of the exposure is attributable to arsenic concentrations in the flour and in meat. Table C.6. also shows that the exposure to lead from food consumption is almost equal to the Reference Dose. The health effects associated with exposure to lead are discussed in detail in Appendix E. For all of metals, concentrations detected in flour were used to represent concentrations in the entire "flour" commodity group, which includes rice (see Table C.2). The accuracy of the risk estimates for metals depends to a great degree on how well the concentration of metals in flour represent the concentrations of metals present in rice.

V. Limitations

There are many important limitations to the methods presented in this Appendix. The most important uncertainty is the accuracy of the pesticide residue, metals concentration and dietary consumption data used in the calculations. For example, as discussed earlier, residue averages reported as <0.01 mg/kg were assumed to be equal to 0.01 mg/kg, but may actually be much smaller. Often, when the US EPA is faced with similar data limitations, it is assumed that the distribution of pesticide residues is uniform between zero and the reported upper-bound. The mean of the distribution, or 0.5 of the reported upper bound, is then used to represent the residue concentration. In this analysis, if we were to assume all the observations reported as "<0.01 mg/kg" were actually 0.005 mg/kg, population cancer risks would be estimated to be about 8 cases per year instead of 14.

The risk calculations also assumed that the concentration levels reported in the Thai FDA data were the concentrations in foods as eaten. This assumption may be inaccurate for a number of reasons. If the residues presented in the Thai FDA data were measured "at the farmgate," rather than at the market, they are probably an overestimate of actual residues, since residue loss may occur during shipping and handling. Furthermore, consumer preparation of foods (cooking, washing, peeling) may lead to reductions in residues as well. On the other hand, pesticides can concentrate in certain commodities during processing. There were no data available, however, to estimate the magnitude of these effects on the concentrations of pesticides in the Thai diet.

The pesticide calculations were based on average residue values for samples analyzed between 1982 and 1985. However, the use and production of many of these pesticides have been banned in

Table C.6. Exposure From Metal in Food

Metal	Exposure (mg/kg/day) Flour	Meat	Fish	Milk and Milk Products	Vegetable	Fruit	Sugar	Fruit juice and drink products	Total Exposure (mg/kg/day)	Comparison To RfD (% of RfD)
Mercury	3.4E-05	5.6E-06	4.9E-05	2.3E-06	1.1E-06	1.0E-06	8.0E-07	3.0E-06	9.6E-05	32.0%
Lead	1.6E-03	6.4E-05	1.3E-04	3.2E-05	5.0E-05	2.7E-05	2.1E-05	1.5E-05	1.9E-03	97.0%
Copper	7.9E-03	4.9E-04	1.5E-04	3.0E-05	1.0E-04	1.8E-04	1.7E-05	4.2E-05	8.9E-03	22.0%
Cadmium	5.3E-04	9.1E-05	1.2E-05	6.8E-06	7.7E-06	8.1E-05	6.4E-06	6.0E-06	7.4E-04	37.0%
Zinc	3.6E-02	2.5E-05	3.8E-03	8.5E-04	5.4E-04	1.6E-04	1.9E-05	7.8E-05	4.1E-02	8.8%
Arsenic	4.9E-04	1.1E-03	5.6E-05	1.6E-05	1.4E-05	1.6E-05	4.2E-05	6.0E-06	1.7E-03	120.0%

Thailand during the 1980's. Benzene hexachloride (BHC) was banned in Thailand in 1980, DDT in 1983, and dieldrin in 1988 (ONEB, 1989b). Therefore, the current residues of these compounds may be lower. However, given the persistence of the chemicals in the environment, and given the fact that farmers may continue to use existing stocks of these pesticides for several years after a ban is instituted, the residues may not disappear altogether for many years. For example, in the U.S., many organochlorine pesticides were banned or restricted in the 1970's, yet ten years later, residues of these pesticides were still found in fish, birds, and human tissue (although at levels about half the levels found in the 1970's (Council on Environmental Quality, 1984)).

Matching of commodity categories for the consumption data and the residue and metals data is another source of uncertainty. The possible effect on the results of our analysis is unknown, since it is unknown how well the inaccurate matching could have under- or over-estimated contamination for any one category of food. Furthermore, data were not available for all categories of foods, so these calculations do not account for all possible sources of exposure in the diet.

Finally, the dietary consumption data may also be a source of uncertainty. Consumption data used in this analysis represent 1976 dietary patterns. Consumption rates may have increased since 1976 due to a rise in per capita income. Given the same residue and metals data, higher consumption rates would lead to an increase in exposure to pesticides and metals residues. However, since we do not know which foods would be consumed in greater amounts, it is impossible to estimate the magnitude of the possible increase in risks.

Appendix D. Health Risks from Disposal of Solid and Hazardous Wastes

I. Definition of the Problem

Solid Wastes

Collection of solid wastes

Collection of solid wastes in Bangkok is generally effective; the Bangkok Metropolitan Administration (BMA) reportedly collects from an impressive 85 percent of the population on a daily basis (Starobin and Kornberg, 1989). It has been estimated that 4,700 metric tons of waste are collected daily from Bangkok, with the total climbing every year. Collection is performed by approximately 3,000 laborers (Hubbard, 1987), who are reported to scavenge the wastes for recyclable materials before delivering to municipal facilities. A thriving market is available for the recyclable materials. Because the scavenging leads to increased human contact with the garbage, however, it probably results in increased health risks for those involved (Starobin and Kornberg, 1989). Also included in the municipal waste stream is nightsoil from septic tanks. Nightsoil is collected for a fee from individual households, and it is estimated that illegal disposal of these wastes is very common (Hubbard, 1987).

Uncollected solid waste

The fraction of Bangkok's solid waste that is not collected may cause health risks primarily by providing food and breeding grounds for rodents and insects that act as vectors of microbiological disease. Improperly-disposed nightsoil may be a particularly important problem, as most of the important microbiological diseases are transmitted through human feces. Health risks from these sources are discussed in Appendix F.

Disposal of solid wastes

Until recently, collected wastes have been received by three dumping areas, each of which also had composting facilities and small incinerators to burn the compost reject. Although the compost plants had a rated capacity of about 28 percent of the current waste stream, operating problems have prevented their processing much of the solid waste. As a result, more than 90 percent of Bangkok's collected solid wastes has been disposed of by open dumping. Sanitary conditions at these facilities have been deplorable. According to Starobin and Kornberg,

> Bangkok's three landfills can be described as open dumps. They have no liners; surface water collection systems are rudimentary if they exist at all; leachate collection systems and methane control systems have not been installed; the waste is not covered with soil; and the concept of dividing the landfill into cells is applied rather broadly... Fires burn

constantly; rodents and insects live and breed on site; the odors are noisome; drainage water is contaminated; and leachate from the site percolates into nearby water sources.

It is estimated that existing landfill capacity will be soon be exhausted, so that alternative sites and facilities are urgently needed. Two of the existing facilities (Soi Onnum and Nong Khaem) can no longer receive wastes from the city. Plans are in process for a four-year project to barge some of the wastes to a new site in Samut Prakarn (The Nation, 1987a,b).

Hazardous Wastes

According to a report prepared for the National Environment Board (NEB), over the past seven or eight years, a 10 percent economic growth rate in Thailand has led to the doubling of national industrial capacity and a corresponding doubling of the generation of hazardous wastes. The rate of production of non-recyclable hazardous wastes is expected to grow to almost two million tons per year by the early 1990's. Most of this growth is in industrial production capacity, and most of the growth in hazardous waste production is taking place in Bangkok. A major goal of the Thai Government's Sixth Plan for 1986-1991 was to control development in the Bangkok Metropolitan Area and to promote growth in other parts of the country. According to ONEB (1989a), however:

> "given the apparent magnitude and direction of current growth trends, it would appear that simply maintaining the present distribution of industrial activities in the country would constitute a major success for Government efforts to control growth...it has therefore been assumed that future distributions of hazardous waste generating activities will be similar to the present [1986] distribution."

In addition to the generation of hazardous wastes, industrial development has also led to increased pollution of air and water, as discussed in Appendices A and B. Production and storage of large volumes of hazardous materials also presents risks of catastrophic accidents, leading to fires, explosions, or the release of large quantities of toxins into the environment (The Nation, 1989g).

There are a number of examples of damages from improper management of hazardous wastes in Thailand. In one village, discarded casings from lead batteries were burned as fuel for coconut sugar production. Lead released through fumes and ashes contaminated nearby air and soil, leading to the death by lead-poisoning for one girl in the village. Investigations by the Institute of Environmental Research discovered that an asphalt-type material made from old battery casings had been used to pave

a road through the village. Children played on the road, and families raised vegetables in soil with extraordinarily high concentrations of lead. Drinking water was likewise contaminated, and emissions from a nearby smelter raised air concentrations as high as 130 ug/m^3. Later, in 1985, cadmium, lead, and manganese were reported at "alarming concentrations" in the upper reaches of the Chao Phraya River. An accidental spill from a zinc smelter was found to be the source of the heavy metal contamination (ONEB, 1989a).

Improper disposal of infectious wastes can also pose significant risks. Although some hospitals do make some effort to separate infectious wastes, much of this waste is believed to be co-disposed with ordinary solid wastes. These wastes pose a particular hazard to scavengers, since many of the infectious waste materials (e.g. syringes) are easily resold (ONEB, 1989a).

As pressures for responsible management of hazardous wastes have increased, so has the need for facilities in which safe treatment and disposal can be accomplished. One existing facility (the Bangkhuntien Center) now receives hazardous wastes from Bangkok, and two more facilities are expected to begin operation in the near future. The existing facility manages only a small fraction of the hazardous wastes generated in Bangkok; most of the waste is currently sent to poorly controlled municipal dumps for co-disposal with municipal wastes, or else disposed of illegally.

Since solid and hazardous wastes are often disposed together, risks from disposal of these wastes are discussed together in this Appendix. Solid and hazardous waste disposal facilities present potential health risks through the following primary exposure pathways:

- Small populations of humans live on top of or near the landfills, and earn a living by scavenging through the raw, uncovered garbage in search of recyclable materials. These individuals are potentially exposed to pathogens and toxins through inhalation and physical contact with the wastes.
- Organic compounds may volatilize from the uncovered landfills or be emitted by open fires; as emissions drift over nearby resident populations, individuals may be exposed to health risks from inhalation of toxic contaminants,
- Contaminants from the landfills can leach into aquifers near the sites, or runoff into nearby surface water bodies, resulting in ecological risks or health risks to humans who use the water for bathing or drinking.

The risks to scavenging populations from these facilities are of particular interest. As stated by Kungskulniti et al. (1989):

Although the risks of garbage scavenging are as old as human habitation, the co-mingling of hazardous chemical and medical wastes creates an entirely new category of exposures and risks, ones that would not exist in either strictly modern or traditional settings. These conditions illustrate one of the interactions that can develop because of the "risk overlap" between traditional and modern risks in rapidly growing developing countries.

II. Data Acquired

A rigorous evaluation of health risks from collection of solid wastes and the disposal of solid and hazardous wastes in Bangkok would require the following data:

- estimates of the quantities and types of wastes collected and disposed,
- engineering data on practices, controls, and site characteristics for the waste disposal facilities,
- geohydrological and meteorological data for sites receiving the wastes,
- patterns of use for groundwater and surface water near the sites,
- data concerning the composition of wastes or the concentrations of contaminants in leachate or emissions from the facilities, and
- sizes of exposed populations, including residents near landfills and those involved in scavenging activities.

With such data, we could model expected pollutant releases to each environmental medium from each solid and hazardous waste disposal site and project the resulting human exposure and health risk. However, as discussed below, we were able to obtain only limited data for each of these categories.

Type and quantity of wastes

Estimates of the quantity of solid waste disposed were obtained from Starobin and Kornberg (1989). The current quantity is assumed to be about 4,700 metric tons per day. Data could not be obtained for the composition of municipal wastes from Bangkok. ONEB (1989a) provides detailed estimates of the quantities of hazardous wastes generated in Bangkok. It also includes a risk assessment in which "risk factors" are applied to each type of hazardous waste to derive a relative ranking of their hazard to human health in Thailand. The study provides informative discussions of the locations of urban and industrial development, of the locations of existing and proposed waste treatment and disposal sites, of local hydrogeology, and of the various individual industries generating hazardous wastes in Thailand.

As shown in Table D.1, heavy metal sludges and solids dominate the current and anticipated future hazardous waste stream, accounting for more than 70% of hazardous waste generation. This category is followed by oils (10%), acid wastes (7%), and infectious wastes (4%). The remaining 8% of the wastes is divided among the ten other categories listed in the table. Table D.2 lists the principal sources of waste generation in the Bangkok region. As can be seen from the table, hazardous waste generation is dominated by the metal smelting industry (64%), followed by lesser contributions from manufacturing (23%) and hospitals and laboratories, commercial/service, and marine/harbor, each of which accounts for about 4% of total wastes.

According to ONEB (1989a), the highest risks to public health are probably associated with the disposal of heavy metal sludges and solids from smelting activity in the Bangkok area. Based on the "Relative Hazard Risk" for each chemical identified, sizes of exposed populations, quantities of wastes involved, specific characteristics of the wastes and the level of treatment and disposal technology utilized, the authors derived "environmental risk factors" for disposal of each waste type in Thailand. Values ranged from nearly 20,000,000 for heavy metal sludges and solids to 400,000, 400,000, and 100,000 for infectious wastes, acid wastes, and alkaline wastes, respectively. Other categories of hazardous wastes were assigned relatively low environmental risk factors

Site characteristics

We were unable to obtain detailed data concerning municipal waste disposal site characteristics, except to determine the approximate location of the three existing landfills. As mentioned above, we know that controls for environmental protection are minimal at these facilities, which are without cover, liners, or leachate collection systems. One geological advantage of the Bangkok area (at least with respect to protection of groundwater quality) is that thick, multiple layers of clay tend to provide protection against contamination for fresh water aquifers. A geological profile of the Lower Chao Phraya Basin is illustrated in Figure D.1. Since heavy metals and some organic contaminants tend to be relatively immobile in clay, risks from groundwater contamination are reduced. As reported in ONEB (1989a), however, poor construction and uncontrolled abandonment

Table D.1

Projected Hazardous Waste Quantities by Waste Type

Waste Type	Hazardous Waste Quantities, Thousand Tonnes/Year			
	1986	1991	1996	2001
Oils	120	220	390	690
Liquid Organic Residues	0.19	0.31	0.52	0.88
Organic Sludges and Solids	3.70	6.70	12	21
Inorganic Sludges and Solids	12	19	32	54
Heavy Metal Sludges and Solids	830	1,500	2,500	4,400
Solvents	20	36	67	120
Acid Wastes	81	120	200	310
Alkaline Wastes	22	34	54	86
Off Spec Products	0.01	0.03	0.05	0.11
PCB	2.50	*	*	*
Aqueous Organic Residues	0.12	0.24	0.50	1
Photo Wastes	8.80	16	30	58
Municipal Wastes	7.20	12	19	31
Infectious Wastes	47	76	120	200
Total	1,300	1,900	3,500	5,900

Source: NEB (1989).
* Figures have been rounded independently.
* Total existing quantity estimated at 2,500 tonnes. It has been assumed that no new PCB-containing materials were imported to Thailand after the mid-1970's.

Table D.2

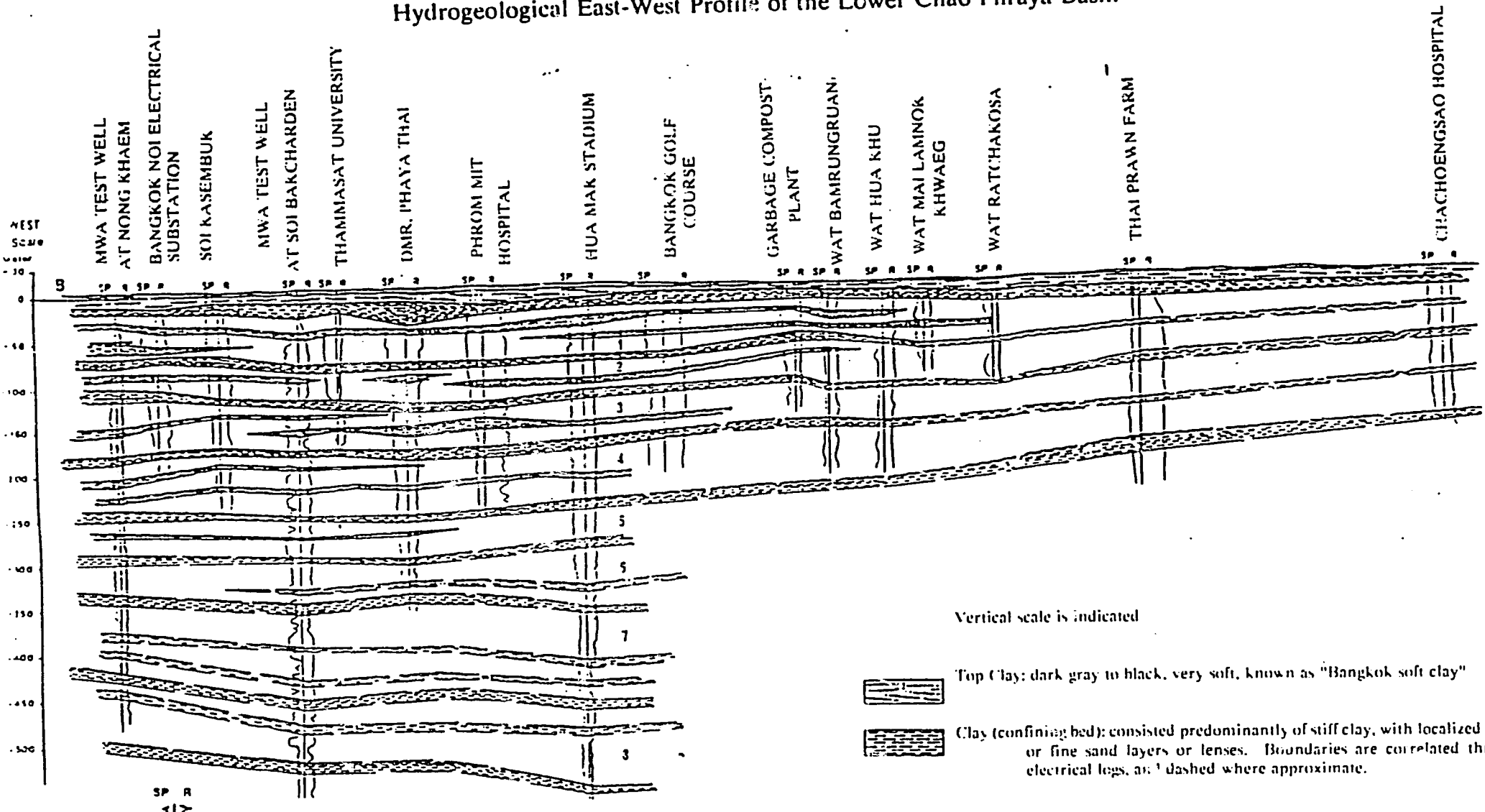
Summary of Hazardous Waste Quantities by Generator Group

Generator Group	Hazardous Waste Quantities (tonnes/yr), 1989 Bangkok	Thailand
Agricultural	2,700	3,700
Commercial / Service	31,000	43,000
Municipal Solid Wastes	5,100	7,200
Hospitals and Laboratories	33,000	47,000
Manufacturing	190,000	270,000
Marine / Harbor	30,000	43,000
Coal and Lignite Based	570	800
Petroleum Based	1,400	2,000
Metal Smelting	520,000	740,000
Total	820,000	1,100,000

* Figures have been rounded independently.
Source: NEB (1989).

Figure D.1

Hydrogeological East-West Profile of the Lower Chao Phraya Basin



SOURCE: DEPARTMENT OF MINERAL RESOURCES,
GROUNDWATER IN BANGKOK, 1985

of wells has led to the hydraulic interconnections between surface water sources and the highly productive deep aquifers, reducing the protection afforded by the clay layers.

Contaminant concentrations

Limited data were available on the concentrations of a few contaminants in leachate from the facilities, and on the concentrations of some contaminants in the air at these facilities. Table D.3 presents data on contaminant concentrations in leachate from existing municipal facilities. Table D.4 presents data on the concentrations of selected contaminants found in air on and around the On-Nuj dumpsite (Kungskulniti, et al., 1989). These data can be used for a rough evaluation of health risks presented by these facilities. These calculations will be described in Section III.

III. Analytical Steps to Arrive at Risk Estimates

For individuals living on or near a dumpsite, the constant exposure to pathogens, insects, rodents, and particulate emissions from burning wastes are likely to result in significant individual risks from communicable diseases and chronic respiratory disease. In fact, the On-Nuj scavenger study (Kungskulniti et al., 1989) reported substantial rates of respiratory, diarrheal and skin disease as well as diminished respiratory function among the scavengers; the relatively high concentrations of certain air contaminants and microbiological drinking water contaminants reported for the site suggest these health problems may be associated with environmental conditions. For example, total suspended particulate concentrations in the community were measured at 490 ug/m^3; these concentrations are five times higher than the Thai standard for average annual concentrations of TSP. In addition, stored water had fecal coliform concentrations ranging from 17 to 53 fecal coliforms per 100 ml, compared to a Thai drinking water standard of less than 2 coliforms per 100 ml. Water in ponds and shallow wells had fecal coliforms as high as 450,000 coliforms per 100 ml.

154

In addition to risks of communicable and respiratory disease and diminished respiratory function, there may also be long-term health risks, such as cancer, resulting from the ingestion of groundwater contaminated by migration of pollutants from the landfill, or from the inhalation of contaminants that volatilize from the landfill. Rough assessments of upper-bound risks from these pathways are discussed below.

Risks from contamination of groundwater

Table D.3 provides data about toxic constituents in leachate from municipal facilities. Of the two sites for which data are reported, poly-chlorinated biphenyls (PCBs), chromium, and mercury concentrations were available only for the leachate of On-Nuj. Making some assumptions, we can obtain some insight into the potential risks resulting from leachate with these concentrations of contaminants. As a conservative estimate of risk (that is, an estimate likely to overestimate risk), we assumed that the leachate is mixed with the total volume of water withdrawn from wells in the Bangkok area. This assumption is conservative, since actual dilution will probably be far greater, since not all of the water in the receiving aquifers will be withdrawn for consumption. As discussed in Appendix B, approximately 3000 wells in the Bangkok area withdraw approximately 257,000 m^3 of water daily. If we assume that each of three municipal landfills serving Bangkok are 1000 rai in area (1.6 km^2), and that average precipitation in Thailand is 8-280 mm/month or 0.27-9.3 mm per day, then it follows that the maximum quantity of leachate expected from the landfills is 430-15,000 m^3/day. If this quantity is diluted by the 257,000 m^3 withdrawn from wells in Bangkok, then concentrations in the leachate will be reduced by a factor of 20 to a factor of 600 before ingestion by humans. For simplicity, we will assume a dilution factor of 300. If an average individual of 54 kg ingests an average of 2 liters per day of leachate diluted by a factor of 300, he or she will be exposed to approximately 7×10^{-5} milligrams of PCBs per day per kilogram of body weight (mg/kg-day), 1×10^{-4} mg/kg-day of chromium, and 5×10^{-4} mg/kg-day of mercury. The U.S. EPA has established a risk reference dose (RfD) for chromium of 5×10^{-3} mg/kg-day, and for mercury of 4×10^{-4} mg/kg-day. Thus, even under these very conservative assumptions, the risk reference dose for chromium is not exceeded; however, the RfD for mercury is exceeded by 25 percent. For PCBs, the U.S. EPA Cancer Assessment Group recommends a cancer potency estimate of 7.7 $(mg/kg\text{-}day)^{-1}$. Combining this potency estimate with the estimated exposure yields an individual risk of 5×10^{-4}. This level of exposure would lead to an annual cancer case per year if the size of the population exposed to this level of PCBs in groundwater exceeded 140,000 persons. A population of this size would live within about a three-kilometer radius of each dumpsite, given an average population density in Bangkok of 3500 persons/km^2.

Table D.3

Characteristics of Solid Waste Leachate
(Stored in Dumpsite Ponds)[1]

Description	Nong Khaem	On-Nuj[2]	On-Nuj[3]
pH	8.2–9.0	7.6–9.1	7.9–8.6
Alkalinity[a] as $CaCo_3$	6,100	3,200	1,000–8,900
BOD[b]	690	310	150–1,230
COD[c]	14,000	3,300	1,900–8,800
TKN[d]	570	220	580–1,700
Phosphate	22	7.8	23
Suspended solids	150	110	110–2,400
Colour (Pt–Co)	–	0	3,500–23,000
Chromium VI	–	–	1
Mercury	–	–	3.8–4.5
PCB	–	–	0.6–0.7

Sources:
1. This table taken from ONEB (1988). Units in mg/l, except pH.
2. Panswad 1982, Annual average of 1979–1980.
3. Pattamapirat, 1986.

Notes:
[a]Alkalinity is the capability to buffer changes of pH.
[b]BOD (Biochemical Oxygen Demand) denotes the organic content readily digestable by organisms. Length of BOD test, i.e. 5 or 7 days, not reported.
[c]COD (Chemical Oxygen Demand) is the quantity of oxygen used for the chemical oxidation process.
[d]TKN (Total Kjeldahl Nitrogen) is the sum of organic and ammonia nitrogen content.

Table D.4. Concentrations and Estimated Health Risks of Selected Volatile Organic Contaminants Measured at the On-Nuj Dumpsite (1)

Contaminant	Cancer Potency (mg/kg/dy)-1	RfD (mg/kg/day)	Concentration (ug/m3)		Exposure (mg/kg/day)		Cancer Risk Estimates			Comparison to the RfD (3) (Percent)	
							Individual Risk		Cases Per Year		
			Onsite	Community	Scavengers	Resident	Scavengers	Resident		Scavengers	Resident
Benzene	2.9 x 10-2		13	14	5 x 10-3	5 x 10-3	1 x 10-4	2 x 10-4	2 x 10-2		
Toluene		2	700	31	1 x 10-1	1 x 10-2				6%	1%
Ethylbenzene		0.1	120	6	2 x 10-2	2 x 10-3				20%	2%
m- and p-Xylene		0.3	330	12	5 x 10-2	4 x 10-3				18%	1%
o-Xylene		0.3	110	4	2 x 10-2	1 x 10-3				6%	0%
Methylene Chloride	1.4 x 10-2		26	NR (2)	4 x 10-3	-	6 x 10-5	-	1 x 10-3		

(1) Source: Table 12, Kungskulniti et al., 1989.

(2) NR = none reported

(3) Calcualted as (Exposure/RfD) x 100.

True risks are likely to be several orders of magnitude lower than these upper-bound estimates. The clay layers underlying Bangkok would probably retard movement of leachate from the landfill to the underlying aquifer. However, were the contaminants to reach a productive aquifer (through improperly constructed wells, for example), the dilution factor is likely to be greater than a factor of 300, since, as was mentioned above, the quantity of water in an aquifer available for dilution will exceed the quantity withdrawn for drinking. Both of these factors would tend to decrease the estimated risks.

Risks from contamination of air on and around the dumpsites

Only limited data are available to estimate risks from contamination of ambient air as a result of dumping or landfilling of solid and hazardous wastes. Levels of selected volatile organic compounds (VOCs) were measured at the On-Nuj dumpsite and in the community surrounding the dumpsite. The data on these compounds are presented in Table D.4. Using these data, we estimated risks from volatile organic contaminants to those living on or near open dumpsites. For these contaminants, we estimated risks for two subpopulations: those engaged in daily scavenging, and those living in the surrounding community. Those living in the community were assumed to be exposed to concentrations equal to those found in community monitoring. The actual scavengers were assumed to be working on the dumpsite 10 hours per day, and to be in the community for the remaining 14 hours per day; the concentrations to which these individual are exposed was assumed to be a weighted average of the contaminant levels found on the dumpsite and in the community. Based on these assumptions, and the assumption that the average individual breathes 20 m^3 of air per day, exposures were calculated for individuals in each of the subpopulations. The individual risks were combined with estimates of population size to obtain population risk estimates. The size of the population surrounding the On-Nuj dumpsite was reported to be 2000, with about 400 persons actually engaged in scavenging activity on a daily basis (Kungskulniti et al., 1989). Assuming that the size of the population around the On-Nuj site is representative of the size of the populations around all three of the major open dumps in Bangkok, the total population exposed to On-Nuj community levels of contaminants is approximately 6000, and those exposed to onsite levels is about 1200.

An additional population is exposed to wastes during their transport to the landfill. As mentioned above, trash is collected by a labor force of about 3,000, some or all of whom may be engaged in scavenging activity. Risks to these individuals are probably lower than those to workers at the landfills, but these risks could not be quantified.

IV. Discussion of Findings

Risks from Collection of Solid Wastes

Potential risks associated with poor collection of municipal solid wastes appear to be minimized by an effective system for waste collection. An exception is the collection of nightsoil, for which failures in the collection system probably result in significant impacts to the environment and public health. Since pickup of nightsoil from septic tanks requires payment of a fee, an estimated 80 percent of households dispose of their nightsoil illegally, by discharge or dumping in klongs, sewers, ponds, or other locations (Hubbard, 1987). The health impacts of the improper disposal of nightsoil by 80 percent of such a large population are likely to be significant. Risk from microbiological disease are discussed in Appendix F.

Risks from Disposal of Solid and Hazardous Wastes

Between collection and delivery to landfills, wastes are typically scavenged for recyclable materials, and those engaged in this activity are potentially exposed to pathogens and other health hazards. A population of several thousands of individuals is thought to live on top of or near the landfills, and to earn their livelihood by scavenging the wastes for recyclable materials. As discussed above, risks to these individuals from microbiological contaminants are likely to be high, as are risks from inhalation of suspended particles. Based on upper-bound risk estimates, groundwater contamination may pose some risk due to mercury and PCB concentrations. Results of the assessment of risks from exposure to hazardous pollutants in air are presented in Table D.4. This table shows that individual risk from the two carcinogenic contaminants measured at the site, benzene and methylene chloride, pose moderately high individual risks, in the range of 1×10^{-4}; however, because the populations exposed are small, the overall population risks (in cases of cancer per year) are negligible (less than one case per year). None of the RfDs for noncarcinogenic contaminants are exceeded; the compound with the greatest exposure as a percentage of its RfD is ethylbenzene; exposure to this compound to daily scavengers onsite contributes only 20 percent of the RfD.

A third category of wastes will be illegally dumped or otherwise disposed of improperly. The extent to which humans will be exposed to contaminants from these wastes is impossible to determine without additional data.

V. Limitations

The estimate of the major likely health risks to scavenging populations, communicable and respiratory disease, could not be addressed quantitatively. Furthermore, estimates of risk to groundwater was severely limited by the lack of information on the actual characteristics of the waste disposal sites. Furthermore, for both groundwater and air pathways, even rough estimates of risk were possible for a limited number of contaminants. It is likely that there are many more hazardous contaminants in air and in groundwater for which no measurements were available.

Appendix E. Health Risks from Exposure to Lead and Other Metals

I. Definition of Problem

This appendix discusses a dimension of Bangkok's environmental quality that spans several environmental media and numerous industrial and non-industrial activities. Its objectives are (1) to identify the magnitude of the health risks associated with exposure to lead and other metals, and (2) to identify the dominant sources of that exposure. For lead exposures, data are available to allow at least limited progress toward both of these objectives. Calculations for identifying the magnitude of total health risks from lead are based on dose-response data relating lead concentrations in the blood of Bangkok's population to health effects. Similar dose-response data are not available for other metals; their potential effects on health are discussed qualitatively.

Sources of potential exposure to lead and other metals in Bangkok include occupational exposure, inhalation of air contaminated by vehicle or industrial emissions, ingestion of drinking water contaminated by industrial effluents or the deposition from air, and ingestion of contaminated food. Food can be contaminated if it is grown in contaminated soil, or if metals from contaminated air is deposited on food as it is grown, transported, or sold at roadside vendors.

II. Data Acquired

Lead

Average Blood Lead Levels

Adverse health effects from exposure to lead, especially neurological effects, have been recognized for centuries, and have been studied in great detail in the last few decades. Since manmade sources of lead usually dominate exposure, humans living in remote areas are typically less exposed than those living in urban areas of modern societies (U.S. EPA, 1986a). Direct monitoring of an individual's or population's exposure to lead through inhalation and ingestion is difficult, but internal levels of exposure to lead can be measured through samples of any one of several biological tissues, including blood, urine, semen, hair, teeth and bone. The most common measure of exposure is the concentration of lead in blood (PbB), which is typically expressed in micrograms of lead per deciliter of whole blood (or ug/dl of "blood lead"). Because adverse health effects have long been associated with lead, and because of the usefulness of blood lead as a measure of exposure, several large cross-sectional and

longitudinal studies have quantified statistical associations between blood lead and the incidence of various adverse health effects. Experimentation with animals has provided additional understanding of some of the physiological impacts of exposure to lead, and has established causality for some health effects.

Reports of average blood lead levels in Bangkok differ by source. In 1981, Dr. W. Vbonront measured the amount of lead in blood from 100 samples nationwide plus another 100 samples in Bangkok, and reported an average concentration of 15.6 ug/dl. Dr. Nvarart at the Neurological Hospital reported 16.5 and 16.3 ug/dl for males and females, respectively. Dr. Montathip of the Department of Occupational Health (1982) sampled the blood of 1000 patients of VD clinics, with 250 samples from each of four regions of Thailand to derive an average of 16.5 ug/dl. Health Research Reports from the Division of Occupation Health report average blood lead levels of 22.7 ug/dl in 1980. Lastly, The Nation (a Thai newspaper) reported in 1989 that "Siriraj Hospital surveys show that Bangkok children registered blood lead levels of 40 microgrammes per deciliter" of blood lead, that "newborns, whose blood in the umbilical cord was tested, showed a lead content between 11 and 32 microgrammes per deciliter" and that "according to the same surveys, Bangkok adults have an average of 45 microgrammes per deciliter of lead in their blood" (The Nation, 1989d). These different estimates of blood lead levels may reflect an increase in levels between 1980 and 1989, a higher concentration in Bangkok than in the rest of the country, sampling from different populations, or other factors.

Sources of Exposure to Lead

One source of exposure to lead is the inhalation of contaminated air. We estimated average lead concentrations in ambient air (0.34 ug/m^3) by averaging reported concentrations from 7 monitoring stations in Bangkok. As an alternative estimate of air concentrations, we also used the average of curbside monitoring data (1.9 ug/m^3) from ten sample locations during the same period. Data for converting air concentrations of lead into expected increments to average blood lead levels were obtained from studies published by the U.S. Environmental Protection Agency (U.S. EPA, 1986a).

A second source of exposure to lead is the ingestion of drinking water. Lead can enter drinking water supplies through contamination of the source of the supply (e.g., point and non-point sources of lead entering the Chao Phraya north of the MWA water intake), from the distribution system if lead from pipes or solder is released into tap water prior to its consumption, or from air deposition into uncovered household water storage vessels. In Bangkok, results from water sampling suggest that average lead concentrations in water are between 20 and 50 ug/l. The lower of these estimates was

derived by assuming that values beneath the detection limit reflect zero concentrations, while the higher estimate simply excluded the six samples for which concentrations could not be determined.

A third pathway is the ingestion of contaminated food. Based on reported concentrations of lead in various food items and reported average rates of consumption for those food items, we derived estimates for levels of exposure to lead as a result of the ingestion of food. Adults are estimated to ingest 102-200 ug/day of lead with their food, and children are estimated to ingest 19-37 ug/day[1].

Other possible sources of lead include childhood ingestion of lead-based paint, or ingestion of locally-contaminated soils, such as soils near an open dump. These pathways of exposure were not investigated in this project.

Table E.1 summarizes calculations used to estimate lead exposure from individual pathways. For air, low and high estimates of slopes and air concentrations were combined to derive a range in increments of blood lead levels expected to result from inhalation of lead from the air in Bangkok. U.S. EPA (1986a) has reviewed numerous studies of the relationship between concentrations of lead in ambient air and concentrations of lead in the blood of children and adults. In summarizing those studies, the authors conclude that for adults, a reasonable slope is 2 ug/dl of blood lead for each increment of 1 ug/m^3 of lead in ambient air. For children, the corresponding slope is 3-5 ug/dl per ug/m^3, where the higher estimate includes indirect exposure from children's inadvertent ingestion of contaminated soil. As shown in Table E.1, these estimates were combined with average concentrations of lead in air to derive an expected increment of 1-4 ug/dl in the blood lead of adults, and 1-10 ug/dl in the blood lead of children.

As with ambient air, average concentrations of lead in drinking water can be converted to expected increments in blood lead levels, if we assume that studies based on U.S. data can be applied to Bangkok. Based on Marcus (1989), we assumed that each 1 ug/l increment in water lead concentration up to 15 ug/l is associated with an increment of 0.17 ug/dl of blood lead, and that each

[1]Details of these calculations to derive lead exposure from food per kilogram body weight were provided in Appendix C. For calculations particular to lead, we assume that the average child (0-3) weighs 10 kilograms and consumes the same quantity of food per unit of body weight as an adult. This assumption is likely to result in low estimates of exposure for children.

Table E.1

Estimated Contribution of Individual Exposure Pathways
To Blood Lead Levels (PbB) in Bangkok

	Exposure	Slope for Adults (ug/dl) per (ug/m^3)	Contribution to Blood Lead for Adults (ug/dl)[a]	Slope for Children (ug/dl) per (ug/m^3)	Contribution to Blood Lead for Children (ug/dl)[a]
Air	0.34–1.9[b]	2[c]	1–4	3–5[c]	1–10
Water	20–50[d]	0.06[e]	1–3	0.043[e]	1–2
Food (Adults)	102–200[f]	0.04[g]	4–8		
(Children)	19–37[f]			0.2[g]	4–7
Total[h]			6–15		6–19
Total as Pct of Current PbB[i]			15%–94%		13%–120%

[a]Estimated increment to mean blood lead that is explained by this pathway. Calculated as product of exposure and slope.
[b]From air monitoring samples taken in the Bangkok area, measured in ug/m^3. Lower estimate is average of ambient concentrations, higher estimate is average of curbside concentrations.
[c]Source: U.S. EPA (1986a). Higher value for children includes effects of indirect exposure through dust and soil.
[d]Mean values of samples from Bangkok in ug/l. Lower value sets non-detect values to zero; higher value is with non-detect values excluded.
[e]Source: Marcus (1989).
[f]Expressed in ug/day. Derivation of these estimates is discussed in Appendix C.
[g]Source: U.S. EPA (1986a).
[h]Total of estimated contributions for air, water, and food.
[i]Current blood lead levels assumed to be 16–45 ug/dl for adults and 16–40 ug/dl for children.

increment above 15 ug/l results in an additional 0.043 ug/dl of blood lead for children. Since average concentrations of lead in Bangkok's drinking water are thought to be higher than 15 ug/l, the lower slope was most appropriate for this application. For adults, each increment of 1 ug/l of lead in water was assumed to result in 0.06 ug/dl of lead in the blood. By combining these slopes with the estimated range of likely average values for lead concentrations in water, we derived estimated increments in blood lead levels for children and adults.

The estimation process is similar for lead exposure through food. As explained above, we estimate that the average adult in Bangkok ingests 102-200 ug/day of lead in his or her food. The average child ingests 19-37 ug/day. These estimates of exposure must next be combined with estimated uptake slopes for exposure through ingestion. U.S. EPA (1986a) has estimated that each 1 ug/day of exposure to lead in food for adults results in an increment of 0.04 ug/dl in blood lead for adults, and 0.2 ug/dl for children. By combining these slopes with estimated exposure for adults and children, we estimated food's contribution to blood lead levels in Bangkok.

Other Metals

Data from the Ministry of Public Health (Division of Occupational Health) shown in Table E.2 provide estimates of the "body burden" of selected contaminants for residents of Thailand. These values were obtained in about 1980, and are not specific to Bangkok. Because of the concentration of industrial activity in or near Bangkok, concentrations of these contaminants in blood for Bangkok residents are likely to be higher than for the country as a whole. In addition, rapid industrial growth over the last 10 years is likely to have led to further elevation of these concentrations. This trend may be offset in part, however, by increased efforts to reduce environmental contamination, and by reductions in the amount of lead used in gasoline. Implications of these mean values will be discussed below.

The presence of measurable contaminant concentrations in human blood, urine, or hair can provide a useful and direct measure of past or current exposure. However, because data are not available to relate the concentrations of these metals to concentrations in human tissue, the relative contribution of air, water and food exposures to total exposure cannot be determined.

Table E.2

Levels of Selected Chemicals in Biological Samples, 1980

	Tissue (units)	Estimated Distribution for Thailand[a] (Mean and S.D.)
Lead	Blood (ug/dl)	22.68 (8.60)
	Urine (ug/l)	136.88 (64.51)
	Hair (ug/g)	5.20 (3.96)
	Semen (ug/dl)	23.57 (6.88)
Manganese	Blood (ug/dl)	2.59 (1.08)
	Urine (ug/l)	24.74 (15.13)
	Hair (ug/g)	3.19 (2.41)
Cadmium	Blood (ug/dl)	1.61 (0.59)
	Urine (ug/l)	15.50 (9.64)
Mercury	Blood (ng/g)	8.47 (2.75)
	Urine (ng/l)	9.19 (8.22)

[a]Source: Division of Occupational Health Research Reports.

III. Analytical Steps to Derive Risk Estimates

Lead

Much of the available literature linking blood lead to adverse health effects is based on studies conducted in the U.S. (U.S. EPA, 1985; U.S. EPA, 1986a,b;, U.S. EPA, 1989). Even within the U.S., the association between lead and health effects (in particular hypertension in adults or elevated erythrocyte protoporphyrin levels in children) has been shown to differ according to gender, race, and even economic status. Generalization of findings from one population to another should therefore be performed with caution. In the absence of locally-derived dose-response relationships, however, this report uses findings from these studies to derive rough quantitative estimates of the extent of likely health effects from lead in the air, water and food supply of Bangkok.

Figure E.1 summarizes some of the health effects associated with different levels of lead in the blood of children. At 15-20 ug/dl, one expects to find evidence of interference with heme synthesis, interference with the functioning of the central nervous system (including statistically measurable losses to IQ), and interference with metabolism of Vitamin D. Other more recent research suggests interference with hearing and growth (Schwartz, 1986, Schwartz and Otto, 1987). At 40 ug/dl, one expects to find reduced synthesis of hemoglobin, and peripheral nerve disfunction. As shown by Figure E.2, the "thresholds" of observable effects are similar for adults. At 16 ug/dl, one expects to find evidence of effects on blood in females, and elevated blood pressure in males. At the 45 ug/dl, one can expect interference with the functioning of the peripheral nervous system, the kidneys, and the testes.

As can be seen from the Figures E.1 and E.2, the lower limits or "thresholds" of blood lead at which these effects appear are often uncertain. Blood lead levels at which effects have been "observed" have decreased markedly as prospective studies and larger data bases have allowed better resolution of statistical differences. In fact, recent research suggests that some dose-response relationships for lead exposure may be without thresholds (e.g., Schwartz, 1988). Recent studies have also suggested that even small increments in the average blood lead levels of large populations can have significant adverse effects on public health, regardless of background levels of exposure (U.S. EPA, 1985, 1986b, and 1989a,b). Moreover, researchers have found that levels of blood lead typically follow a log-normal distribution, so that a small fraction of individuals within any given population will evidence blood lead levels significantly higher than the reported average. The reported

Figure E.1

Summary of Lowest Observed Effect Levels for Key Lead-Induced Health Effects in Children

Lowest observed effect level (PbB)*	Heme synthesis and hematological effects	Neurological effects	Renal system effects	Gastrointenstinal effects
80-100 μg/dl		Encephalopathic signs and symptoms	Chronic nephropathy (aminoaciduria, etc.)	Colic, other overt gastrointestinal symptoms ↓
70 μg/dl	Frank anemia			
60 μg/dl		Peripheral neuropathies ↓		
50 μg/dl		?		
40 μg/dl	Reduced hemoglobin synthesis	Peripheral nerve dysfunction (slowed NCV's)		
	Elevated coproporphyrin	CNS cognitive effects (IQ deficits, etc.)		
	Increased urinary ALA	↓		
30 μg/dl		?	Vitamin D metabolism interference	
		↓	↓	
15 μg/dl	Erythrocyte protoporphyin elevation	Altered CNS electrophysio-logical responses		
	↓	↓		
10 μg/dl	ALA-D inhibition		?	
	PY-5-N** activity inhibition	?		
	↓			
	?			

* PbB = blood lead concentrations.

** PY-5-N = pyrimidine-5'-nucleotidase.

Source: U.S. EPA (1986a)

Figure E.2

Summary of Lowest Observed Effect Levels for Key Lead-Induced Health Effects in Adults

Lowest observed effect level (PbB)*	Heme synthesis and hematological effects	Neurological effects	Effects on the kidney	Reproductive function effects	Cardiovascular effects
100-120 μg/dl		Encephalopathic signs and symptoms	Chronic neuropathy		
80 μg/dl	Frank anemia				
60 μg/dl				Female reproductive effects	
50 μg/dl	Reduced hemoglobin production	Overt subencephalopathic neurological symptoms		Altered testicular function	
40 μg/dl	Increased urinary ALA and elevated coproporphyrins	Peripheral nerve dysfunction (slowed nerve conduction)			
30 μg/dl					Elevated blood pressure (white males) aged 40-59
25-30 μg/dl	Erythrocyte protoporphyrin (EP) elevation in males				
15-20 μg/dl	Erythrocyte protoporphyrin (EP) elevation in females				
<10 μg/dl	ALA-D inhibition				?

* PbB = blood lead concentrations.

Source: U.S. EPA (1986a)

arithmetic mean (22.68 ug/dl) and standard deviation (8.60) of blood lead for Thailand can be used to show that the geometric standard deviation (GSD) of blood lead in Thailand is identical to those of blood lead distributions measured in the U.S. and elsewhere (1.4 ug/dl), so that 2-3 percent of individuals can be expected to have blood lead levels more than twice as high as their population's geometric mean.

The methods used to estimate health effects from lead for this report are nearly identical to those described in detail in U.S. EPA (1989a) and U.S. EPA (1989b), and are quite similar to those currently being used by the U.S. EPA Office of Drinking Water to estimate expected benefits from alternative standards for lead in drinking water in the U.S. These methods have been described in detail in previous documents, and will be summarized only briefly here. In general, we apply numerous dose-response curves to population mean blood lead levels to compare expected rates of particular health effects under baseline and improved environmental conditions. Although tentative dose-response relationships are available for many more health effects than are evaluated here, this analysis selects only those effects that have previously been used and are currently being used for major regulatory impact analyses in the U.S. These include the effects of lead on blood pressure, heart disease, and death in adult males, and on neurological development and the need for medical treatment of asymptomatic individuals for children. Recent research has linked lead exposure for pregnant women to adverse effects on the developing fetus (Davis and Svendsgaard, 1987) and even to possible increases in rates of infant mortality (U.S. EPA, 1989b). Other research has indicated possible health impacts for exposed women (Rabinowitz, et al., 1987; Silbergeld et al., 1988). However, because controversy still surrounds efforts to quantify those effects, they have not been included in the present analysis.

It is assumed, based on the information described above, that background blood lead levels in Bangkok average 16-45 ug/dl for adults, and 16-40 ug/dl for children. The low estimate (16 ug/dl) for Bangkok resembles average levels of blood lead in the U.S. in 1978 as measured in the second National Health and Nutrition Examination Survey (NHANES II): 14.9 ug/dl for 2-year old children, 10.8 ug/dl for women of ages 15-45, and 15.4 and 17.7 for 40-59 year old white and black men, respectively (National Center for Health Statistics, 1981).

Estimates of significant adverse health risks associated with those levels of blood lead in the U.S. motivated the phase-down of lead in U.S. gasoline and attempts to reduce lead concentrations in drinking water and food. Because of these efforts, current blood lead levels in the U.S. are now estimated to have fallen to 6-8 ug/dl in children and 4-6 ug/dl in adults. To derive a rough indication of the impact of lead on public health in Bangkok, this analysis estimates the health benefits that might

be expected if controls in Bangkok could reduce blood lead concentration to levels comparable to current levels in the U.S.

For children, two health effects are examined in this analysis: interference with intellectual development and the need for remedial treatment for asymptomatic children. Both of these effects have short-term and long-term economic implications. The U.S. Center for Disease Control (CDC) has developed a protocol for testing and treatment of children exposed to lead. Individual children are classified into groups based on measured levels of erythrocyte protoporphyrin and blood lead (see Figure E.3). The CDC's classification scheme includes six categories (I, Ia, Ib, II, III, and IV) and the level of prescribed medical treatment increases with each level of classification. Children classified into Group IV are considered at urgent risk. Immediate treatment (usually chelation therapy) is required; higher blood lead levels within Group IV imply significant risks of severe and permanent brain damage.

For adults, only males of ages 20 years or more are considered in the analysis, because the best available estimates of dose-response relationships were derived for this group. Only cardiovascular effects from lead exposure are considered because reliable quantitative dose-response relationships are not available for other effects. First, the analysis uses results from a multi-variable logistic regression of blood lead versus hypertension (high blood pressure) to estimate the fraction of adult males (of age 20 years or more) likely to have hypertension at various levels of mean blood lead. (Hypertension is defined for these calculations as diastolic blood pressure exceeding 90 mm Hg). Second, the results, from a semi-log regression of blood lead versus blood pressure, are taken to estimate likely shifts in the distribution of blood pressures that are expected to result from changes to population mean blood lead. As argued by Pirkle et al. (1987) and U.S. EPA (1986b, 1989a), increases in blood pressure can be expected to result in increased risks of heart attack, stroke, and death. We, therefore, combine estimated shifts in average blood pressure with results from multi-

Figure E-3.

Risk Classification of Asymptomatic Children

Blood Lead (ug/dl)	---- Erythrocyte Protoporphyrin (EP) ----			
	<35	35-74	75-174	>175
Not done	I	a	a	a
<24	I	Ia	Ia	b
25-49	Ib	II	III	III
50-69	c	III	III	IV
>70	c	c	IV	IV

a. Blood lead test required to identify risk.

b. Erythropoietic protoporphyria.

c. This combination not generally observed. Retest.

Source: CDC (1985). Preventing Lead Poisoning in Young Children. Table 2-A, p. 11, taken from U.S. EPA (1987b).

variable logistic regressions of rates of heart attacks, stroke, and death against blood pressure. Data for these latter regression coefficients were obtained from large prospective studies of white males (ages 40-59) in the U.S.

Because estimates of total risk depend on the number of individuals vulnerable to each of the health effects discussed above, this analysis uses local demographic data to determine the number of children of ages 0-7, the number of adult males 20 or more years of age, and the number of adult males of ages 40-54 or 40-59. The total population of Bangkok is assumed to be approximately 5.9 million.

Other Metals

As discussed above, dose-response relationships are not available to relate concentrations of other metals in human tissues to potential health effects. However, comparing the findings reported in Table E.2. to literature findings on the health effects of these metals allows a qualitative evaluation of the extent to which exposures to these metals may result in health risks for the Thailand population, and presumably, for Bangkok residents.

IV. Discussion of Findings

Lead

Total Exposure to Lead

Table E.3 summarizes estimates of some of the health benefits that might be obtained if levels of exposure to lead in Bangkok could be reduced to current levels in the U.S. Not surprisingly, estimates differ markedly according to assumptions about background blood lead levels for Bangkok. For all three scenarios, however, the potential benefits of reducing exposure appear substantial. On the assumption that 40-60 year old males in Bangkok respond to blood lead and high blood pressure with patterns similar to those detected in epidemiological studies of white men in the U.S., as many as 0.5 million men in Bangkok could have hypertension as a result of exposure to lead above levels in the U.S. Increased blood pressure as a result of exposure to lead can be expected to result in increased risks of heart attack, stroke, and death. Based on studies linking blood pressure to these effects in the U.S., and on the assumption that these studies are also applicable to men of similar age

Table E.3

Estimated Health Benefit from Reducing Blood Lead Levels in Bangkok To Current Levels in U.S.[a]

	Adult Background 16 ug/dl PbB Children's Background 16 ug/dl PbB[b]	Adult Background 23 ug/dl PbB Children's Background 23 ug/dl PbB[c]	Adult Background 45 ug/dl PbB Children's Background 40 ug/dl Pbb[d]
Adults			
Hypertension (cases/yr)[e]	210,000	320,000	530,000
Heart Attack (cases/yr)[f]	210	320	430
Stroke (cases/yr)[f]	110	210	430
Death (cases/yr)[f]	210	210	430
Children			
CDC Group IV (cases/yr)[g]	530	7,500	64,000
1–2 Points IQ (cases/yr)[h]	75,000	96,000[i]	32,000[i]
4+ Points IQ (cases/yr)[h]	4,300	32,000[i]	110,000[i]
Total Points (pts./yr)[j]	430,000	530,000	750,000

[a]Represents hypothetical benefit to be expected if blood lead levels in Bangkok could be reduced to those currently estimated in the U.S. (5 μg/dl for children and 6.7 μg/dl for adult males). Case estimates require the assumption that epidemiological evidence from the U.S. can be generalized to populations in Bangkok. Since behavior patterns and disease incidence rates differ markedly among populations, results should be interpreted with caution. All results have been rounded to one significant figure.

[b]Based on results from Dr. W. Vbonront of Chlalwhon University (1981) of 15.6 μg/dl (from 200 samples in Bangkok and nationwide), an average of 16.5 μg/dl from a national sample of 100 patients in VD clinics by Ms. Montahip of NOPH, and an average of 16.5 μg/dl for males and 16.3 μg/dl for females, as reported by Dr. Nuavant at the Neurological Hospital. Average levels in Bangkok may be higher than national averages.

[c]From the Meeting of the Expert Committee on Lead Poisoning. Estimated average blood lead levels were 22.68 for Thailand in 1980. Average values for Bangkok may be higher than national averages.

[d]From "To Live and Let Live in a City Loaded with Lead," The Nation, 17 February, 1989. Represents reported estimates for children from surveys at the Siriraj Hospital.

[e]Represents incremental cases of diastolic blood pressure exceeding 90 mm Hg. Based on results from multiple logistic regression, as described in U.S. EPA, 1989b.

[f]Assumes that increased blood pressure caused by lead results in increased risks of cardiovascular disease and death. Logistic dose–response functions are combined with an assumed semi–log relationship for blood lead – blood pressure. For details see U.S. EPA, 1989b.

[g]Represents decrease in number of children in this CDC category as a result of reducing children's average blood lead levels. Asymptomatic children in this category require immediate medical attention if risk of serious brain damage is to be avoided.

[h]Represents children with lead–induced losses in IQ. Even small losses to large populations may result in statistically significant reductions in total productivity, if losses to IQ persist into adulthood.

[i]At higher blood lead levels, large reductions in IQ become more common, and smaller reductions become less common.

[j]Represents total points of IQ lost for each year's cohort of 7–year olds. Losses are summed across all affected individuals to derive an aggregated total. For U.S. workers, the present value of lost future earnings for one point decrement in IQ has been estimated to be about $500 (U.S. EPA, 1989b).

in Bangkok, as many as 400-800 men could suffer heart attacks or strokes, and as many as 200-400 men per year could die as a result of excess exposure to lead.

Results for children are equally dramatic. As shown in Table E.3, our calculations suggest that as many as 50,000 to 60,000 children would be expected to fall into the Group IV category of the CDC's classification scheme as a result of differences between blood lead levels in Bangkok and those in the U.S. Treatment of these children to reduce blood lead and erythrocyte protoporphyrin levels is expensive; failure to treat them is likely to result in neurological damage or other health effects that, if allowed to persist, will continue into adulthood.

Included in this analysis is lead's interference with mental development. Based on regression results from Fulton et al. (1987), we estimate that 30,000-70,000 children could suffer losses of 4 or more IQ points as a result of exposure to lead in excess of levels now encountered in the U.S. A far larger population will show lesser reductions in intelligence, leading to a total estimate of 400,000-700,000 points of lost IQ for the city as a whole. If these losses persist throughout each affected individual's lifetime (and new research suggests that losses do in fact persist), and if associations between IQ and economic productivity estimated for the U.S. can be generalized to Bangkok, then these lost IQ points would be likely to result in significant economic losses for Bangkok.

Note that results are quite sensitive to assumptions about average blood lead levels in Bangkok. Even with the more conservative assumptions of background blood lead levels, (levels roughly comparable to those observed in the U.S. 10-15 years ago), these results suggests that benefits from such reductions would be substantial, and that reducing exposure to lead is a policy objective worthy of high priority. Of course, if exposure to lead could be reduced still further, even more benefits could be achieved.

Contribution of Individual Exposure Pathways to Lead Exposure in Bangkok

In order to reduce human exposure to lead in Bangkok, it is important to determine the sources of that lead exposure. Although sufficient data for precise identification of those sources are not available, we have derived at least rough estimates of the relative importance of three likely pathways, as explained above. Table E.4 summarizes estimates of the contribution to total blood lead levels explainable by each of the three pathways (air, water and food) examined individually. As can

Table E.4

Relative Contribution of Three Pathways of Exposure
To Lead In Bangkok[a]

	Adults	Children
Percent from Air[b]	8%–40%	10%–70%
Percent from Water[c]	10%–40%	5%–30%
Percent from Food[d]	40%–80%	20%–80%

[a]Based on estimated increment to blood lead levels caused by exposure through each medium, as reported in Table F.1. Percentages represent individual medium as fraction of total increment in blood lead accounted for by these three media of exposure. Ranges represent uncertainty caused by alternative assumptions for all three exposure pathways. For each combination of assumptions, totals for the three media sum to 100%, by definition. Potential contributions from other sources are not considered in these estimates, but are included in the results presented in Table F.2.

be seen from Table E.1, combining "high" estimates for each of the three pathways leads to a sum of total exposure of about 15 ug/dl for adults, and about 19 ug/dl for children. If current values of blood lead in Bangkok are actually 40-45 ug/dl (as reported in The Nation (1989d)) these totals leave much of the current body burden unexplained. If, on the other hand, actual values are closer to the lower end of the range evaluated, then most or all of the current level of lead in blood can be explained by air, water, and food.

Table E.4 provides a modified presentation of results from Table E.1. To capture the relative importance of these three pathways, the Table E.4 provides estimates of each pathway's contribution as a fraction of the sum from all three pathways. For a lower bound estimate of the relative importance of air, for example, it is assumed that air contributes 1 ug/dl, water contributes 3 ug/dl, and food contributes 8 ug/dl. With these assumptions, air would contribute about 8 percent of the total from all three pathways. Note that this result is independent of the assumption used for background blood lead levels in Bangkok. As can be seen from Table E.4, food appears likely to dominate non-occupational exposure for adults, followed by approximately equal contributions from air and water. For children, water appears to be the least important pathway, which suggests the relative importance of air and food, but this is impossible to determine with certainty.

If total health risks from current blood lead levels are indeed as high as suggested by Table E.2, and if air, water, and food all appear to be significant contributors to total exposure, then the next logical research objective is to identify the sources of exposure through each pathway, and the most promising methods of reducing them. A brief discussion of each of these three pathways is provided below.

Exposure to Lead from Ambient Air

Lead is released to ambient air from a variety of sources, among them emissions from vehicles using leaded fuels and from smelters and other industrial sources. Estimates of health effects associated with leaded gasoline have motivated the phase-down of lead in gasoline in the U.S., resulting in marked reductions in average blood lead levels. Gasoline-powered vehicles, which constitute 90 percent of the cars and 20 percent of light trucks and buses in Bangkok, rely on fuels with relatively high lead content, so that average levels of lead in gasoline sold in Bangkok are approximately 0.45 g/l, compared to the current standard of 0.026 g/l in the U.S. (The Nation, 1989d). Recent articles in The Nation (1989a-f) have provided conflicting reports of expected concentrations of lead in gasoline in the near future. Government officials have stated that lead levels in gasoline will soon be reduced to 0.15 ug/l, but other evidence suggests that such a rapid reduction is unlikely, given constraints imposed by existing refinery characteristics and lead requirements of the existing fleet of vehicles. Another likely source of lead in

the air of Bangkok is emissions from industrial facilities. Lead and zinc smelters, in particular, are known to have demonstrable effects on blood lead levels in surrounding areas.

Exposure to Lead in Drinking Water

Lead can enter drinking water through contamination of the source, or through subsequent contamination within the distribution system and household storage containers. Controversial plans to route a 3 kilometer section of the Changwattana-Bangklo expressway over the Prapa Canal (which supplies 25 percent of Bangkok's drinking water) have led to public concerns that the expressway might lead to the deposition of additional lead into the water supply (The Nation, 1989a-d). In defense of their proposed routing, the Expressway and Rapid Transit Authority of Thailand (ERTAT) argues that in a worst case scenario, the maximum amount of lead in the canal would be 25 ug/l. Opponents question ERTAT's projections, however, and argue that the true amount will be higher. A study conducted by the National Environment Board (NEB) and other agencies concluded that the maximum concentration expected in the canal would be 34.6 ug/l. In either case, examination of Table E.1 shows that at 20-50 ug/l, lead concentrations in Bangkok's water may already place a significant burden on the public health of Bangkok residents. Any sizable increase to exposure could be expected to result in non-trivial health risks. Proponents of the plan also argue that future reductions in the lead content of gasoline will reduce potential contamination of the canal. As briefly mentioned above, however, others contend that major reductions in the lead content of gasoline used in Bangkok are unlikely, at least within the next few years.

Exposure to Lead from Food

As can be seen from Table E.4, food appears to be a significant contributor to total exposure to lead. If reported average concentrations of lead assumed for various food items in Appendix C are accurate, then as much as 80 percent of total exposure may occur through this pathway. The source of the lead content in food cannot be identified from data available for this analysis, but one possible source is the deposition of airborne lead onto soil and plant surfaces. If, for example, lead smelters are located near agricultural areas, then soil and crops might be contaminated by lead deposited from smelter emissions. Another possibility might be the use of contaminated water for irrigation. Deposition of lead particles on food sold at road-side stands may be important. Results from this analysis suggest that determination of the source of lead contamination in food should be given a high priority.

Other Metals

Table E.2 shows that average measurements of manganese concentrations in the hair of Thai subjects were 3.19 ug/g in 1980, with a standard deviation of 2.41 ug/g. Recent epidemiological research has found that such levels can lead to measurable losses of neurological function. One study, for example, compared populations from three areas of the same region of Greece in which manganese (Mn) concentrations in drinking water were known to vary widely. A random sample of 188 individuals, all of whom were over 50 years of age, was drawn from the three areas. The subjects were tested for concentrations of manganese in blood and urine and were submitted to a thorough neurological examination. Average concentrations of Mn in the hair of these subjects were 3.51, 4.49, and 10.99, for groups A, B, and C respectively; individuals from these three groups showed progressively higher prevalence of neurological scores indicating chronic manganese poisoning (Kondakis et al., 1989). If the current concentrations of manganese in the hair of Bangkok residents are similar to the 1980 levels for Thailand as a whole, then we would expect approximately 20% of the residents of Bangkok to have hair concentrations of manganese exceeding those of group B of the Kondakis et al. study, for which neurological effects were statistically detectable. Of these, about 10 percent (or about 100,000 persons) could be expected to be 50 or more years of age.

More recent data from Institute of Environmental Research (1984) suggest lower levels of manganese in the blood of Bangkok residents than estimates provided in Table E.2, with an average of 1.47 ug/dl and standard deviation of 0.80. This same study found that manganese levels for workers in a dry cell plant averaged 2.13 ug/dl, with a standard deviation of 0.75 ug/dl. A similar study conducted in Belgium compared 141 workers in a plant producing manganese oxides and salts with 104 controls. The manganese workers were found to have concentrations of manganese ranging from 0.10 to 3.59 ug/dl, while the controls ranged from 0.04 to 1.31 (Roels et al., 1987). That concentrations of manganese in the blood of average Thai citizens are comparable to the concentrations observed for manganese workers in Belgium is disturbing.

Table E.2 shows that the average concentration of cadmium in urine is estimated to be 15.50 ug/l for Thailand. These concentrations were found to have a rather large standard deviation , so that about 90 percent of all values would be expected to fall between 0 ug/l and 30 ug/l. By comparison, Kowal et al. (1979) found that blood cadmium levels in the U.S. range from 0.59 to 0.77 ug/l. Concentrations of cadmium in Thai blood are also high. The Division of Occupational Health Research Reports list an average of 1.61 ug/dl for cadmium in blood, with a standard deviation of 0.59. For comparison, a recent

study in the U.S. examined cadmium levels in 40 male workers from a primary smelter, along with those of 36 male hospital workers taken as controls (Mueller, et al., 1989). The mean concentration of blood cadmium for the smelter workers was 1.02 ug/dl; the mean for controls was 0.2 ug/dl. The authors found elevations of N-acetyl-beta-D-glucosaminidase (NAG), urine alanine-aminopeptidase (AAP), and gamma-glutamyltranspeptidase (GGT) in the smelter workers as compared to the controls, suggesting chronic renal tubular nephrotoxicity. With a mean of 1.61 and standard deviation of 0.59 ug/dl, 85 percent of the residents of Thailand (and presumably of Bangkok) would be expected to have blood cadmium levels higher than the smelter workers analyzed in that study.

As reported in Table E.2, average mercury levels in Bangkok were 8.47 ng/g (SD=2.75) and 9.19 ng/1 (SD=8.22) for blood mercury and urine mercury, respectively[2]. According to U.S. EPA (1976), the lowest whole-blood concentration of methyl mercury associated with toxic symptoms is about 200 ug/g; values reported for Thailand are well beneath this standard. Chronic exposure to mercury has been associated with fetal effects, damage to the peripheral and central nervous systems, and possibly with increased incidence of leukemia (Janicki, et al., 1987).

As discussed above, exposure to lead appears high enough to cause significant adverse health problems. In addition, recent research with rats has found that the absorption of lead, cadmium, and manganese is increased by administering these metals jointly. Simultaneous exposure increased brain content of all three metals, concentration of manganese in the liver, renal concentration of lead, and renal and testis content of cadmium (Shukla and Chandra, 1987). If these effects are also applicable for humans, then the cumulative damage caused by simultaneously high levels of all three of these metals (as observed in Thailand) may exceed the health effects predicted based on concentrations of the contaminants individually.

Reports of average body burdens of lead, cadmium, and manganese indicate alarmingly high exposure to these metals in Thailand. By themselves, however, these high values do not identify the industrial, occupational or domestic practices or natural conditions responsible for the exposure, or the environmental pathways through which it occurs. High blood levels in Thailand for cadmium, lead, and manganese (and possibly higher levels in Bangkok) might possibly be explained by the prevalence of metals-related industries in the country. Dry cell battery plants, and five of the country's seven lead smelting plants are found in and around Bangkok (NEB, 1989). According to NEB (1989), the metal

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[2]The extraordinarily low values reported by this source probably reflect misreporting of the units of measure, but correct units could not be determined.

smelting industry within Bangkok produces more than half a million tons of hazardous wastes per year, most of which is heavy metal sludges and solids.

A clear association between proximity to metal smelters and concentrations of heavy metals in blood has been demonstrated repeatedly in scientific literature throughout the world (see, for example, Silvany-Neto, et al., 1989; Carvalho, et al., 1989; Chenard, et al., 1987). Air emissions from smelters can lead to substantial exposure through direct inhalation of the metals, and also to indirect exposure through deposition on food, or on soil that is inadvertently ingested. Discharge of contaminated effluent into streams can lead to contamination of drinking water or fish. Soil concentrations of heavy metals near smelters tend to be quite high; for example, Maravelias et al. (1989) found concentrations of 1,300-18,000 parts per million near a smelter in Greece. Finally, concentrations of metals in the blood of smelter workers are typically elevated. It is quite plausible, then, that the prevalence of the metals and smelting industries in Bangkok may be largely responsible for high body burdens of lead, cadmium, and manganese.

Based on the high reported concentrations of lead in the food supply of Bangkok, one might expect to find high levels of metals in soil from the agricultural regions where much of the agricultural produce consumed in Bangkok originates. Similarly, it is possible that soil concentrations of lead, cadmium, and manganese are high in the Bangkok area, leading to additional exposure (especially for children) through inadvertent ingestion of soil or dust. The relatively inexpensive task of sampling soil and/or food products from these areas could help clarify the causes of exposure to these metals.

V. Limitations

As mentioned above, all of the results reported in Tables E.1 through E.4 must be interpreted with caution, because they involve the application of results from studies in the U.S. to Thai populations that could differ dramatically in behavior patterns and physiological response. In addition, results are sensitive to values for parameters that are not known with certainty, as illustrated by the ranges of results reported in the preceding tables. Nevertheless, even within these limitations, our results are sufficient to suggest that health effects from exposure to lead in Bangkok are likely to be substantial, and that no single source of exposure can be dismissed as insignificant.

Much of the discussion of risks from exposure to other metals relies on reported averages for tissue concentrations of heavy metals for residents of Thailand. If these averages are not accurate, or if they are not applicable to Bangkok, then much of the interpretation offered above may be inappropriate.

Appendix F. Health Risks from Microbiological Disease

I. Definition of Problem

This appendix evaluates the health risks in Bangkok associated with a wide range of diseases that are related to environmental pollution and caused by microbiological agents. The diseases include acute diarrhea, dysentery, cholera, dengue fever, and many others. The agents responsible for the diseases include bacteria, protozoa, viruses, and helminths (worms).

These diseases are very prevalent in developing countries and tropical climates. Their incidence is closely related to poverty, poor sanitation, poor housing, malnutrition, limited water supplies, lack of sewage disposal and treatment, and inadequate health care and education. Some of these contributing factors are associated with environmental pollution and are thus relevant to this project (e.g., lack of sewage treatment). For this reason, we are including some microbiological diseases among the health risks caused by environmental problems in Bangkok. However, many other factors contributing to these diseases are outside the scope of this project (e.g., malnutrition). We have evaluated the microbiological diseases that are common in Bangkok and selected the 14 that are most closely related to environmental pollution to cover in this appendix. The microbiological diseases we have termed "environmentally related" include:

- o Acute diarrhea
- o Dysentery
- o Enteric fever (typhoid, paratyphoid)
- o Encephalitis
- o Tetanus
- o Acute poliomyelitis
- o Typhus and other rickettsioses
- o Dengue hemorrhagic fever
- o Malaria
- o Cholera
- o Hepatitis A
- o Rabies
- o Leptospirosis
- o Helminthiases

Table F.1 shows that these diseases together are responsible for about 16 out of 300 deaths per 100,000 persons that occur each year in Bangkok (or roughly five percent of the deaths). Diarrhea in particular is one of the leading causes of infant mortality. These diseases are undoubtedly responsible for a substantially higher proportion of morbidity in Bangkok. Microbiological diseases are common, treatable, and transitory; they rarely result in death.

Table F.1 Death Rate per 100,000 population (all ages) by cause of death, Bangkok, 1983-1986

Diarrheal diseases	10.6
Hemorrhagic fever	2.7
Malaria	2.7
All other infectious diseases	-
Rabies	-
Tetanus	-
Tuberculosis	5.3
Malignancy	58.4
Diabetes mellitus	8
Psychosis/drug dependence	5.3
Meningitis, encephalitis, epilepsy	-
Heart diseases, hypertensive diseases	47.8
Cerebrovascular diseases	13.3
Pneumonia, bronchitis, asthma	13.3
Peptic ulcer, appendicitis, intestinal obstruction	2.7
Cirrhosis of liver, hepatitis	15.9
Nephrosis, prostrate	-
Obstetric and perinatal causes	5.3
Pyrexia of unknown origin	8
Ill-defined cause	18.6
Accidents and injury	31.9
Suicide	5.3
Homicide	10.6
Senility without psychosis	29.2
Tyroid	-
Anemia	2.7
Congenital anomalies	-
TOTAL	297.6

Source: "Review of the Health Situation in Thailand: Priority Ranking of Diseases." NEB, 1987.

Most of the agents responsible for these diseases are spread through oral ingestion of human feces. An infected individual excretes the agent, which may live and (in some cases) multiply in the feces. Another individual can be infected through eventual ingestion of the fecal matter itself, or fecally contaminated water, food or soil. There are two steps in the infection via this pathway. First, fecal matter must be present in the human's immediate environment. This can result from unsanitary use/care of latrines, indiscriminate defecation by children, seasonal flooding which brings fecal material up from cesspools and into surface water or klongs, other release of sewage into surface water or klongs, etc. For infection to occur, humans must make the necessary type of contact with the source. Generally speaking, this would mean direct contact, with transfer of fecal matter from, for example, hands to mouth, or ingestion of fecally contaminated water or crops fertilized with sewage. The specific pathways are quite varied and do not lend themselves to generalization. They can range from a mother with fecal matter contaminated with a diarrheal agent under her fingernails infecting her infant while breast-feeding, to eating raw produce that has been freshened at the market by fecally contaminated water. Furthermore, the degree of importance of each pathway varies with each disease; for some, contaminated water may be a primary source of inoculation (bacterial diarrhea), whereas for another, walking barefoot on untreated human waste may be the chief source (ascariasis).

Another group of diseases is transmitted by vectors such as insects (mosquitos, flies, lice), arachnids (mites), and animals (rats). Typically, the agent is transmitted to the human through the bite of a vector, as is the case with malaria and rabies; however, other transmission routes do exist for other diseases.

All of the diseases which are not formally vector-related can also be transmitted via vectors in the following fashion. Pathogens can be transported on the legs and bodies or in the digestive tracts of certain vectors, particularly flies, cockroaches, and other insects which breed in and/or eat feces. For example, a fly breeding near a latrine could pick up *Shigella* bacteria from infected feces, then land on uncovered human food, where this bacteria could multiply to a level high enough to infect those who eat the food. This pathway is normally a less significant route of transmission.

Environmental conditions in Bangkok of concern in this project can contribute to the spread of these diseases in six important ways.

1. Lack of water. Having a reliable potable water supply available in the home for washing and drinking is a key preventive measure for these diseases. When water is unavailable or in limited supply in the home, cleaning of one's person, surroundings, food and utensils all decline. Individuals without a water connection may purchase or acquire limited amounts of it from community stand-pipes or elsewhere, perhaps carrying it home in unsanitary containers. Erratic water supplies may necessitate water storage, and uncovered water storage containers ("klong jars") can provide breeding areas for several disease agents.

Some, but relatively few, Bangkok residents are without a water supply in their homes. About 75% of Bangkok's population is provided with piped water by the Metropolitan Water Authority (MWA), a higher proportion than prevails in many other large cities in developing nations. Many among the remainder of the population are served by piped water obtained from licensed or unlicensed private ground-water wells. Even in the areas generally served by piped water, though, not all residences have it. In the Klong Toey slum, which is connected to the MWA network, 13% of the residences do not have a water connection (Mansoor, 1990).

However, water supplies to homes provided with piped water are not always reliable. In many areas served by the MWA, water pressures are low and erratic. MWA has difficulty supplying sufficient water to meet Bangkok's rapidly growing demand and maintaining adequate pressure throughout the system. As a result, some customers have installed pumps to draw water through the distribution system to their homes, supplementing the erratic system pressure. Another result is that many customers store water during periods when pressure is available, to provide for their needs during periods when water pressure is insufficient. Ninety-six percent of the Klong Toey residents were found to store water (Mansoor, 1990).

2. Contaminated water. Many of the microbiological agents can live in water and are commonly transmitted when contaminated water is used for drinking or bathing. Although water supplied to most of Bangkok's population is well treated and meets bacteriological standards upon leaving the treatment plants, problems in the distribution system frequently result in contaminated water at the tap. A basic principle in management of a water supply system is to keep the distribution mains and pipes under positive pressure, so that holes in the pipes result in clean water leaking out rather than potentially contaminated water leaking into the pipes. In Bangkok, though, low water pressures and even negative pressure (from suction pumps at residences) in some areas apparently allow substantial infiltration of external water into the distribution system. The water distribution pipes often go through highly contaminated areas -- klongs, septic fields, backed-up storm drains, etc. -- and infiltration of external

water introduces microbiological agents into the drinking water supply. Bangkok residents are quite aware of the frequent microbiological contamination of their tap water, and many of them commonly purchase bottled water or boil tap water for drinking. However, they may still be exposed to waterborne disease agents through bathing or washing food, containers and implements in contaminated water. Infiltration of contaminants into the water distribution system should decline over time as the system is upgraded. An indicator for the physical integrity of the distribution system is the volume of "unaccounted-for" water -- water sent from the treatment plants into the distribution system but ultimately not received by customers. Unaccounted-for water has declined as a proportion of the water supplied by the MWA from 63% in 1971 to 34% in 1988 (Government of Thailand, 1989).

3. Lack of sewage conveyance. The bulk of the microbiological diseases of concern are transmitted by the fecal to oral pathway. This pathway can be broken by assuring that human excrement is conveyed away from people. In Bangkok, however, much of the human waste remains near people and domestic animals. Separate sanitary sewers are rare. Typically, domestic waste treatment consists of individual septic tanks or cesspools. The treatment provided by these units is minimal in Bangkok's moist, clay soils, and the density of fecal loadings in many areas is far greater than the soil's assimilative capacity. Homeowners may allow septic tanks to fill up and overflow. Furthermore, as discussed in Appendix B, septic tanks are often illegally connected by pipes directly to storm sewers and klongs to avoid the charge to the homeowner for collection of nightsoil. Often, the storm sewers and klongs themselves are blocked or inappropriately constructed, providing little drainage. Thus, the fecal solids and liquids retain their virulence and remain near where people live, eat, and recreate, increasing the potential for direct contact with fecal matter. That material which does flow through the klongs drains to the river, where those living or working in river communities may be exposed. In a survey of the Klong Toey slum, one of the poorer areas of Bangkok, but by no means unique, researchers found fecal solids exposed outside of 5% of the houses surveyed, and fecal solids exposed in cesspools beneath 70% of the houses. Even in an upgraded area of the slum, where storm drains were built to carry domestic wastewater away, 30% of the residences were found still to rely on cesspools where wastewater collected and did not flow to the drains. Fifty-five percent of the residences were connected to drains that had obvious restrictions on flow that caused wastewater to accumulate, and only 15% were connected to drains that provided the intended efficient flow of wastewater (Mansoor, 1990).

4. Lack of sewage treatment. Appropriate treatment of sewage, in a sewage treatment plant or in septic tanks and cesspools under proper loading and soil conditions, will remove nearly all the harmful microbiological agents from wastewater. After such treatment, wastewater can then be safely discharged into waterways to which humans and animals will be exposed. With more treatment, the

wastewater can safely be used for irrigation of crops or even domestic water supplies. In Bangkok, only 2% of the city residents are served by sewage treatment plants. The remainder either discharge their sewage directly into storm drains, klongs or the river, or have it treated (often inadequately, as described above) in septic tanks and cesspools. Inadequately treated or untreated wastewater is responsible for the high levels of microbiological contamination of the Chao Phraya and Bangkok's klongs. This wastewater may also contaminate ground water used for drinking purposes. Microbiological agents in wastewater are very unlikely to percolate through the clay layers overlying Bangkok's aquifers, but they may contaminate ground water when an improperly constructed or located water well provides a conduit from the surface to the underlying aquifer.

5. Uncollected solid waste. About 80% of Bangkok's solid waste is collected and either scavenged or taken to one of three dump sites. The uncollected remainder can provide food and breeding ground for vectors such as rats and flies involved in transmission of disease. Uncollected solid waste may also clog storm drains, contributing to accumulation of fecal matter and wastewater, exacerbating flooding, and providing pools of stagnant water for mosquito breeding. More information on solid waste disposal can be found in Appendix D.

6. Flooding. Periodic flooding in Bangkok can bring fecal material and people into closer contact. During flooding, klongs and storm drains that normally convey sewage away can back up and overflow; cesspools may overtop with a rising water table; people may have to wade through contaminated water that they can normally avoid; and flood pools may also provide breeding grounds for mosquitos. We list flooding as one of Bangkok's environmental problems because it is one result of ground-water withdrawals and land subsidence.

This list of multiple ways in which environmental problems in Bangkok contribute to increased incidence of microbiological diseases is not meant to imply that these diseases are caused exclusively by environmental factors. To the contrary, factors that we consider "non-environmental", or outside the scope of this study, are probably more important. Critical non-environmental factors that contribute to disease incidence include:

o Poor personal hygiene. Cleaning, washing, wiping after defecating, keeping flies and mosquitos away, wearing shoes, etc. all are important.

o Inadequate health care and education. Several of these diseases may be prevented by immunization, and all can be treated effectively. Better knowledge about which practices to

avoid and which to emphasize would be helpful. Street food vendors, for example, often wash their utensils and dishes quickly in water before use by another customer. The water is often contaminated, and it might be better instead to wipe the materials with a more sanitary paper towel which is subsequently disposed.

o Lack of/non-use of toilets. Again, it is important to keep fecal material away from people. Lack of toilets or indiscriminate defecation, typically by children, either in the home or outside, creates problems.

o Overcrowding and inadequate housing. Close living conditions increase person-to-person disease transmission. Lack of running water, toilets, screens on windows, refrigeration and garbage disposal facilities increases incidences of disease.

o Poor nutrition and food preparation. Malnutrition not only makes individuals more susceptible to many of the microbiological diseases, but increases the severity of the diseases once they are contracted. Improper food preparation and storage -- leaving foods out and exposed, insufficient cooking, etc., -- can contribute to many problems.

II. Analytical Methodology and Data Used to Develop Risk Estimates

Overview

Microbiological agents require a much different method of risk assessment than do the chemical contaminants evaluated in the remainder of this study. Health impacts from chemical contaminants are estimated using standard risk assessment techniques that calculate the risk posed by a contaminant by comparing the degree to which people are exposed to a contaminant (the dose) to a measure of the likelihood that the contaminant will cause a certain health effect at that exposure level (such as an RfD, cancer potency factor or other dose-response function). This estimate is then multiplied by the number of people so exposed to estimate the risk to Bangkok's population. This approach is not applicable to the analysis of risks from microbiological agents for several reasons:

o No satisfactory dose-response functions are available for microbiological agents that indicate the likelihood of contracting disease given a dose of the causative agent. Previous exposure (i.e. level of resistance), level of nutrition, and varying response to different strains are some of the complicating factors which have hindered development of dose-response functions.

o Even if dose-response relationships were available, there is virtually no appropriate monitoring data on the levels at which different microbiological agents are present in the numerous environments in which humans may be exposed to them. Concentrations of these agents are extremely variable with time, place and human activities.

For environmentally-related microbiological diseases, an alternative risk assessment procedure was used that required collection of different types of data than for the other environmental problems. Data were collected to support four steps in our analysis:

1. Identification and description of relevant diseases;

2. Estimation of the reported incidence (morbidity and mortality) for each of the selected diseases;

3. Estimation of the degree of under-reporting inherent in the statistics on incidence of these diseases; and

4. Estimation of the degree to which the environmental problems covered by this study -- as opposed to other causes -- are responsible for the incidence of these diseases.

The data acquired in each of these four steps are described below.

Selection of diseases

The first step of the analysis consisted of identifying those diseases of public health concern in Bangkok that are clearly related to environmental factors. For example, many of the helminths (e.g., roundworm, hookworm) are transmitted through human contact with fecally-contaminated soil, particularly when individuals do not wear shoes. Bacterially related diarrheal diseases are transmitted when an uninfected individual comes in contact with feces containing bacteria excreted by an infected individual. A prominent route for this transmission involves fecal contamination of drinking water. Both helminthiasis and acute diarrhea are diseases influenced strongly by environmental conditions such as the degree to which human waste in Bangkok is treated and/or conveyed away from people, and the quality of the drinking water supply at the tap. On the other hand, influenza, sexually transmitted diseases, and measles are examples of the group of important communicable diseases which we did not select as being environmentally related.

To develop the list of microbiological diseases of concern in this project, we examined available health statistics for Bangkok (described below) from the Bangkok Metropolitan Authority (BMA) and Thailand's National Epidemiology Board (NEB). We reviewed each of the diseases listed by these sources, and assessed which of them were environmentally related. The remaining diseases were dropped from consideration. For each of the diseases of interest, we collected data on its effects, severity (e.g. degree of disability, years of life lost, length of hospital stay, work hours lost) and pathways of transmission. Some of these data were taken from a Thai source (NEB, 1987b), while others were taken from standard medical texts and references listed at the end of this appendix. A brief description of each of the 14 microbiological diseases or disease categories that we have termed environmentally-related is included as Attachment A to this Appendix.

Reported incidence of these diseases

Health statistics for Bangkok were obtained from two major sources: the Bangkok Metropolitan Authority (BMA) and the National Epidemiology Board (NEB). The BMA data consisted of morbidity figures for both inpatients and outpatients in fiscal years 1987, 1988, and 1989 (Tables F.2 and F.3). The inpatient data was broken down by specific disease, whereas the outpatient data was tabulated by bodily system infected, thus making the latter figures much less helpful. BMA's data came from over 300 public and private hospitals and clinics in Bangkok, and incidence was reported separately for Bangkok residents and for non-residents who came to Bangkok to be treated.

Data from the NEB were obtained on morbidity and mortality for the calendar year 1987 only. Although the NEB's tabulation covers the entire country, separate subtotals are available covering the Bangkok metropolitan region. The NEB's figures appear to include cases from a broader range of

Table F.2 Morbidity Among Bangkok Residents from Infectious and Parasitic Diseases -- Inpatients

	1987 Number	1988 Number	1989 Number	Change from 1987 to 1989	Average # cases per year
Environmentally Related Diseases					
Acute diarrhea	6977	8768	6653	-5%	7466
Dengue hemorrhagic fever	9045	3735	3157	-65%	5312
Dysentery (bacillary, amoebic)	605	836	420	-31%	620
Malaria	273	371	216	-21%	287
Enteric fever (typhoid, paratyphoid)	266	278	154	-42%	233
Cholera	275	143	73	-73%	164
Encephalitis	155	182	142	-8%	160
Hepatitis A (a)	51	54	37	-27%	47
Tetanus	47	46	33	-30%	42
Rabies	9	4	99	1000%	37
Acute poliomyelitis	12	44	30	150%	29
Leptospirosis	25	32	23	-8%	27
Typhus and other rickettsioses	11	20	13	18%	15
SUBTOTAL	17751	14513	11050	-38%	14438
Both Environmentally Related and Not					
All other infective and parasitic diseases (b)	7075	5015	4801	-32%	5630
environmentally related : 15% Low estimate	1061	752	720	-32%	845
40% High estimate	2830	2006	1920	-32%	2252
TOTAL Environmentally Related : Low estimate	18812	15265	11770	-37%	15283
High estimate	20581	16519	12971	-37%	16690
Non-Environmentally Related Diseases					
Tuberculosis of respiratory system	1259	1301	1119	-11%	1226
Other tuberculosis, including late effects	155	196	133	-14%	161
Leprosy	17	17	7	-59%	14
Diptheria	70	33	13	-81%	39
Whooping Cough	32	39	41	28%	37
Streptococcal sore throat and scarlet fever	145	134	73	-50%	117
Meningococcal infection	12	22	7	-42%	14
Small pox	-	-	-	-	0
Measles	787	728	171	-78%	562
German measles	143	70	99	-31%	104
Hepatitis (B, nonspecific)	881	929	645	-27%	818
Trachoma	21	32	27	29%	27
Syphilis	251	180	92	-63%	174
SUBTOTAL	3773	3681	2427	-36%	3294

Notes:

Sources: Bangkok Metropolitan Authority compilation of data from public and private hospitals and clinics

(a) Number of Hepatitis A cases = (total # Hepatitis A cases reported by BMA)(percentage of total # NEB reported Hepatitis cases that are Hepatitis A)

(b) includes non-environmentally related infectious & parasitic diseases

Table F.3 Morbidity Among Bangkok Residents -- Outpatients

	1987 Number	1988 Number	1989 Number	Change from 1987 to 1989	Average # cases 1987 to 1989
Infectious and Parasitic Diseases	199163	254129	217892	9%	223728
Neoplasms	60336	60569	51868	-14%	57591
Endocrine, Nutritional, Metabolic Diseases	175523	176369	142091	-19%	164661
Diseases of Blood and Blood-forming Organs	19052	19824	17069	-10%	18648
Mental Disorders	82180	90939	93667	14%	88929
Diseases of the Nervous System	301796	307673	264789	-12%	291419
Diseases of the Circulatory System	191580	209850	175696	-8%	192375
Diseases of the Respiratory System	646579	1028022	611466	-5%	762022
Diseases of the Digestive System	359645	386992	326163	-9%	357600
Diseases of the Genito-urinary Tract	198104	219810	198564	0%	205493
Complications of Pregnancy, Childbirth, Puerperium	170398	154992	82149	-52%	135846
Diseases of Skin and Subcutaneous Tissue	265575	323786	215747	-19%	268369
Diseases of Musculoskeletal System and Connective Tissue	242882	254024	220377	-9%	239094
Congential Abnormalities	8372	8434	7706	-8%	8171
Certain Causes of Perinatal Morbidity and Mortality	7580	4205	2283	-70%	4689
Symptoms and Ill-defined Conditions	258165	325754	202947	-21%	262289
Accidents, Poisoning, Violence	185160	196710	193881	5%	191917
Other Activities	558427	-	-	-100%	
TOTAL	3930517	4022082	3024355	-23%	3472842

Source: Bangkok Metropolitan Authority compilation from public and private hospitals and clinics

hospitals, public and private clinics, and physicians than the BMA figures. It is not clear that the NEB's totals for Bangkok exclude visitors and count Bangkok residents only.

The NEB's figures differ from those from the BMA for several reasons, the separate effects of which we cannot estimate quantitatively. We have chosen to use the BMA figures for our primary risk calculations, primarily because the BMA figures provide us with more years of data and more recent data. Also, the NEB figures are lacking in that several diseases relevant to this study are not included (e.g. helminth infections); this can perhaps be explained by the fact that NEB only records data for "notifiable" diseases. The BMA classification of diseases is also not ideal for our purposes; for example, the various helminth infections we are concerned with are lumped with other diseases under the BMA's "Other infectious & parasitic diseases" heading. Helminthiasis, although not a notifiable disease and generally not life-threatening, is nevertheless a major public health concern because of its great prevalence.

For risk analysis purposes, we have averaged the BMA data for the three years 1987-1989 to obtain an annual estimate of reported incidence. This has been done separately for the inpatient and the outpatient data:

- o For inpatients, statistics are available separately for most of the diseases of interest to us. However, some fraction of the "Other infectious and parasitic diseases" category is also relevant to our concern. Common diseases within this category that we wish to count as environmentally related include those due to helminths and salmonella (in addition to typhoid and paratyphoid). Common diseases within this category not counted include many varieties of food poisoning, chickenpox, herpes, infectious mononucleosis and fungal infections. On balance, we guess that 15 - 40% of the cases of "Other infectious and parasitic diseases" consist of environmentally related diseases.

- o For outpatients, no finer breakdown beyond "Infectious and parasitic diseases" is available. We assume that the same proportion of outpatient "Infectious and parasitic diseases" are environmentally related as was calculated for inpatients.

Inpatient and outpatient cases of the environmentally related diseases are added to provide an estimate of annual reported incidence. These calculations are shown in Table F.4

Table F.4 Estimating the Reported Incidence of Environmentally Related Microbiological Diseases

To calculate total cases (inpatient and outpatient) of infectious and parasitic diseases (I&P) which are environmentally related:

		1987	1988	1989	Average 1987 to 1989
(a) Total inpatient cases of I&P diseases:		28599	23209	18278	23362
(b) Total inpatient cases of I&P diseases, environmentally related (Low estimate):		18812	15265	11770	15282
(c) Total inpatient cases of I&P diseases, environmentally related (High estimate):		20581	16519	12971	16690
Percent of total inpatient cases that are environmentally related:					
(d) Low estimate	(b)/(a)	0.66	0.66	0.64	0.65
(e) High estimate	(c)/(a)	0.72	0.71	0.71	0.71
(f) Total outpatient cases of I&P diseases:		199163	254129	217892	223728
Total outpatient cases of I&P diseases, environmentally related:					
(g) Low estimate	(d)*(f)	131006	167145	140310	146352
(h) High estimate	(e)*(f)	143326	180876	154627	159836
TOTAL CASES OF I&P DISEASES, ENVIRONMENTALLY RELATED:					
Low estimate	(b)+(g)	149818	182410	152080	161635
High estimate	(c)+(h)	163907	197395	167598	176527

RANGE across three years: 149,818 to 197,395 cases/year

AVERAGE: About 170,000 cases/year

Under-reporting

It is widely known and accepted that health statistics compiled through reports by hospitals, clinics and physicians substantially understate the number of cases of disease that actually occur. The degree of under-reporting varies with the severity of the disease. Individuals with diseases that have severe symptoms will usually seek medical attention and be counted in the statistics. Individuals with less serious diseases and less severe symptoms may just let their illnesses run their course or treat themselves with medicine purchased at a pharmacy.

It is not possible to estimate precisely the degree of under-reporting associated with the reported disease incidence in Table F.4. Some relevant information is:

o Mansoor (1990) investigated the Klong Toey slum in Bangkok, and compared patient visit records at the clinics serving the area with the results of a survey of residents about how often they had been sick with three diseases. Mansoor concluded that individuals sought medical attention at the following rates for the three diseases: diarrhea 12%, upper respiratory infections 18%, and scabies 4%. The results for scabies were insignificant because of a low sample size; Mansoor concluded that 10 - 20% of the individuals who are sick will seek medical attention in such a way as to enter the statistical count. This figure agrees roughly with the estimate by a BMA physician serving the area that about 1/5 of those sick will report to a clinic or doctor.

o While studies of disease reporting in the U.S. are of only tenuous relevance to Thailand, the results may nevertheless be interesting. The U.S. EPA in its analysis supporting regulations governing microbiological contamination of drinking water estimated that the actual incidence of waterborne disease exceeds that reported by a factor of 4 to 25 (U.S. EPA, 1987d). The U.S. Centers for Disease Control has estimated that perhaps 25 times as many cases of food-borne illness occur as are reported (CDC, 1982).

We have adopted Mansoor's estimate that 10 - 20% of those ill will seek medical attention. It appears reasonable to apply Mansoor's findings for the relatively non-severe diseases he considered to our set of diseases also. The great majority of the reported cases to which we will apply this factor are similarly for relatively non-severe diseases.

To account for under-reporting, we have multiplied the reported incidence of environmentally-related microbiological diseases in Bangkok by a factor of 5 - 10.

Apportioning the incidence of these diseases among causes

In this step of the process, we seek to estimate the proportion of the incidence of environmentally-related diseases that is attributable to environmental problems within the scope of this project. It is not possible to do this in any rigorous quantitative sense.

Although these diseases each have a predominant pathway by which they are transmitted (e.g., fecal-oral, vector-related), wide variations are possible in the details of the pathways responsible for each case of disease. For example, shigella cause bacillary dysentery through the fecal-oral route. One case may arise when the feces of an infected individual are routed directly to a klong without treatment. A water supply pipe traversing the klong has corroded and allows the fecally-contaminated water to infiltrate, and an individual contracts shigellosis when he subsequently drinks the contaminated water. This case can clearly be attributed to environmental problems -- lack of human waste conveyance and treatment, and contaminated drinking water. Another case may arise, though, when feces from an infected individual are left on a toilet seat, and a child handles the toilet seat and then a kitchen counter on which food is being prepared. The shigella then multiply in the food, infecting other members of the family when the food is eaten. This case involves none of the environmental problems within the scope of this project.

In short, it is not possible to have sufficient data to determine the specific pathways by which most cases of microbiological disease arise. As described at the beginning of this Appendix, in most instances multiple factors -- both environmental and non-environmental -- probably contribute to disease transmission and persistence.

Some qualitative judgments can be offered, however. Feachem (1981) reviewed the characteristics of each disease (e.g., typical pathways, minimum infective dose, ability of the agent responsible to survive outside the host) in order to determine the likely contribution of specific factors to its transmission and persistence. Feachem ranked each of several alternate approaches according to its effectiveness in preventing and/or controlling each disease. The list of control approaches includes improvements in: water quality, water availability, excreta disposal, excreta treatment, personal and domestic cleanliness, drainage and sullage disposal, and food hygiene. For each illness (or, in some cases, type of illness), alternate control approaches were ranked as being of no (0), little (1), moderate

(2), or great (3) importance as elements of disease control strategies. Feachem's chart summarizing his judgments is shown as Table F.5. We regard several of Feachem's control approaches as being environmentally related: water quality, water availability, excreta disposal, excreta treatment, and drainage and sullage disposal. Others of his control approaches -- personal and domestic cleanliness, and food hygiene -- are not environmentally related. Feachem's chart provides both a rough guide as to how much responsibility for incidence of these microbiological diseases in Bangkok can be assigned to environmental problems, and a useful foundation from which to formulate risk management strategies for the diseases.

III. Discussion of Findings

Environmentally related microbiological diseases resulted in about 12,000 - 21,000 reported inpatient cases annually over 1987 - 1989 among Bangkok residents. Acute diarrhea and dengue were by far the most prevalent of the microbiological diseases, with an average of 7466 and 5312 cases per year, respectively. The number of inpatients per disease then fell off sharply; dysentery, amoebic and bacillary combined, came next with 620 cases. The following four diseases were grouped closely together: malaria, 287 cases; enteric fever (typhoid, paratyphoid), 233 cases; cholera, 164 cases; and encephalitis, 160 cases. The remaining diseases had an average of fewer than 100 cases per year: hepatitis A, tetanus, rabies, acute poliomyelitis, leptospirosis, and typhus and other rickettsioses. Among the inpatients with "other infective and parasitic diseases" might have been about 800 - 2300 with the environmentally related diseases of helminthiasis and salmonella food poisoning.

The great majority of these cases were represented by relatively non-severe diseases (severity scores of 6-8 in the severity ranking presented in Table H.1). Only four of the diseases scored higher than 8: encephalitis (16), tetanus (11), poliomyelitis (10), and rabies (10). Of these four more severe diseases, encephalitis is the most prevalent, but accounting for only about 1% of the inpatients. For the purposes of eventual comparative ranking of Bangkok's environmental health problems, we will thus characterize the microbiological diseases as being of generally quite low severity.

Between 1987 and 1989, the number of inpatients estimated to have environmentally related microbiological diseases declined by 37%[1]. Although this appears to represent a real improvement in public health, we cannot be sure. The great majority of the decline is accounted for by dengue

[1]. Over this same period, Bangkok's population increased by about 7%.

Table F.5 Importance of Alternate Control Measures for Environmentally Related Microbiological Diseases

Diseases and Agents	Water Quality	Water Availability	Excreta Disposal	Excreta Treatment	Personal/domestic Cleanliness	Drainage/sullage Disposal	Food Hygiene
Diarrheal diseases and enteric fevers							
Viral agents (e.g. enteroviruses, rotavirus)	2	3	2	1	3	0	2
Bacterial agents (e.g. Shigella, Salmonella)	3	3	2	1	3	0	3
Protazoal agents (e.g. Giardia lamblia)	1	3	2	1	3	0	2
Poliomyelitis & Hepatitis A	1	3	2	1	3	0	1
Worms w/ no intermediate host							
Ascaris	0	1	3	2	1	1	2
Trichuris	0	1	3	2	1	1	2
Ancylostoma, Necator (Hookworm)	0	1	3	2	1	0	1
Beef & pork tapeworms	0	0	3	3	0	0	3
Worms w/ intermediate aquatic stages							
Schistosoma	1	1	3	2	1	0	0
Skin, eye, & louse-borne infections	0	3	0	0	3	0	0
Infections spread by water-related vectors							
Malaria	0	0	0	0	0	1	0
Dengue	0	0	0	0	0	1	0
Bancroftian filariasis	0	0	3	0	0	3	0
Encephalitis	0	0	0	0	0	1	0
Rabies (a)	0	0	0	0	0	0	0
Tetanus (a)	0	0	0	0	0	0	0
Leptospirosis	1	0	0	0	0	0	0
Typhus	0	1	0	0	1	0	0

(a) Related somewhat to solid waste disposal

Source: adapted from Feachem (1981)

KEY: 3 of great importance in controlling disease
2 of moderate importance
1 of minor importance
0 of no importance

fever, the incidence of which fluctuates widely with whether environmental conditions (e.g., floods) are favorable to mosquitos. The decline might be explained by an unusually high incidence of dengue in 1987.

The data available to us on disease prevalence for hospital outpatients in Bangkok was not disaggregated to the extent we wished. Assuming that the relative prevalence of different infectious and parasitic diseases is the same among outpatients as among inpatients, we estimate about 150,000 reported outpatient cases of environmentally related diseases annually. Adding outpatients and inpatients, we estimate about 170,000 reported cases of these diseases annually. Assuming that only 10 - 20% of the actual cases of these diseases are reported, **we estimate 850,000 - 1,700,000 cases per year of environmentally related microbiological disease among Bangkok's population.**

These cases of disease can not, however, be attributed solely to environmental problems within the scope of this study. **Non-environmental causes, such as inadequate personal hygiene and improper food preparation, are as or more important than environmental causes for these diseases.** In eventually comparing the health risks posed by Bangkok's different environmental problems, we must not count all of the incidence of environmentally related microbiological diseases as environmentally caused. Other conclusions (based largely on Feachem [1981]) regarding factors responsible for these diseases are:

o The most important environmental factors are clearly: (1) Providing sufficient, reliable water to the population to support washing and other sanitary practices; and (2) Providing means -- toilets and sanitary sewers -- to get human excrement away from the immediate human environment. They are highly important in reducing the incidence of a broad range of microbiological diseases.

o Other environmental factors are less important. The quality of the water supplied is less important than is having a reliable water supply in the first place. Treating human excreta properly (e.g., in sewage treatment plants) is much less important that conveying it away from people. Although water quality and sewage treatment are not vital in controlling most types of microbiological disease, they are of significant value in reducing, respectively, the traditional water-borne bacterial diseases (cholera, typhoid, shigella) and some helminth-related diseases.

o More complete collection and better disposal of household refuse is another environmental factor that can be of value in reducing incidence of several vector-related diseases (those relating

to rats and insects such as flies and cockroaches that can feed on garbage). We estimate this value as rather low.

o Better drainage and reduced flooding can marginally reduce the incidence of mosquito-related diseases such as malaria, dengue and bancroftian filariasis.

IV. Limitations

There are substantial uncertainties inherent in our analysis of the risks associated with environmentally-related microbiological diseases. For most other environmental problems analyzed in this study, the major uncertainties involve the appropriateness of ambient monitoring data for the pollutants involved and the validity of dose-response relationships assumed for the pollutants. For microbiological diseases, though, our risk analysis approach has been different and the resulting uncertainties are different. For this problem, we collected information on incidence of adverse health effects and speculated about how much of this incidence might be attributed to the causes of interest. Major uncertainties arise at four points in this approach:

1. In estimating the actual, as opposed to reported, incidence of diseases. It is widely believed that only a fraction of those contracting a disease, especially one that does not involve terribly severe symptoms, will seek medical treatment for it in a way that will cause a case of the disease to be counted in the official health statistics. Estimates of the degree of under-reporting that results are not precise. We assumed that only 5 - 10% of the cases of the relatively non-severe diseases covered here would be reported. This assumption could easily be inaccurate by a factor of two higher or lower.

2. In determining which diseases to focus on as "environmentally related and caused by microbiological agents". We chose 14 diseases or groups of diseases, and gathered incidence data on them. Reviewers of this paper might well choose a larger or smaller set of diseases to focus on. One might, for example, disagree with our characterization of rabies as being environmentally related. We reason that open dumping of municipal waste and uncollected garbage are environmental problems within the scope of this project, and that they increase Bangkok's population of potentially rabid rats, dogs and other animals. Others may contend that the incidence of rabies is most related to the fraction of urban animals that are rabid, and that this depends on non-environmental factors associated with the extent to which rural animals make their way into urban areas.

3. In estimating the fraction of outpatients reported as having infectious and parasitic diseases that actually had "environmentally related" infectious and parasitic diseases. The data we acquired for outpatients was not sufficiently disaggregated by disease to meet our objectives. We assumed that the distribution of specific infectious and parasitic diseases among outpatients resembled that among inpatients. This is probably inaccurate; those with less severe diseases probably comprise a larger fraction of outpatients than of inpatients. If so, more outpatients with infectious and parasitic diseases will have environmentally-related diseases than inpatients, and our assumption leads to an underestimate of the actual prevalence of these diseases.

4. In apportioning the incidence of environmentally related diseases among environmental and other causes. There is so much uncertainty in doing this that we do not even attempt it quantitatively. We prefer to leave our conclusions here in qualitative terms: that environmental problems covered in this project are important causative factors in the estimated 850,000 to 1,700,000 annual cases of microbiological diseases, but that non-environmental factors are probably equally or more important.

Appendix F, Attachment A. Description of Environmentally-Related Microbiological Diseases

1. Acute Diarrhea

Diarrhea is symptomized by loose, watery stools and may also be accompanied by fever, appetite loss, and vomiting. Those can lead to dehydration and electrolyte loss.

Many pathogens can cause diarrhea in humans; of these, the ones most likely to infect Bangkok residents are: *E. coli*, *Salmonella* species (other than the *Salmonella* species causing typhoid and paratyphoid), *Vibrio* species (other than that causing cholera), some Enteroviruses, Rotavirus, and *Giardia lamblia*.

All of these diarrhea-causing pathogens are transmitted primarily via the fecal-oral route. The Enteroviruses, Rotavirus, and *Giardia lamblia* all have a relatively low infective doses and are immediately infective once excreted, thus, their main transmission pathway is direct, from person to person. Insufficient waste treatment contributes less to transmission than do poor personal hygiene, improper/no use of toilets/latrines, and inadequate water supply that prevents washing. The other diarrhea-causing pathogens, all bacteria, have much higher infective doses. However, they also are more persistent in the environment and can multiply outside of the human host on a suitable substrate, e.g. food, which is the most common for the Salmonella species. For the Salmonella species, other mammals can also serve as reservoirs, so, for example, the infection can be spread from a human to a pig (pigs will eat human feces if they are allowed to do so) and then back to a human via direct contact with the feces or via contaminated water, etc. An additional pathway, mainly for the bacteria, is through produce grown in fields fertilized with untreated human or animal waste.

While the proportion of cases resulting in fatality is much lower for acute diarrhea than for many other diseases, it is nevertheless one of the most prevalent illnesses in Bangkok. The disease is of greatest concern in the very young and the very old; diarrhea is one of the leading causes of infant mortality in Thailand. It can also have less readily measurable health effects. Frequent and/or extended attacks may promote malnutrition, which in turn can hinder physical development and lower resistance to other illnesses.

2. Dengue, dengue hemorrhagic fever

A general infection is characterized by flu-like symptoms. Cerebral and pulmonary hemorrhaging may cause death in patients with dengue.

Dengue is transmitted by a vector, the *Aedes aegypti* mosquito. Because this species of mosquito prefers to breed in relatively clean water, the transmission of this disease is not directly related to environmental pollution. Breeding areas might include water stored in uncovered pots or cisterns. Stagnant pools created by blocked drains or by solid waste disposed in wastewater and sullage canals are less likely to be of interest to the *Aedes aegypti*; however, it is possible that this species will adapt to and accept polluted water as breeding ground.

Dengue has the highest crude mortality rate (NEB) in Bangkok of any of the diseases discussed in this appendix. Morbidity data alone places dengue first, according to NEB statistics, and second only to diarrhea, according to the BMA. Major epidemics of dengue break out in Thailand every few years, but the disease has a strong endemic presence in the country.

3. Dysentery

Dysentery, similar to acute diarrhea in its symptoms, except with abdominal cramping and the presence of bloody stools, is of two types: one caused by the bacterial genus *Shigella* and the other brought on by the protozoan *Entamoeba histolytica*. These pathogens cause Shigellosis, or bacillary dysentery, and Amoebiasis, or amoebic dysentery, respectively. From the colon, the pathogen causing amoebiasis can localize in other organs, forming abscesses on the liver, lung, or brain, for example. A patient who coughs up brownish material and exhibits the other symptoms mentioned most likely has a lung abscess associated with amoebic dysentery.

Although both types of dysentery are transmitted to humans by the fecal-oral pathway, the unique characteristics of bacteria and protozoa make their environmental transmission media differ. *Ent. histolytica* is similar to the diarrheal viruses and Giardia described above in that it cannot survive long outside the host; however, it is immediately infective in excreted matter and requires only a relatively small amount to infect. For these reasons, the primary transmission pathway is direct and person-to-person. Several studies have discovered "family clustering of infection and intra-familial transmission." While a direct transmission route seems to be the most prevalent in areas with endemic infection, especially in areas of overcrowding and inadequate waste disposal, sewage-contaminated drinking water and produce contaminated by untreated human waste used as fertilizer represent two other transmission media. Bacillary dysentery is also transmitted via the direct fecal-oral path. For example, latrine/toilet seats or door handles may become contaminated with infected feces and then a subsequent user may transfer it to his mouth. Indiscriminate excretion around the house by infants and small children can lead to infection of other family members. Contaminated water can also serve as a transmission medium. The human carrier who handles food can spread the disease. This pathway is more important for *Shigella* spp. than for *Ent. histolytica*, because *Shigella* spp. are capable of multiplying outside of the host, most often on food. Control of amoebic and bacillary dysentery requires universal use of sanitary human waste disposal facilities; this requires personal hygiene education. Proper waste treatment will have some effect over the long run.

For both types of dysentery, infection does not necessarily produce symptoms. In fact, up to 80 percent of those infected with *Ent. histolytica* may be symptomless carriers. For those who do show symptoms and seek medical help, the average hospital stay is about 2 weeks for bacillary dysentery and about 1.5 weeks for amoebic dysentery. (NEB review, p.85) Children are highly susceptible to dysentery. As with diarrhea, frequent or sustained bouts of infection can lead to malnutrition and can affect development of the brain and rest of the body.

4. Cholera

The cholera patient experiences diarrhea accompanied by a dry mouth, anuria (inability to urinate), and a weak pulse. Symptoms may also include vomiting and a body temperature lower than normal.

Vibrio cholera is the bacterium responsible for causing cholera. It is most commonly thought to be a waterborne disease; however, there exists some controversy as to whether this is the most important transmission path. Some experts agree with the conventional notion that cholera is primarily water-borne. Others hold that in lower income communities in developing countries, water is not one of the major transmission pathways. (see Feachem for more) Here again, the primary transmission route is from person to person, fecal-oral. This can be direct, from person to person, or by food or water contaminated by fecal material.

Low gastric acidity may affect a person's susceptibility to *V. cholera*. Hypochlorhydria (low gastric acidity), brought on by malnutrition, gastric surgery, or other causes, can allow person to infected by a much smaller dose.

5. Malaria

Malaria's primary symptoms include: fever which spikes at regular intervals, usually between 48 and 72 hours, with chills, body aches, headaches, and sometimes delirium. In addition, the spleen, liver, and kidneys become enlarged. In malignant falciparum malaria, parasitized red blood cells can clog the vessels in the brain. This type of malaria is often lethal; it can lead to delirium progressing to coma, renal failure, or liver failure.

The *Anopheles* species of mosquito are carriers of the protozoan *Plasmodium*, which cause malaria. If a human is bitten by a mosquito carrying this disease, he will become infected. As with the *Aedes aegypti*, which transmits dengue, this genus prefers to breed in unpolluted, albeit stagnant, water; thus, malaria is not directly environmentally related. The possibility does remain that the *Anopheles* will adapt locally to breed in unclean water. Thus, solid waste- or sewage-blocked storm drains could become acceptable breeding grounds for these mosquitoes, which would exacerbate the current problem.

Malaria affects all age groups. Prevention consists of either destroying breeding areas or avoiding contact with mosquitoes, either by securing screens in windows and doors, especially at night when mosquitoes come out to feed, or by sleeping under netting, or both. Thus, malaria is not highly preventable; however, once detected, it is highly treatable. The average hospital stay for malaria patients is 9 days. (NEB)

6. Enteric Fever

Typical symptoms of the enteric fevers include nausea, abdominal pain, vomiting, and fever. Sometimes a light rash will appear on the abdomen.

Both typhoid, or typhoid fever, and paratyphoid, are enteric fevers. Each is caused by a species of *Salmonella*, *S. typhi* and *S. paratyphi*, respectively. Unlike the other Salmonelloses, the enteric fevers have no non-human reservoirs, so transmission takes place from person to person, by the fecal-oral route. Transmission can be direct person to person or can occur through ingestion of fecally contaminated water or food. A fairly high dose is required to infect, but as *S. typhi* and *paratyphi* can multiply outside of the host, most often on food, generation of an infective amount can readily occur. Like amoebic dysentery, typhoid can be spread by asymptomatic carriers as well as patients.

Typhoid, unlike many of the other diseases described here, is vaccine-preventable. While not as prevalent in Bangkok as some other microbiological diseases, the facts that immunity can develop and that a symptomless carrier state does exist, allow for epidemic prevalence.

- 234

7. Hepatitis A

Fever, nausea, abdominal pains, and jaundice all signify a case of hepatitis A, or infectious hepatitis.

Hepatitis A is caused by a virus which is spread by the fecal-oral route, primarily through direct person to person contact. Prevalence is generally high in conditions of overcrowding and poor sanitation. One can develop immunity to hepatitis A through repeated exposure and low-grade infection. Acquired immunity should permit development of a vaccine; however, a successful one has not yet been created. In the absence of an effective vaccine, hepatitis A prevention and control rely on high levels of personal hygiene and proper human waste disposal.

8. Encephalitis

Encephalitis, an inflammatory disease affecting the brain, spinal cord, and meninges, is characterized by fever, stupor, tremors, and can occasionally lead to convulsions or coma.

Another vector-related disease, encephalitis is caused by a virus transmitted by mosquitoes. In the United States the most common vectors of encephalitis are the *Culex pipiens* and other *Culex* species. If the same can be assumed for Thailand, then the maintenance of encephalitis in Bangkok is waste-related, unlike the mosquito-transmitted diseases previously described, for the *Culex* mosquitoes seek out polluted water as their breeding ground. For example, sewage-polluted canals and solid-waste-blocked storm drains would provide favorable habitat for these mosquitoes.

Encephalitis has a fairly high case-fatality ratio with a nearly four percent mortality rate. (NEB 1987 data) There is a vaccine against encephalitis.

9. Tetanus

Puncture wounds, other minor wounds, and burns provide pathways through which soil, dust, or feces contaminated with the bacterium *Clostridium tetani* can infect humans and other mammals. Infection results in contractions of the muscles of the neck and, occasionally, trunk. Sometimes, the area of infection will become stiff also.

The habit of not wearing shoes, in conjunction with uncollected solid waste, which might contain sharp objects, makes some Bangkok residents particularly susceptible to tetanus infection. Gleaners who work the landfills for resalable items also run the risk of becoming infected. A human can also contract tetanus as a result of a dog or rat bite.

Tetanus, although it has a low morbidity rate, has a high case-fatality ratio. Ten percent of cases are fatal; twenty percent of tetanus cases in infants result in death. (NEB 1987 data)

10. Leptospirosis

An average case of leptospirosis can produce the following symptoms: spikes in body temperature, headache, nausea, vomiting, and muscle aches. A more severe case of leptospirosis is called Weil's disease, which is characterized by jaundice, bleeding, and renal failure.

Bacteria which infect both man and other animals, the *Leptospira* species are not very persistent in the environment, but they are immediately infective and require relatively small doses to infect. Urine of rats, dogs, or other mammals which contains leptospires can contaminate water. Contact with this water, either through ingestion or bathing, can infect humans. Direct contact with infected animals can also cause the disease in humans.

Although relatively rare, a case of leptospirosis can be quite serious and ends in death for over 5 percent of its victims. (NEB 1987 data)

11. Rabies

Rabies affects the central nervous system, causing headache and fever at its onset and resulting in slight or partial to complete paralysis. Mortality is due to respiratory paralysis.

Humans become infected with rabies from the bite of a dog, bat, or other animal that has the Rabies virus. In the United States, rats and mice which live in urban areas are generally not rabid (UPM, p.55) Solid waste collected in landfills and uncollected solid waste near houses may provide food and suitable nesting areas for wild animals, which in turn could infect domestic animals. Not only those who live on or near the landfills would be at risk.

The number of cases of rabies each year is not high; however, the case-fatality ratio is extremely significant: for 100 percent of those who contracted rabies in Bangkok in 1987, the disease was fatal. (NEB 1987 data) For those who seek medical attention immediately after being bitten, the required treatment, undertaken when the animal tests positive for rabies or when it cannot be found, is long and painful.

12. Typhus

Fever, chills, headache, body aches, and a generalized rash which develops into fixed lesions characterize typhus. In advanced cases, the patient may exhibit gangrene of the extremities, delirium, or stupor leading to coma.

Lice, fleas, and mites are the primary vector carriers of the Rickettsia which cause typhus. When humans scratch the bites, the arthropods' excreted Rickettsia is worked into the skin. Rats can also host Rickettsia-infected fleas and in this way act as a non-human reservoir which sustains the infected flea population. Louse-borne infection is responsible for most epidemic outbreaks of typhus. This type can be transferred directly for person to person in crowded and unsanitary settings. Mites transmit a type of Rickettsia which causes what is referred to a scrub typhus. It is not evident what type of typhus predominates in Bangkok. Any situation in which humans live in close proximity to rats will promote the spread of typhus. Household garbage provides both food and habitat for rats and mice; uncollected solid waste and disposal of solid waste at unsanitary landfills can both be expected to increase the prevalence of rats and mice.

Typhus infects a very small portion of Bangkok's population; the average number of cases for the years 1987-89 was 15.

13. Poliomyelitis

Symptoms characteristic of poliomyelitis are fever, headache, gastrointestinal upset, and neck and back stiffness. Acute cases result in paralysis.

Poliomyelitis is caused by an enterovirus, one of three polioviruses. The fecal-oral and oral-oral routes are the main transmission pathways. Inadequate personal and domestic hygiene (sometimes due to unreliable water supply) and improper waste disposal combine to increase the spread of polio.

Most cases of polio are seen in children, who have not yet built up an immunity to the poliovirus. If measures are undertaken to control the spread of the disease, they may succeed in reducing prevalence, but the cases which do occur run the risk of being much more serious; when exposure to the disease is limited, so are chances of acquiring resistance, which is built up through a series of sub-clinical infections. Poliomyelitis, although it puts those who do contract it into the hospital for an average of one month (NEB, p.85), is highly preventable; it has been nearly eliminated in the U.S.

14. Helminthiasis

The helminths are of three basic types: nematodes (roundworms), cestodes (tapeworms), and trematodes (flukes). ~~These re~~sponsible for infections in Southeast Asia include: *Ascaris lumbricoides* (roundworm), *Necator americanus* and *Ancylostoma duodenale* (hookworm), *Schistosoma* species (liver fluke), *Taenia saginata* and *Taenia solium*, (beef and pork tapeworms, respectively), *Trichuris trichura* (whipworm), and *Wucheria bancrofti* (Bancroftian filariasis). The helminths do not reproduce inside the host, so repeated inoculation is necessary to produce a heavy infection. On the whole, however, the helminths are extremely persistent in the environment. Many are resistant to conventional sewage treatment. Infection is widespread in Bangkok and heavy infection appears with moderate frequency.

Both *Ascaris* and *Trichuris* are transmitted by the fecal-oral pathway. Fecal matter and fecally contaminated soil are the two major sources; infection is not water related. The eating of raw vegetables which have been grown in fields fertilized with raw sewage represents another possible source. There may be no clinical effects in light infestations; heavier cases may induce abdominal pain, vomiting, restlessness, and gastrointestinal disturbance. The health effects of ascariasis and trichuriasis range from malnutrition and its consequences to perforation or complete obstruction of the intestine in the case of ascariasis.

Ancylostoma duodenale and *Necator americanus* are the two species of hookworm that infect humans. Both are transmitted by the fecal-cutaneous route. Eggs are passed in the feces; then, if a suitable substrate is found (warm, moist, shaded soil or other organic matter), they go through two molts and in the third stage larval form they can penetrate the skin of humans. Improper waste disposal or inadequate waste disposal facilities, indiscriminate defecation, and the habit of not wearing shoes combine to increase the risk of infection. For the *A. duodenale* the fecal-oral route is also significant, particularly in areas where crops are fertilized with raw sewage and are eaten raw. Light hookworm infections produce few clinical effects; however, the predominant health effect of a more severe infection is iron deficiency anemia, as hookworms feed on the host's blood. Malnutrition represents another possible health effect.

Schistosomiasis, caused by the *Schistosoma* species, differs from many of the other helminths in that it requires an intermediate host before it can infect humans. Egg-contaminated feces, or in the case of one species, urine, reaches water inhabited by the suitable host snail. Release of untreated sewage into canals, indiscriminate defecation, and urination into or while in surface water are some of the possible water contamination pathways. The eggs hatch and the first larval form, once finding a host, passes through the next few developmental stages. The larvae leave the host in final stage and, upon coming into contact with a human, penetrate the skin. Heavy infections are characterized by abdominal swelling due to the enlargement of the liver and spleen. Any activity that will bring humans in contact with contaminated water that is inhabited by snails will encourage infection. Although fairly uncommon, drinking contaminated water can allow larval penetration of the oral mucosae.

The beef and pork tapeworms, *Taenia saginata* and *Taenia solium*, are transmitted through ingestion of raw or insufficiently cooked beef or pork containing the tapeworm larvae. Intermediate hosts, pigs and cattle are themselves infected by ingesting contaminated human waste, either by directly eating human feces or by grazing in pastures fertilized with untreated nightsoil. These tapeworms can present a health problem to Bangkokians, if the meat they eat is infective and if food preparation habits do not provide for thorough cooking of meat. Symptoms can range from none to abdominal pain, loss of weight, and digestive disturbance. Here again, the major health threat is malnutrition. A much more serious, albeit rarer, condition occurs when a human ingests the parasite at the egg stage. The egg hatches and the organism can make its way into the blood stream, later to lodge itself and develop into a cystic larva anywhere in the body, in the heart, for example.

Bancroftian filariasis, unlike the other helminthiases discussed here, is a vector-related disease. The *Culex pipiens* mosquito favors polluted water for its breeding grounds; it can often be found in stagnant pools caused by clogged storm drains or around pit latrines. The infection is transmitted through the bite of a mosquito carrying *Wuchereria bancrofti*. The results of chronic infection, although rarely life-threatening, are quite severe. Many infected show no clinical symptoms, but in acute cases, lymphatic swelling can lead to obstruction, which in turn can cause elephantiasis of the legs or groin area are the common symptoms.

For all types of helminthiasis, the primary threat to public health is malnutrition, due to competition between the host and parasites for nutrients. Malnutrition can in turn lead to reduced immunity to other infections, growth retardation, reduced working capacity, etc. A survey performed in 1982 discovered that an average of 54.6 percent of the national population was infected with one or more types of helminth. (NEB, p.93) Although minor infection may not produce any visible symptoms, these infections are important because the infected then act as carriers.

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Appendix G. Summary of Techniques Used by the U.S. EPA in Human Health Risk Assessment

I. The Risk Assessment Process

Human health risk assessment is the process of estimating the likelihood or probability that a given chemical exposure or series of exposures may damage the health of exposed individuals. The National Academy of Sciences (1983) divides the risk assessment process into four discrete components:

- Hazard identification
- Dose-response assessment
- Exposure assessment
- Risk characterization

This appendix describes the information and methods typically used by U.S. EPA risk assessors to accomplish each of these steps.

Hazard Identification

The hazard identification component of risk assessment consists of gathering and evaluating all relevant data bearing on whether or not a chemical substance poses a specific hazard. Hazard identification determines whether it is scientifically appropriate to infer that health effects observed under one set of conditions (e.g., in experimental animals) will likely occur in other settings (e.g., in humans). It is a qualitative determination based on judgments about such factors as the type of health effect produced, the conditions of exposure and the metabolic processes within the body that govern chemical behavior. Generally, hazard identification focuses separately on carcinogenic and non-carcinogenic effects.

For a potentially carcinogenic substance, the U.S. Environmental Protection Agency has developed a formal procedure for weighing the toxicological, epidemiological, and other information. Data from studies of the effect of the substance on animals and humans are reviewed. Separate judgments about the degree of evidence of carcinogenicity are made based on the animal and human data. The following categories are used:

- sufficient evidence
- limited evidence
- inadequate evidence
- no data available
- no evidence of carcinogenicity

Human and animal evidence of carcinogenicity is then combined into a single statement about the weight-of-evidence that the substance is a human carcinogen. The scheme includes the following groups:

- Group A - human carcinogen
- Group B - probable human carcinogen
- Group C - possible human carcinogen
- Group D - not classifiable as to human carcinogenicity
- Group E - no evidence of carcinogenicity in humans

The Agency typically treats chemicals classified in categories A and B above as carcinogens, while those that fall in categories D and E are not treated as carcinogens. Chemical substances classified as category C receive varying treatment. EPA's approach to combining human and animal data is presented in Table G.1.

In determining whether a substance causes non-carcinogenic health effects (e.g., mutagenic effects, systemic toxicity), the Agency's hazard identification/weight of evidence determination is not yet formalized and is much more qualitative. Studies assessing the effects of the substance are reviewed. The studies are evaluated based upon the following factors:

- quality of data
- resolution of the studies; (significance of the studies as a function of the number of animals or subjects)
- relevance of route and timing of exposure
- appropriateness of dose selection
- replication of effects

Table G.1. Categorization of Carcinogenicity Based on Animal and Human Data

Human Evidence	Animal Evidence				
	Sufficient	Limited	Inadequate	No Data	Evidence of No Effect
Sufficient	A	A	A	A	A
Limited	B1	B1	B1	B1	B1
Inadequate	B2	C	D	D	D
No Data	B2	C	D	D	E
Evidence of No Effect	B2	C	D	D	E

- number of species examined
- availability of human epidemiologic study data

A subjective summary judgment is then made about whether or not the substance causes the particular non-carcinogenic health effect.

Dose-Response Assessment

Dose-response assessment involves estimating quantitatively the connection between the amount of exposure to a substance and the likelihood of the health effect of concern occurring. Dose-response assessment may be performed separately for multiple different health effects that may be caused by a single substance. One study may examine the potency with which a substance causes cancer; another study may examine the different degree to which it may cause toxic effects of the liver. There are two general approaches to dose-response assessment depending on whether the health effects under investigation involve threshold relationships or not.

Non-Threshold Effects

For non-threshold effects, an attempt is made to extrapolate response data from doses in the experimental range to response estimates in the dose ranges typical of most environmental exposures. Typically, the dosage to which animals have been subjected in experiments are much higher than those to which humans are exposed in the environment. The greatest number of such dose-response extrapolations have been performed in the field of carcinogen risk assessment. Once again, the EPA's Cancer Guidelines give the most detailed instructions on how to perform dose-response assessments (US EPA, 1986d). In general, EPA considers the risk of cancer to be linearly related to dose (US EPA, 1986d). At this time, the Agency does not consider any level of exposure to carcinogens as de minimus or safe (US EPA, 1986d). The Agency also provides guidance on such issues as choice of extrapolation model; how to consider benign tumors; adjustment of dosage from animals to humans, etc.

The slope of the carcinogenic dose-response function in the low dose range is estimated for each chemical through use of a linearized multistage model. Using this model, a cancer potency factor (symbolized as Q_1^*) is derived that can be used in quantitative risk assessment.

The product of Q_1* and the dose of the chemical yields an estimate of the lifetime cancer risk to an individual receiving that dose level for a lifetime. Q_1^* is expressed in terms of risk-per-dose, and thus has units of (mg/kg/day)-1. The linearized multi-stage model gives a plausible upper-bound estimate to the slope of the dose-response curve in the low dose range. In essence, this means that risk estimates using EPA's Q_1*s are not likely to be lower than true risks. EPA notes that a procedure does not yet exist for making the "most likely" or "best" estimates of risk within the range of uncertainty defined by the upper and lower limit estimates.

Threshold Effects

The other major approach to dose-response assessment examines threshold effects. This approach applies primarily to systemic toxicants and non-carcinogenic health effects. Dose-response assessment for substances exhibiting threshold responses involves calculating what is known as an Acceptable Daily Intake (ADI) value. The ADI for a chemical is the daily dose below which significant risk of adverse effects is not expected. More recently, the Agency has preferred to use a new term, the Reference Dose or RfD, to avoid the connotation of acceptability.

Briefly, EPA's approach for calculating an RfD involves taking an appropriate lifetime-adjusted No Observed Effect Level (NOEL), No Observed Adverse Effect Level (NOAEL), Lowest Observed Effect Level (LOEL) or Lowest Observed Adverse Effect Level (LOAEL) -- derived from either laboratory animal and/or human epidemiology data -- and dividing it by the appropriate uncertainty (safety) factors. Safety factors are intended to establish an RfD sufficiently far below an experimentally devised LOAEL that adverse effects are extremely unlikely to be observed for a dose at the RfP level. The more uncertainly there is in translating the experimental conditions under which the LOAEL was observed to conditions of human exposure, the greater the safety factors will be. Typical safety factors used include:

- A factor to prevent sensitive individuals from an effect observed among average individuals
- A factor to provide for extrapolation from animals to humans
- One or more factors to reflect uncertainty in the experimental setting, such as low sample sizes, short term studies, etc.

For most chemicals with only animal toxicological data available, the RfD is set at between 1/10 and 1/1000 of the experimentally observed LOAEL. For a few chemicals with very good human toxicological and epidemiological information, the RfD is set close to the LOAEL. Ideally, for all non-cancer effects, a set of route-specific and effect-specific thresholds will be developed. If information is available for only one route of exposure, this value is used in a route-to-route extrapolation to estimate an appropriate threshold for the other relevant routes of exposure.

If exposure occurs above the RfD level, the Agency currently has no accepted method to determine the probability of an adverse health effect occurring. A dose exceeding an RfD does not necessarily mean that the effect will occur: the probability of the effect occurring increases as the dose increases above the RfD. There is much work being carried out both within and outside of EPA to develop more quantitative approaches to dose-response assessment for non-carcinogens. For some non-carcinogenic health effects and chemicals, epidemiological studies have been used to estimate dose-response relationships.

Exposure Assessment

Evaluating human exposure involves describing the nature and size of the population exposed to a substance and the magnitude and duration of their exposure. The major areas to be evaluated when estimating exposures are (US EPA, 1986e):

- source assessment - a characterization of the sources of contamination
- pathways and fate analysis - a description of how a contaminant may transport from the source to the potentially exposed population
- estimation of environmental concentration - an estimate using monitoring data and/or modelling of contaminant levels around a source
- population analysis - a description of the size, location and habits of potentially exposed human and environmental receptors
- integrated exposure analysis - the calculation of exposure levels and an evaluation of uncertainty

An integrated exposure assessment quantifies the contact of an exposed population to the substance under investigation via all routes of exposure and all pathways from the sources to the exposed individuals.

For risk assessments involving chronic exposure, human exposure (mg/kg/day) is calculated as dose averaged over the body weight (kg) and lifetime (days):

$$\text{Average Daily Exposure} = \frac{\text{Total Dose}}{\text{Body Weight x Lifetime}}$$

The total dose (mg) is a function of the following parameters:

- *Contaminant concentration* represents the concentration of the contaminant in the medium (air, food, drinking water) contacting the body. Typical units are mass/volume (e.g., ug/l or ug/m^3) or mass/mass (e.g., mg/kg).

- *Contact rate* is the rate at which the medium contacts the body (through inhalation, ingestion or dermal contact); typical units are mass/time (e.g., mg/day) or volume/time (e.g., m^3 or l/day). EPA has developed a series of average contact rates for different media. For example, it is typically assumed that the average adult consumes 2 liters of water per day and breathes 20 m^3 of air per day.

- *Exposure duration* is the length of time for contact with the contaminant (e.g, lifetime).

- *Absorption fraction* is the effective portion of total contaminant contacting and entering the body. Entering the body means that the contaminant crosses one of the three exchange membranes: alveolar membrane, gastrointestinal tract, or skin.

Risk Characterization

The final step in the risk assessment process is risk characterization. This step essentially involves putting together in a useful way the information developed in the three previous steps. For non-carcinogens, this step often involves comparing the dose estimates (from the exposure assessment step) to the Reference Dose (from dose-response evaluation step) to estimate the relationship of dose to the threshold level. If the level is not exceeded, we can generally presume there is little likelihood of a health effect. When the RfD is exceeded, there is some likelihood of health effects occurring. For a few chemicals, an epidemiologically derived dose-response relationship may be available that allows quantification of a risk estimate. Again, for non-carcinogens, the use of dose-response estimates is quite controversial and its use has been limited within the Agency at this time.

For carcinogens, risk can be estimated by multiplying the actual human dose by the risk per unit of dose projected from the dose-response modelling:

$$\text{Risk} = \text{Dose} \times Q_1^*$$

Risk may be calculated for both the maximally exposed individual as well as for the aggregate population as a whole.

II. Variations in Risk Assessment Approach

For the contaminants of concern in this project, we attempt to identify the potential adverse health effects that may be associated with chronic exposure to them. We rely primarily on EPA's Office of Research and Development determinations to estimate the effects of each contaminant. We treat carcinogenic and non-carcinogenic compounds differently.

For known and suspected carcinogens, we rely on the EPA's Cancer Assessment Group (CAG) estimates of potency (Q_1^*). The CAG uses a linearized multistage model with zero as the threshold. The CAG Q_1^*-value, which is the plausible upper-bound on the low-dose portion of the curve, is used as the slope of the dose-response curve for carcinogens. For non-carcinogens, we rely on the Agency's Risk Reference Dose (RfD) as the threshold value. The RfD and Q_1^* values have been scientifically peer-reviewed and are accepted throughout the Agency.

Consistent with EPA's final risk assessment guideline for Chemical Mixtures (U. S. EPA 1986f), we will assume additivity of cancer estimates in cases where exposure occurs simultaneously to multiple contaminants. Unless data are available, we do not consider synergistic or antagonistic interactions between contaminants.

Risk estimates are not additive across multiple contaminants for non-carcinogenic health effects, however. An RfD applies for a single health effect for a single pollutant; theoretically, if exposure to two different chemicals resulted in the same health effect at the same rate of exposure, the effects could be additive. However, for most of the chemicals examined here, the health effects upon which the RfDs were calculated represent a range of effects to a number of different target organs. Therefore, if exposure occurs to two different chemicals, each a one-half of their respective RfDs, we cannot add the exposure to say that total exposure equals the RfD. Exposure to a single chemical across multiple routes should be added, however.

In several instances in this project, we follow risk assessment procedures different from the general approaches we have outlined for carcinogenic and non-carcinogenic substances. These variations include:

- For some noncarcinogenic health effects and some substances, epidemiological studies have established a relationship between the incidence of an adverse health effect and the ambient concentration of the substance. (An example is the relationship between airborne particulate matter and various measures of morbidity). In such cases, we use the epidemiological relationship and ambient data in preference to simply comparing the ambient data to the RfD.

- For some pollutants, we have no data on dose-response relationships and/or no ambient data (e.g., microbiological contaminants). In these cases, we attempt to obtain data on the actual incidence of the health effect of concern (e.g., number of disease cases) and speculate about the fraction of such cases attributable to environmental causes.

- As discussed in Appendix E, the risk assessment methods for exposure to lead employ a methodology that differs from the approach used for other contaminants. For a variety of reasons, lead exposure and health effects must be

treated independently. Three important factors account for this special treatment. First, lead health effects are based on non-linear dose-response information. Secondly, because of the nature of the epidemiological data for lead, diseases are disaggregated into those affecting sub-populations of men, women, and children. Finally, the dose response information available is based not directly on an environmental exposure, but rather on a blood lead level. Lead health effects are related to a concentration of lead in the bloodstream, expressed as mean blood level, or PbB. This is in contrast to other contaminants, which have health effects expressed in terms of concentrations in air or amount of contaminant ingested. The health calculations in this report are based on the elevation in blood lead level that results from exposure to lead from various environmental sources. For further discussion of the methods used to estimate risks from exposure to lead, the reader is referred to Appendix E.

III. Presentation of Results

Once the results of the assessments are complete, there are basically two forms in which risks can be presented: risk to the maximum exposed individual (MEI) and risks to the aggregate population as a whole. Nearly all of the risks calculated in this report relate to population risks. One of the key project assumptions is that we are most concerned with the aggregate health of the Bangkok population. We focus less on impacts to particular severely affected populations, for reasons explained in the body of each appendix.

For aggregate population risk, the cancer risk calculation uses exposure estimates for an average member of the exposed population, the Q_1^*, and the size of the exposed population. The calculation is as follows:

Dose (mg/kg/day)	x	potency (Q_1^*) (mg/kg/day)$^{-1}$	x	Population (persons)	=	Number of cases

The results for aggregate populations risk are expressed in terms of number of cases of disease (eg., cancer) contracted over the lifetime of the population. This number can be annualized by dividing by the length of the average lifetime, assumed to be 70 years. For non-carcinogens, we will note the relationship of the dose to the RFD. Results are expressed in terms of percentages of the RFD.

IV. Availability of EPA Risk Assessment Data

A summary of EPA's latest health effects information (hazard evaluation and dose-response information) for several hundred of the most important environmental contaminants is available in the Integrated Risk Information System (US EPA, 1987c). This data base is updated periodically to include the most recent information and is available both on-line and hardcopy. Documents describing elements of the risk assessment process include US EPA's exposure and risk assessment guidelines (US EPA, 1986d; 1986e; 1986f), and National Academy of Sciences (1983).

V. Summary

In the simplest sense, health risks from toxic pollutants are a function of two measurable factors: the intrinsic hazard associated with the pollutant, and the degree of human exposure to that pollutant. To cause a risk, a chemical has to be both toxic and present in the environment at a level that could cause a health threat. (For carcinogens, any level of exposure is generally considered to pose some degree of risk; for health effects other than cancer, it is generally held that chemicals pose health threats only above some finite level of exposure, or threshold.) Risk assessment interprets the evidence on these two points, and then develops a quantitative estimate of the size of the risk involved. In a formal sense, risk assessment involves four discrete steps:

1. *Hazard identification*: This step weighs the available health evidence on a pollutant (from animal studies in the laboratory or from epidemiological studies) to determine if there is enough evidence to link a chemical with a certain health effect.

2. *Dose-related assessment*: The next step is to develop a potency figure for the pollutant, or an estimate of the strength with which it produces the effect in question as a function of intake or exposure. This potency varies widely across chemicals, health effects, and pathways of exposure.

3. *Exposure assessment*: This step identifies the particular population exposed to concentrations of a pollutant (such as in the air or drinking water), and then estimates or directly measures the degree of their exposure.

4. *Calculation of risk*: On one level, this is a simple multiplication of exposure and potency. Also important is the presentation of the information. The final assessment should display all relevant information pertaining to the decision at hand, including such factors as the nature and weight of evidence for each step of the process, the estimated uncertainty of the component parts and of the final result, the distribution of risk across various sectors of the population, the assumptions used in filling in gaps in the data, and so forth.

The typical outputs of the risk assessment process are estimated risks to individuals and risks to populations:

1. Risk to an individual is defined as the increased probability that an individual exposed to one or more chemicals will experience a particular adverse health effect during the course of his or her lifetime. It is important to realize that the risk estimated for a particular type of exposure is the incremental risk beyond that which a person faces from exposure to other environmental or hereditary causes of disease, sometimes referred to as the background rate of disease. Two types of individual risks may be estimated: (1) average individual risk, for the typical individual; and (2) risk to the most-exposed individual (MEI), who may be particularly close to a source or is highly exposed for some other reason.

2. Risk to the population is the expected increased incidence (number of cases), above the background rate, of an adverse health effect in an exposed population.

These outputs of the risk assessment process provide quantitative estimates of the comparative impacts of different toxic chemicals, sources or pathways. They can be used to identify the most serious human health problems.

Appendix H. Severity Index for Different Health Effects

In addition to considering the number of health effects caused by each environmental problem, we must also consider the severity of these effects. Several schemes have been employed by others in comparing the severity of different diseases. Some approaches base a judgment about severity on an estimate of the relative economic cost associated with a case of each disease. Other schemes have been based upon opinion polls asking the public which diseases they would least like to contract. Our approach here will be to adapt a severity scheme developed by Thailand's National Epidemiology Board (NEB).

In a 1987 report, the NEB assessed the importance to public health of the leading diseases in Thailand. Importance was estimated through use of a scoring procedure that combined data on the incidence and the severity of the diseases. We have adopted the severity portion of the scoring procedure. In the NEB's methods, the diseases were each rated for five aspects of severity:

o Potential for disability. This reflects the degree to which an individual who survives the disease will have a long-term disability.

o Case-fatality ratio. This reflects the fraction of cases of the disease that are fatal.

o Average hospital stay. This reflects the average duration of hospitalization for Thai patients with the disease.

o Preventability. This reflects the degree to which the disease is preventable, through vaccination or otherwise.

o Treatability. This reflects the degree to which the disease typically responds to medical treatment.

The NEB collected data on these five factors for the common diseases in Thailand, and assigned numerical ratings for each factor. The first three factors were scored on a scale of 1 - 5, while the last two factors were scored from 1 - 3. Higher scores mean greater severity.

We supplemented the NEB's list of diseases by gathering data on several more that are relevant in this project, and scoring them using the NEB's procedure. The resulting severity ranking is shown in Table H.1. Several of the health effects we project from environmental pollution in Bangkok are among the most severe diseases -- stroke, cancer, and coronary heart diseases. The bulk of the environmentally-related health effects, such as most of the microbiological diseases and minor respiratory ailments, are among the least severe diseases.

The reader should note that these severity scores are not intended to provide a cardinal measure of severity. We do not mean to imply that a case of cancer, with a score of 17, is about three times as severe as a case of acute diarrhea, with a score of 6. The scores suggest only generally whether a health effect is severe, moderate, or relatively mild.

TABLE H.1 SEVERITY RANKING FOR VARIOUS DISEASES

Rank	Disease	Disability (1-5)	CFR (1-5)	Av. Hosp. Stay (1-5)	Prevent-ability (1-3)	Treat-ability (1-3)	Score
1	Stroke	4	4	5	2	3	18
2	Cancer	4	4	3	3	3	17
3	Assault & Homicide	3	5	3	3	3	17
4	Encephalitis	4	3	4	2	3	16
5	Cirrhosis	3	3	3	3	3	15
6	Mental Illness	4	1	5	2	3	15
7	Drug Addiction	3	1	5	2	3	14
8	Suicide & Attempted	2	5	1	2	3	13
9	Coronary Heary Diseases	3	3	2	2	3	13
10	Diabetes mellitus	2	2	4	3	2	13
11	Traffic Accident	3	2	3	2	2	12
12	Drowning	1	5	1	2	3	12
13	Tuberculosis	2	1	5	2	1	11
14	Tetanus	2	3	3	1	2	11
15	Hypertension	2	2	2	2	2	10
16	Occupational Accident	3	1	2	2	2	10
17	Leprosy	4	1	1	2	2	10
18	Poliomyelitis	1	1	5	1	2	10
19	Diptheria	2	1	4	1	2	10
20	Rabies	1	5	1	1	2	10
21	Malaria	2	1	2	2	1	8
22	Peptic Ulcer	1	1	2	2	2	8
23	Pneumonia	1	1	2	3	1	8
24	Venereal Disease	1	1	3	2	1	8
25	Hepatitis A	1	1	2	3	1	8
26	Intest. Obstructions & Hernia	1	1	2	3	1	8
27	Cholera	1	1	2	3	1	8
28	Pertussis	1	1	2	2	2	8
29	Conjunctivitis	1	1	1	3	1	7
30	Influenza	1	1	1	3	1	7
31	Appendicitis	1	1	1	3	1	7
32	Enteric Fever	1	1	2	2	1	7
33	Leptospirosis	1	2	1	2	1	7
34	Acute Diarrhea	1	1	1	2	1	6
35	Dengue Haemorrhagic	1	1	1	2	1	6
36	Dysentery	1	1	1	2	1	6
37	Measles	1	1	2	1	1	6
38	Typhus	1	1	1	2	1	6
39	Helminthiasis	1	1	1	2	1	6
40	Rubella	1	1	1	1	1	5

Source: Adapted from National Epidemiology Board of Thailand. Review of the Health Situation in Thailand: Priority Ranking of Diseases. 1987.

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