A Comparative Assessment of

AIR POLLUTION PUBLIC HEALTH RISKS

In Two Israeli Metropolitan Areas

1995 - 1999

January 2003

Israel Ministry of the Environment
Israel Union for Environmental Defense (Adam Teva v'Din)
Tel Aviv Municipality
Ashdod-Yavne Regional Association of Towns for Environmental Protection
U.S. Environmental Protection Agency (USEPA)

A project funded, in part, by a grant from the United States – Israel Science and Technology Foundation

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Although this document has been independently reviewed by qualified scientists, as with all risk assessments, there are many areas of uncertainty associated with this analysis. The authors of this report have attempted to reasonably describe the strengths and limitations of this comparative risk analysis. Notwithstanding, there may be areas of uncertainty not identified in this report that could affect the results and conclusion of this analysis. Interpretation and application of the results of this analysis, as with all risk assessments, should be done with due regard to uncertainty.

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Overview

Air pollution contributes to death and illness in almost all countries. The World Health Organization estimates that air pollution will cause about 8 million deaths worldwide by 2020 (WHO, 1999). This report summarizes a binational collaborative effort between the United States and Israel to evaluate public health risks associated with air pollution in two Israeli regions, Tel Aviv and Ashdod. Extensive technical or scientific background is not required to understand the main body of this report. The report is organized into an Executive Summary and five main sections: "Introduction," "Background," "Project Approach," "Risk Characterization," and "Conclusions and Recommendations." The Introduction (Section 1) describes the comparative risk project's goals, scope, management, and organization. The Background (Section 2) describes air pollution standards and guidelines in Israel and elsewhere and the risk assessment process. Project Approach (Section 3) describes the risk assessment process as applied in this comparative risk project. Risk Characterization (Section 4) provides risk estimates and discusses the results within the context of the uncertainties associated with the comparative risk assessment. Conclusions and Recommendations (Section 5) summarizes important results, discusses the research and policy implications of the results, and makes recommendations. The report also includes six appendices, which provide technical details:

A. Regulation and Monitoring of Air Pollution in Israel,

B. Assessment of Exposures to Ambient Concentrations,

C. Health Assessment,

D. Results,

E. Summary of Air Pollution-Related Epidemiology Studies in Israel, and

F. References.[1]

[1] The appendices have been prepared so that they can be read as separate technical documents by individuals with specific disciplinary interests. As a consequence, there are some redundancies between the main body of the document and these appendices. Appendix E was almost completely rewritten concurrently with the scientific peer review and therefore has not undergone the same level of review as the rest of this document.

Foreword

In a nation beset with near-daily threats to public security, bringing the attention of policymakers and the general public to problems that are seemingly less immediate is no small challenge. Yet the threats posed by environmental pollution are real, tangible, and quantifiable according to modern risk assessment methodology as practiced widely in the United States and elsewhere. Through rigorously conducted risk assessment analysis, it is now possible to translate different levels of human exposure to environmental contaminants into reliable estimates of morbidity and mortality among affected populations.

This study, A Comparative Assessment of Air Pollution Public Health Risks in Two Israeli Metropolitan Areas, applies risk assessment methodology to air pollution hazards in two metropolitan areas that stretch along Israel's Mediterranean coastal plain: Greater Tel Aviv, in the center of the country, and the Ashdod-Yavne region, lying some 25 kilometers to the south of Tel Aviv. During two-and-a-half years of intensive research and analysis, a joint team of Israeli and U.S. experts have examined air quality monitoring results in these two regions, and compared them with health data from Israel and other parts of the world. Applying accepted risk assessment tools to the collected data, the team has arrived at a number of findings regarding illnesses and premature death resulting from exposure to air pollution in the two regions.

Beyond the potential practical applications of our joint research findings, this study represents an important effort in cooperation. The sharing of expertise between Israeli and U.S. research team members has been an invaluable exercise; just as pollution knows no boundaries, so the sharing of knowledge and ideas need not be confined within national borders. Equally important has been the sharing of information and expertise among government officials and scientists from the non-governmental sector in Israel. This cooperation has yielded practical results as well as an enhanced regard for professionalism inside and outside government agencies in Israel.

In the spirit of bilateral cooperation, the research team involved in this study would like to thank the United States - Israel Science and Technology Foundation for its support of this study.

A Comparative Assessment of Air Pollution Public Health Risks in Two Israeli Metropolitan Areas: 1995-1999

EXECUTIVE SUMMARY

Purpose and Goals

The project's purpose is to provide information for Israeli decisionmakers, the public, and nongovernmental organizations about potential air pollution impacts on human health. The project goals are two-fold: (1) to assess public health risks posed by existing air quality and (2) to build internal professional capacity within Israel to understand and conduct air pollution risk assessments. Specifically, we estimated and compared public health risks posed by several common air pollutants: particulate matter (PM), ozone (O_3), sulfur dioxide (SO_2), and nitrogen dioxide (NO_2). Attributable cases of mortality and illness were estimated in the Tel Aviv and Ashdod regions of Israel, with a combined population of approximately 1.2 million people. This study can form the foundation for future risk-based analyses of air pollution sources and control strategies.

Air pollution contributes to death (mortality) and illness (morbidity) in most countries. The World Health Organization estimates that air pollution will cause about 8 million deaths per year worldwide by 2020 (WHO, 1999). In Israel, population growth, vehicle use, and electrical demand, among other factors, contribute to the deterioration of air quality. Ambient air quality monitoring indicates violations of Israeli standards for several pollutants. Additional information about Israel's air pollution laws and monitoring is provided in Appendix A of this document. Monitoring data, however, are not adequate for determining which pollutants pose the greatest threats to health, nor do they provide an adequate basis for determining cost-effective solutions to air pollution. Risk assessment can provide a context for air quality monitoring data.

The risk assessment was a joint project between the Israeli Ministry of Environment (MoE); the Israeli Union for Environmental Defense (IUED, in Hebrew, Adam Teva V'Din), a nongovernmental environmental advocacy organization; and the United States Environmental Protection Agency (EPA). The technical workgroup that conducted the analysis comprised representatives of MoE, IUED, the Ashdod-Havel-Yavne Area Association of Towns for Environmental Protection, the Tel Aviv municipality, and EPA. The Israel Ministry of Health and the Israel Central Bureau of Statistics made significant contributions to the project.

Methods

The relationships between air pollution exposures and adverse human health responses appear similar across many geographic regions of the world. Human health responses observed with air pollutant exposures[1] in North America were used quantitatively to estimate the health effects of air pollutant exposures in Israel. Using data from countries other than Israel allowed a more complete characterization of air pollution risks than would otherwise be possible. This North American concentration-response information was combined with local ambient air pollutant concentration data to estimate the portion of regional death and illness attributable to local air pollution exposures. Israeli data used include populations and sub-population sizes, death and illness rates, and monitored ambient air pollution concentrations. This approach, described in detail in Appendices B and C, has been used successfully elsewhere to assess air pollution public health risks. It should be noted that only a subset of air pollutants, health effects, and exposure durations were evaluated.

Uncertainty

Embedded in any risk assessment are judgments for which complete scientific agreement has not yet been achieved. Risk assessors are often faced with several plausible approaches to a problem that result in different answers to the question of risk. Additionally, the assessor always has to cope with imperfect scientific data and questions that cannot be answered with available data. The judgments or science-policy choices necessary to conduct a risk assessment can have considerable influence on the results. Consequently, risk assessment should be viewed as a formal process for analyzing data, estimating risks and uncertainties, and presenting the results in a consistent manner, rather than as an approach leading to a precise and accurate statement of risk (NRC,1994).

[1] Data on observed or estimated health effects that occur in response to air pollution exposures is termed in this document "concentration-response."

Findings

Air pollution in the Tel Aviv and Ashdod regions is estimated to increase both death and illness. Increased mortality is associated with both short- and long- duration exposures to air pollution. Illness appears most notably elevated for hospital admissions and for respiratory symptoms in children. Of the pollutants evaluated, PM and O_3 are responsible for the greatest portion of cases of mortality and illness attributable to air pollution (several percent of normally occurring cases). This translates to hundreds to thousands of additional cases of death or illness per year in the populations of the Tel Aviv (about 1 million) and Ashdod (about 220,000) regions. The percentage of total annual deaths and illness attributable to air pollution is generally similar in the Tel Aviv and Ashdod regions. The differences in the estimated number of deaths and illness per year are mostly, but not entirely, due to differences in the size of the populations of Tel Aviv and Ashdod. It should be noted that significant risks are posed by exposures below current Israeli ambient air quality standards. It is expected that other urban areas in Israel with air pollution levels similar to those of Tel Aviv and Ashdod will experience similar public health impacts. More specifically:[2]

- *MORTALITY:* Mortality due to long term exposure attributed to $PM_{2.5}$ in Greater Tel Aviv and Greater Ashdod from anthropogenic sources is approximately 8 percent per year of the total mortality in both regions. In Greater Tel Aviv, there was an estimated average of 620 (95 percent confidence intervals CI = 340-900) deaths per year for 1995-1999 and in Greater Ashdod, an estimated average 90 (50-130) deaths per year for 1998-1999. Estimated mortality due to O_3 in Greater Tel Aviv and Greater Ashdod from anthropogenic sources is approximately 1 percent of the total annual mortality in both regions. In Greater Tel Aviv, there was an estimated average of 80 (20-130) deaths per year, and an estimated 10 (5-20) additional deaths per year in Greater Ashdod for1995-1997.
- *HOSPITALIZATION:* In Greater Tel Aviv for 1997, hospitalization attributed to $PM_{2.5}$ due to all respiratory causes from anthropogenic sources for all ages is estimated to be approximately 2 percent of all hospitalizations. This represents an estimated 370 (50-620) additional cases in 1997. Hospitalization due to all respiratory causes attributed to PM_{10} for ages ≥65 is estimated to be 6 percent, or 800 (720-1,110) additional cases. Estimated hospitalization due to cardiovascular causes attributed to PM_{10} for ages 65 is 3 percent, or 1220 (660-1,760) cases. PM data were not available for Ashdod in 1995-1997.

  Estimated hospitalization due to all respiratory causes attributed to O_3, for ages ≥65 in Greater Tel Aviv and Greater Ashdod is approximately 8 percent and 12 percent of all hospitalization, respectively. This estimaterepresents approximately 1120 (330-1850) and 180 (50-290) additional cases in 1997. An increasing trend in hospitalization is recognizedin both regions from 1995 to 1997. In fact, estimatesalmost doubled in Ashdod.

  Hospitalization due to all respiratory causes attributed to SO_2 in Greater Tel Aviv and Greater Ashdod is estimatedto be approximately 6 percent in both regions, or anestimated 1000 (350-1610) and 220 (80-360) additional cases in 1997. Hospitalization due to respiratory infections attributed to NO_2 in Greater TelAviv and Greater Ashdod is estimated to be approximately 4 percent and 2 percent respectively, or an estimated 615 (250-970) and 60 (20-90) additional cases in 1997. An increasing trend in hospitalization is recognized in Ashdod from 1995 to 1997. The estimated heath effects attributed to SO_2 and NO_2 are considerably more uncertain than are the estimates for PM and ozone.

- *RESPIRATORY SYMPTOMS:* In Greater Tel Aviv, an estimated 20 percent, or about 28,000 (14,400-37,000) cases per year, of respiratory symptoms in children for 1997-1999 are related to PM_{10}. Approximately 14 percent, or an estimated 20,000 (8,200-28,700) cases per year of respiratory symptoms in children for 1997-1999, are related to $PM_{2.5}$. For Ashdod, approximately 20 percent, or an estimated 6,000 (3,000-7,700) cases per year for 1998-1999, are related to PM_{10}, and an estimated average of 15 percent, or 4,400 (1,700-5,900) cases per year, are related to $PM_{2.5}$. In both regions, approximately 1 percent of asthmatic response for all ages is esimated to be attributed to SO_2.

These risk estimates are "best estimates" (i.e., bias neutral in terms of under- or over- estimation of risks). In other words, the estimates do not include any margin of safety for protecting public health. It should be noted that while the health risks to individuals from air pollution are relatively small, the overall public health consequences are considerable. At particular risk for air pollution-related effects are individuals above 65 years of age, children, and individuals with pre-existing diseases. Due to the many sources of uncertainty in the analyses presented here, the risk estimates should not be interpreted as precise measures of risk. The risk assessment can, however, provide useful information relevant to evaluating research priorities and risk management options. Additional details about the results of this study can be found in Appendix D.

[2] See Appendix D for a multiyear analysis. Risk estimates are rounded to the nearest 10.

Recommendations

The joint working group recommends that a comprehensive air quality management plan be developed that will:

1. Address $PM_{2.5}$ pollution and mitigation measures in the Tel Aviv and Ashdod regions to reduce $PM_{2.5}$ emissions from transportation and industry.

2. Address O_3 pollution and mitigation measures in the Tel Aviv and Ashdod regions to reduce O_3 precursors (i.e., volatile organic compounds and NO_2).

3. Evaluate year to year variations in monitored ambient concentrations. Trend analyses should be conducted to determine if pollution levels are increasing over time. Ashdod warrants particular attention.

4. Apply successful mitigation measures to other geographic areas of Israel with similar pollution levels.

5. Initiate public-health-based mitigation strategies, e.g., public health warnings for high pollution episodes.

6. Improve source apportionment for $PM_{2.5}$ and O_3 precursors.

In addition, the workgroup recommends new data gathering and analyses to:

7. Expand monitoring for $PM_{2.5}$ and other important air pollutants for which monitoring data are not available, such as carbon monoxide, lead, and other air toxics.

8. Estimate indoor O_3 concentrations and personal exposures (e.g., time-activity analyses) to reduce the uncertainty associated with O_3 risk estimates.

9. Consider the need for additional Israel-specific health data.

10. Update this assessment as new information warrants.

A Comparative Assessment of Air Pollution Public Health Risks in Two Israeli Metropolitan Areas: 1995-1999

1. INTRODUCTION

Air pollution contributes to death (mortality) and illness(morbidity) in most countries. The World Health Organization estimates that air pollution will cause about 8 million deaths worldwide by 2020 (WHO, 1999). In Israel, population growth, vehicle use, and electrical demand, among other factors, fuel deterioration in air quality. Ambient air quality monitoring indicates violations of Israeli standards for several pollutants. Since the U.S. air pollution standards are generally either quite similar or higher than the corresponding Israeli standards, we also expect a similar trend in the violation of U.S. standards.

Project Purpose and Goals

The project's purpose is to provide information for Israeli decisionmakers, nongovernmental organizations, and the public about potential air pollution impacts on human health. The project goals are two-fold: (1) to assess public health risks posed by existing air quality and (2) to build internal professional capacity within Israel to understand and conduct air pollution risk assessments. Specifically, we estimated and compared public health risks posed by several common air pollutants: particulate matter (PM), ozone (O_3), nitrogen dioxide (NO_2), and sulfur dioxide (SO_2). Attributable cases of death (mortality) and illness (morbidity) were estimated in the Tel Aviv and Ashdod regions of Israel. This study can form the foundation for future risk-based analyses of air pollution sources and control strategies.

Project Organization and Management

This project is unusual in both its management and comparative approach and can serve as a model for future projects. The project had its origin in 1998 during informal discussions between a senior official at the U.S. Environmental Protection Agency (EPA), Office of Research and Development, and the Israel Union for Environmental Defense (IUED), a nongovernmental environmental advocacy organization in Israel. The original concept called for a comparative human health risk assessment across environmental media that could assist Israel in establishing national environmental priorities. The concept evolved and, with the addition of the Israel Ministry of the Environment (MoE) as a project partner, a decision was made to focus the first phase of the comparative risk assessment on air pollution. A joint project proposal was drafted by EPA, MoE,[3] and IUED and submitted to the U.S.-Israel Science and Technology Commission, which approved funding for the project through the U.S.-Israel Science and Technology Foundation.

In April 2000, a workgroup was established, composed of staff from the MoE, IUED, the Ashdod-Havel-Yavne Area Association of Towns for Environmental Protection, the Tel Aviv municipality, and EPA. The workgroup defined and conducted all tasks jointly. The Israeli participants met regularly throughout the project and communicated with their American counterparts at frequent intervals. The workgroup reported to a Steering Committee of three people, one each from MoE, IUED, and EPA. The Steering Committee, in turn, reported at regular intervals to an Executive Committee composed of MoE's Director General and Deputy Director General; the Executive Director of IUED; and the Project Director, Director of the National Center for Environmental Research, EPA Office of Research and Development (Figure 1).[4]

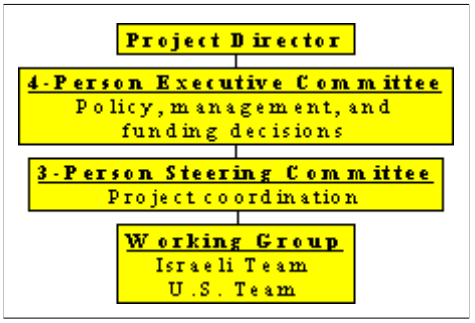

Figure 1: Project Management

[3] Under a 1991 bilateral agreement between MoE and EPA (Israel Ministry of the Environment, 2001).

[4] The Executive Committee at time of project completion (in alphabetical order): Yitzhak Goren, formerly Director General, Israel MoE; Miriam Haran, Deputy Director General, Israel MoE; Peter W. Preuss (Project Director), Director, EPA National Center for Environmental Research, and Philip Warburg, Executive Director, IUED.

Project Scope

Two geographical areas were selected by the workgroup for the comparative air pollution risk assessment: the Greater Tel Aviv metropolitan area and the Ashdod region, located approximately 25 kilometers to the south of Tel Aviv (Figure 2). Tel Aviv was selected because it is the largest urban center in Israel, with a population of over 1 million people. Major sources of Tel Aviv air pollution are transportation and power plants (Figure 3). The Greater Ashdod area, with a population of about 220,000 people, was selected to represent an Israeli industrial city. Sources of Ashdod air pollution include passenger vehicle traffic, truck transportation, a power plant, a petrochemical facility, metal recycling industries, agriculture-related industries, and a large deep-water port on the Mediterranean Sea (Figure 4). Additional factors considered in selecting these geographical areas for study included the availability of baseline health and ambient air quality data.

The project focused on four common air pollutants: PM, O_3, SO_2 and NO_2. The health assessment relied extensively on well-reviewed secondary publications, including several U.S. EPA documents (Air Quality Criteria Documents; Staff Papers; a report to the U.S. Congress entitled, "Benefits and Costs of the Clean Air Act- Appendix D: HumanHealth Effects of Criteria Pollutants" (1996 a,b EPA, 1999); and the World Health Organization's "Guidelines for Air Quality" (WHO, 1999). Primary sources were also reviewed for the risk calculations. Other common air pollutants with known human health effects (such as carbon monoxide, lead, benzene, and/or polycyclicorganic matter) were not evaluated due to limited resources and monitoring data.

Also, no ecological riskassessment of the four pollutants (PM, O3 , SO2, and NO2) was conducted; however, these pollutants are known to have substantial efects on the ecosystem such as crop damage.

Figure 2: Map of Israel Showing the Location of the Tel Aviv and Ashdod Study Areas

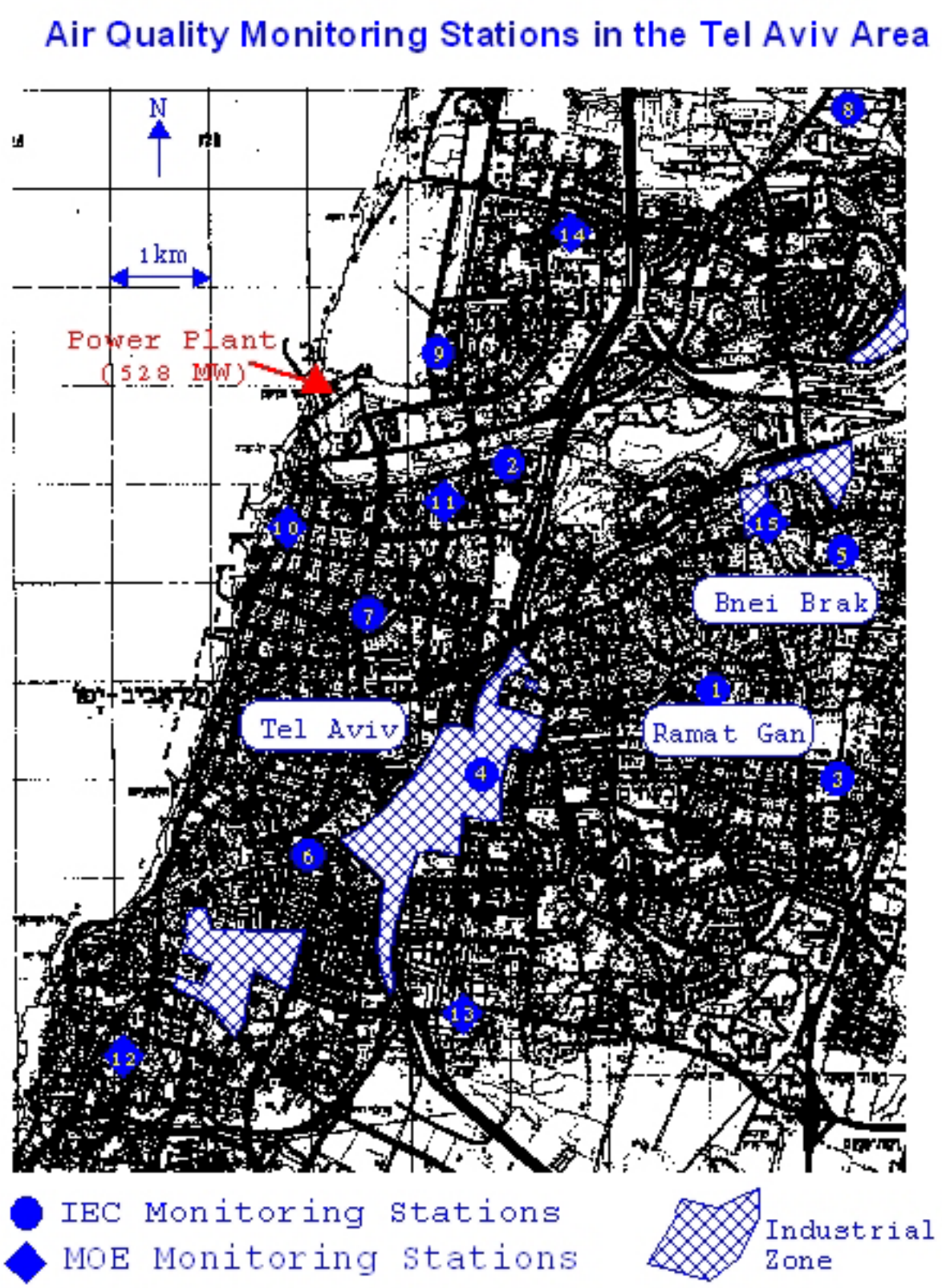

Figure 3: Map Showing Tel Aviv Study Area
The Tel Aviv study area includes the municipalities of Tel Aviv, Benei Brak, Halon, Givatayim, and Ramat Gan. Also shown are the locations of major stationary emission sources (power plant and industrial zones), major traffic arteries, and ambient air monitoring stations operated by the Israel Electric Company and the Israel Ministry of the Environment.

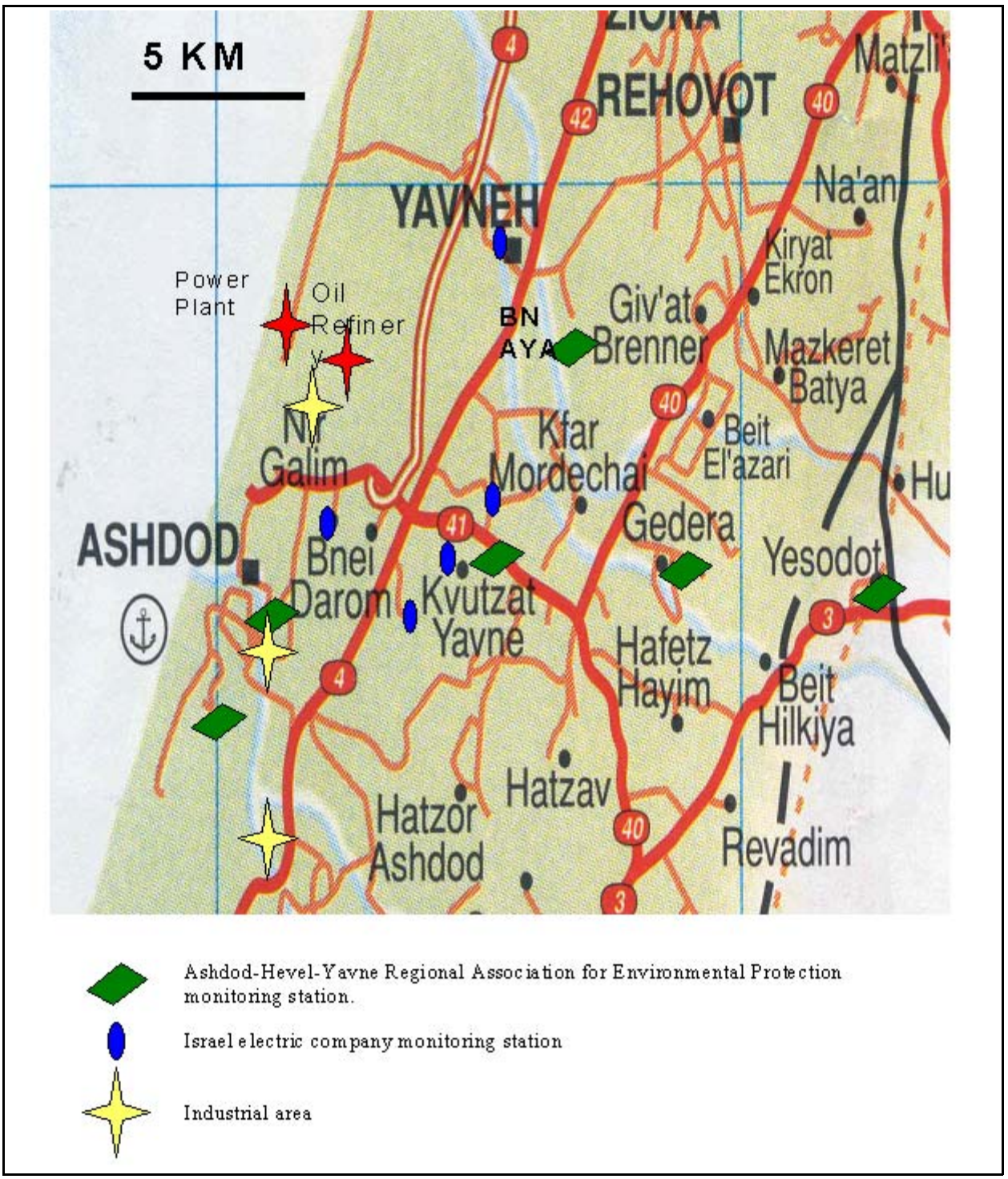

Figure 4: Map Showing Ashdod Study Area
The Ashdod study area includes the city of Ashdod and the towns of Bnaya, Gedera, and Gan-Yavne. Also shown are the locations of major stationary emission sources (sea port, oil refinery, power plant and industrial areas), major traffic arteries, and ambient air monitoring stations operated by the Ashdod-Havel-Yavne Regional Association for Environmental Protection and the Israel Electric Company.

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2. BACKGROUND

Regulatory Context

Israel has an active air pollution regulatory and monitoring program. Ambient air quality standards were issued in Israel under the Abatement of Nuisances Law in 1971 and revised in 1992 (Gabbay, 1998: 85-86). The regulations provide maximum and annual and/or 30-minute or hourly average ambient air concentration levels for O_3, SO_2, carbon monoxide (CO), nitrogen oxides (NO_x), sulfate (SO_4^{2-}), phosphate (PO_4^{3-}), settling dust, suspended particulate matter, respirable particulate matter (PM_{10}), three metals in particulate matter, and eight air toxins (IUED, 1993: C6-C8). Israeli ambient air pollution standards considered in this study are shown in Table 1, together with standards/guidelines from other nations and the World Heath Organization. Additional laws relevant to air quality management are discussed in Appendix A: Air Pollution Regulation and Monitoring in Israel.

Despite these regulations, emission estimates and air quality monitoring results raise concern about air quality in Israel. The comparative risk assessment presented in this document provides context for air pollution emission estimates and ambient air quality monitoring data and can assist the Israeli government, nongovernmental organizations, and the public in developing policy decisions and research priorities.

Table 1: Ambient Air Quality Standards and Guidelines in Israel and Elsewhere*

Pollutants	Duration	Israel	USA - EPA	USA - California	WHO	Germany	Europe
SO_2 µg/m^3	½ hour	500				1000	
	Hour			655			350
	24 hour	280	365	105	125	300	125
	Year	60	80		50	140	
NO_2 µg/m^3	½ hour	940 for NO_x				200	
	Hour			470	200		200
	24 hour	560 for NO_x				100	
	Year		100		40	80	40
CO mg/m^3	½ hour	60			60	50	
	Hour		40	23	30		10
	8 hour	11	10	10	10		
	Year					10	
O_3 µg/m^3	½ hour	230				120	
	Hour		235	180			180
	8 hour	160	157		120		
	Year					50	
PM_{10} µg/m^3	24 hour	150	150	50	**	200	50
	Year	60	50	20	**	100	30
$PM_{2.5}$ µg/m^3	24 hour		65	65			
	Year		15	12			

Conversion factors: 1 mg/m^3 SO_2=0.38 ppb; 1 mg/m^3 NO_2=0.53 ppb; 1 mg/m^3 CO=0.87 ppm; 1mg/m^3 O_3=0.51 ppb

* It should be noted that different countries have different forms of the standards, thereby affecting their actual stringency.

** WHO guidelines are set at concentrations below which no or little public health risks are thought to occur. All concentrations evaluated to date for PM appear to pose some risk to public health. Consequently, no WHO guideline value for PM has been set.

Risk Assessment Process

Risk assessment as a discipline has evolved over the last 50 years to assist in establishing limits that would protect human health and the environment from exposures to hazardous substances. Human health risk assessment entails the evaluation of scientific information on the hazardous properties of environmental agents and on the extent of human exposure to those agents. The product of the evaluation is a statement regarding the probability and degree that populations so exposed will be harmed (NRC, 1994).

Embedded in any risk assessment are judgments for which complete scientific agreement has not yet been achieved. Risk assessors are often faced with several plausible approaches to a problem that result in different answers to the question of risk. Additionally, the assessor always has to cope with imperfect scientific data and questions that cannot be answered with available data. The judgments or science-policy choices necessary to conduct a risk assessment can have considerable influence on the results. Consequently, risk assessment should be viewed as a formal process for analyzing data, estimating risks and uncertainties, and presenting the results in a consistent manner, rather than as an approach leading to a precise and accurate statement of risk (NRC, 1994).

Risk assessment is only one of several inputs to risk management.[5] Policy considerations dictate the extent to which risk information is used in decisionmaking and the extent to which other factors, such as technical feasibility and costs, play a role. Risk managers also can use risk assessments to rank environmental problems.

[5] Risk management is the term used to describe the process by which risk assessment results are integrated with other information to make decisions about the need for, and the methods and extent of, efforts to reduce risk.

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3. PROJECT APPROACH

Risk Assessment Process

The approach taken in this report is to integrate information on human responses to air pollution exposures, collected primarily in North America, with local information on populations, sub-populations, death and illness rates, and air monitoring information. This integration yields estimates of the public health risks associated with local air pollution. The approach can be expressed as follows:

$$\frac{\text{Observed North American Health Risks}}{\text{Monitored North American Concentration}} \approx \frac{\text{Predicted Israel Health Risks}}{\text{Monitored Israel Air Concentration}}$$

Generally consistent associations between health effects and exposures are observed in the United States of America, Canada, Western Europe, and Latin America, but not for cities in Central and Eastern Europe. The basis for the Central and Eastern European differences is unknown, but could result from differences in exposure measures, air pollution mix, climate, health status of the population, health care, etc. (EPA, 2001:6-12, 6-144, 9-35, 9-45.) Use of data collected in countries other than Israel allows a more complete characterization of risk than would otherwise be possible. Relatively few studies of Israeli air pollution health effects have been conducted. In this project, it is assumed that populations in North America and Israel have similar sensitivity to air pollution and exhibit similar responses to exposure. The validity of this assumption is unknown.

Figure 5 illustrates a commonly used risk assessment paradigm: exposure assessment, health assessment, and risk characterization. Key inputs to the risk assessment are also shown. Table 2 provides additional details about the risk assessment inputs and end-products.

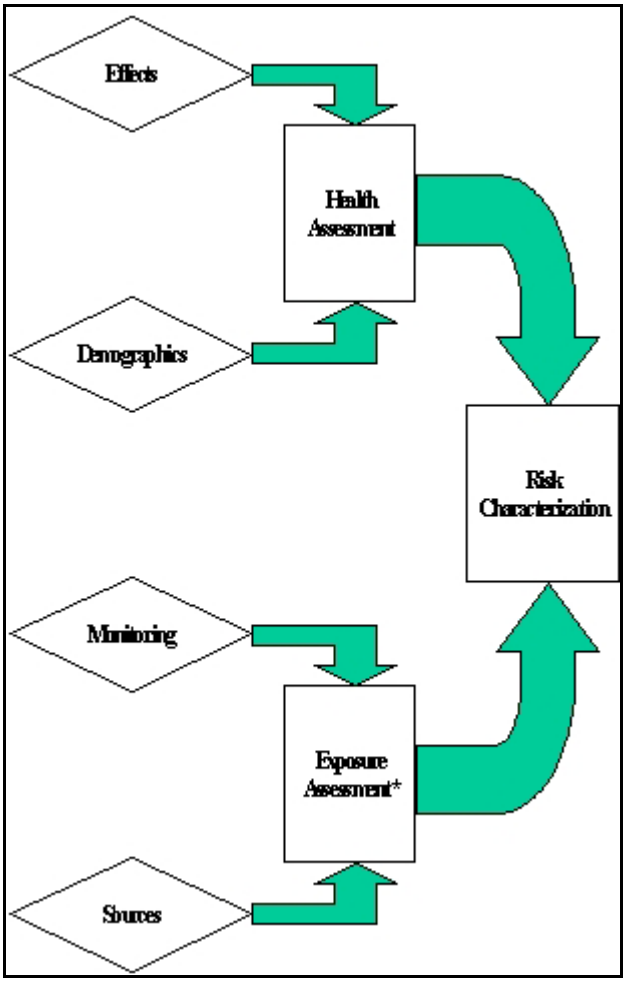

Figure 5: The Risk Assessment Paradigm and Key Inputs
The risk assessment paradigm is shown in the squares and inputs are shown in diamonds.
* Exposure assessment in this document is based on using ambient pollutant concentrations as surrogates for personal exposures, without adjustment for physical, behavioral, or microenvironmental factors that may influence actual personal exposures (see text for further details).

Table 2: Key Components of Risk Assessments

Exposure Assessment*			Health Assessment		Risk Characterization
Sources	**Monitoring**	**Physical and Personal Factors**	**Demographics**	**Effects**	**End Products**
Source Categories – Autos/diesels – Power plants – Industry – Port activities – Small sources *Emissions* *Atmospheric Transformation*	*Ambient Monitoring* – Monitoring sites – Data collection – PM, O_3, SO_2, NO_2 *Data* – Concentration – Averaging times	*Housing characteristics* *Pollutant-Specific Physical factors* – Penetration rates – Deposition rates – Dispersion rates *Microenvironmental concentrations* *Urban, rural and commuting/traffic information* *Demographic data by census tract* *Time-activity characteristics* – By age – By gender – By occupation	*Population Data* – By age – By district – By sub-populations *Baseline Health Data* – Mortality – Hospitalization – Less severe effects	*Adverse Effects* – Mortality – Hospitalization – Less severe effects *C-R** Equations* – Exposure/health integration – Relative risks	*Estimates of Risk* *Characterization of Uncertainties*

* Exposure assessment in this study is based on using ambient pollutant concentrations as surrogates for personal exposures without adjustment for physical, behavioral or microenvironmental factors that may influence actual personal exposures (see text for further details).

** C-R = concentration-response, i.e., the quantitative relationship of ambient air concentrations to health responses or effects.

Exposure Assessment Using Ambient Air Quality Data

The pollutants evaluated here (PM, O_3, SO_2, and NO_2) were chosen, in large part, based on the results of continuous ambient air monitoring conducted for the past 20 years in Israel (see Appendix B: Exposure Assessment). Little or no monitoring data are available for a variety of other common air pollutants known to cause adverse health effects, such as carbon monoxide and lead. Ambient monitoring data indicate widespread exposure to PM, O_3, SO_2, and NO_2 in Israeli urban areas.

Personal exposures to ambient pollutants may be estimated using either direct or indirect approaches. *Direct approaches* measure the contact of the person with the chemical concentration in the exposure media over an identified period of time. Direct measurement methods include personal exposure monitors that are worn continuously by individuals as they encounter various microenvironments and perform their daily activities. *Indirect approaches* use models and available information on concentrations of chemicals in microenvironments, the time individuals spend in those microenvironments, and personal PM-generating activities to estimate personal exposure. However, exposure assessment in this project relies exclusively upon using population-oriented fixed site ambient monitoring data as surrogates for personal exposures for the following key reason.

Most of the available epidemiological studies of PM, SO_2, NO_2 and other pollutants' health effects, in the absence of personal exposure monitoring or modeling data, use ambient outdoor community monitoring measurements (typically 24-h average concentrations) in the statistical analysis of pollution and health effects associations. An important question often raised in the interpretation of results from acute or chronic epidemiological community-based studies of PM and gaseous pollutants is whether the use of ambient stationary site pollutant concentration data influences or biases the findings from these studies. Because the health outcomes are measured on individuals, it is preferable to use personal exposure measurements when available, instead of surrogates such as ambient PM concentration measurements collected at one or more ambient monitoring sites in the community. As described in the recent EPA Draft PM Criteria Document (March 2001), "the use of ambient concentrations could lead to misclassification of individual exposures and to errors in the epidemiological analysis of pollution and health data

depending on the pollutant and on the mobility and lifestyles of the population studied. Ambient monitoring stations can be some distance away from the individuals and can represent only a fraction of all likely outdoor microenvironments that individuals come in contact with during the course of their daily lives. Furthermore, most individuals are quite mobile and move through multiple microenvironments (e.g., home, school, office, commuting, shopping, etc.) and engage in diverse personal activities at home (e.g., cooking, gardening, cleaning, smoking). Some of these microenvironments and activities may have different sources of PM and co-pollutants and result in distinctly different concentrations of PM [and other gaseous pollutants] than those monitored by the fixed-site ambient monitors" (USEPA 2001). Consequently, it is possible that exposures of some individuals may be incorrectly classified if only ambient monitoring data are used to estimate individual level exposures to PM and other gaseous species. However, the information base (on physical, microenvironmental, behavioral, and exposure factors) currently available for either the United States or Israel does not allow us to implement an alternative risk estimation methodology to the one employed here when the health risk coefficients that predominantly exist are based on analysis of ambient concentration-response (C-R) relationships.

Section 4. Risk Characterization combines the monitored ambient concentration data with published C-R relationships[6] from air pollution health studies to derive estimates of health risks associated with exposures to ambient pollutants in Israel. Further characterization of contributions of different indoor and outdoor sources of air pollution to projected health effects will require more difficult and complex analysis in the future.

Health Assessment Using Epidemiological Data[7]

The pollutants evaluated in this study are known to cause a variety of human health effects (WHO, 1999 and EPA, 1999). (See Appendix A for a listing of health effects.) Evidence comes from a substantial body of epidemiologic and clinical studies. Each pollutant is associated with its own, somewhat unique set of health effects. A major challenge of a comparative assessment is choosing effects and C-R relationships that yield reasonable comparisons among pollutants.

Effects

For this project, three measures of public health impacts were evaluated: mortality; hospitalization; and selected, less severe, health effects. These broad categories were chosen to facilitate comparisons across the somewhat dissimilar endpoints associated with each pollutant. Mortality and hospital admissions were the most comparable across pollutants.[8] For the less severe health effects category, characteristic effects of similar severity were selected for comparison. The selected effects are incapacitating and/or adversely impact quality of life, but are unlikely to result in death or require hospitalization. One such effect was chosen for each pollutant except NO_2. The health data for less severe effects associated with exposure to NO_2 were considered inadequate. The less severe effects selected were: lower respiratory symptoms in children (PM), decreased lung function with symptoms (O_3), and increased asthma symptoms in asthmatics (SO_2). It should be noted that these types of less severe effects can have significant effects on public health via the greater number of people potentially impacted relative to more severe effects like mortality and hospital admission. The heath effects described above are summarized in Table 3.

[6] Concentration-response describes the quantitative relationship of ambient air concentrations to health responses or effects. The coefficient is a number that represents the slope of the line from a plot of the magnitude of a defined response for a given concentration.

[7] Epidemiology is the study of the distribution and determinants of diseases and injuries in human populations. In epidemiology studies, the number of cases vs. the number of individuals affected is usually reported. Obviously, in some cases (e.g., mortality) these two measures are the same. Clinical studies are the study of the distribution and determinants of health-related states or events in specified human populations in a more controlled hospital or clinical setting.

[8] Specifically, respiratory and cardiovascular causes for hospital admissions were selected for analysis because these causes are most strongly associated with air pollution. The NO_2 risk analysis was limited to hospital admissions for respiratory infections (vs. all respiratory causes) because the data for NO_2 and effects other than respiratory infections were considered inadequate.

Pollutant	Health Effects
Particulate Matter	• Mortality • Hospital admissions – respiratory and cardiovascular • Respiratory symptoms
Ozone	• Mortality • Hospital admissions – respiratory • Decreased lung function with symptoms
Sulfur Dioxide	• Hospital admissions – respiratory • Increased asthma symptoms
Nitrogen Dioxide	• Hospital admissions – respiratory infections

Table 3: Health Effects Quantified for the Israeli Project

For mortality and hospital admissions, the Israel Ministry of Health provided data specific to Tel Aviv and Ashdod. For less severe health effects, information on the incidence[9] of these effects in Israel was not readily available. For the analysis, it was assumed that the rates of respiratory symptoms in children and O_3 sensitivity are the same in Israel and the United States. The incidence similarity between these two countries is not known. Available information indicates similar rates of asthma in the United States and Israel (The Lungs Clinic, Rabin Medical Center (In Hebrew), http://www.lung-rmc.co.il).

Concentration-Response

The C-R relationship describes the magnitude of a defined health response at given concentrations. As concentrations to which people are exposed increase, the number of people affected increases. North American studies of human responses to measured air pollution, from both epidemiologic and clinical studies, were used to estimate the number of cases of death or illness attributable to air pollution in the Tel Aviv and Ashdod regions.[10] In these studies, the relationship between monitored air pollutant concentrations and observed health effect is quantified. The calculations of risks in Tel Aviv and Ashdod also included local data on population sizes and occurrence of health effects. Where possible, studies of similar design were used to facilitate comparisons across pollutants.

In many cases, multiple C-R studies exist for the same pollutant and health effect. When a single, optimal study could not be identified to characterize the C-R relationship, a science policy choice was made to use an approximation of the mean or central tendency of the most appropriate C-R relationships for the particular effect in question. In some cases, pooled C-R relationships and an estimate of the central tendency were taken from the secondary literature. In other cases, the C-R relationship was taken from one study most closely approximating the mean of the possible C-R relationships. While we have attempted to present a suite of C-R relationships that typify the current peer-reviewed literature, it would also be reasonable to identify alternative C-R relationships that may result in somewhat different results. Additional information on the selection and use of C-R relationships in this comparative risk assessment is found in Appendix C: Health Assessment. The following section (Risk Characterization) combines the monitored ambient concentration data (discussed in the previous section) with the C-R information discussed here to derive estimates of health risks associated with exposure to ambient air pollutants in Israel.

[9] "Incidence" is the rate at which new cases of disease or health disorder arise in a population.

[10] North American studies were used due to the number of studies available and the extensive review they have undergone as part of the U.S. regulatory scrutiny of air pollution. The degree to which North American and Israeli C-R relationships may differ is unknown.

A Comparative Assessment of Air Pollution Public Health Risks in Two Israeli Metropolitan Areas: 1995-1999

4. RISK CHARACTERIZATION

Risk characterization integrates exposure and health assessments to produce estimates of risk. This section provides quantitative estimates of public health risks associated with existing air quality. Additionally, characterizations of the uncertainties in the resulting risk estimates have been developed. The confidence in the underlying data is described in Appendices B: Exposure Assessment and C: Health Assessment. A more detailed description of risks is provided in Appendix D: Results. Risk estimates are rounded to the nearest 10. The risk estimates presented in this document are "best estimates" (i.e., bias neutral in terms of under- or over- estimation of risks). In other words, the estimates do not include any margin of safety for protecting public health.

Risk Estimates

Estimated health risks associated with exposures to ambient man-made (anthropogenic) air pollutants in Greater TelAviv and Greater Ashdod for 1997 are shown in Tables 3, 4, and 5. Estimated risks are shown for mortality, hospital admissions and selected, less severe, health effects. Additional analyses for 1995-1999 and for combined anthropogenic and naturally occurring pollution are presented in Appendix D: Results.

Table 4: Estimated Risks for Mortality – 1997
Annual Incidence (% of total cases)
(95% Confidence Interval)

Pollutant	Health Effect	Population Evaluated	Man-Made Pollution*	
			PM_{10}	$PM_{2.5}$
Greater Tel Aviv				
Particulate Matter	Mortality – Long Term Exposures	≥30	**NA**	**680** (8%) (370-980)
Particulate Matter	Mortality – Short-Term Exposures	All ages	**220** (3%) (60-380)	**220** (3%) (110-280)
Ozone	Mortality – Short-Term Exposures	All ages	**90** (1%) (30-150)	
Greater Ashdod				
Particulate Matter	Mortality – Long Term Exposures	≥30	**NA**	**NA**
Particulate Matter	Mortality – Short-Term Exposures	All ages	**NA**	**NA**
Ozone	Mortality – Short-Term Exposures	All ages	**20** (1%) (10-30)	

* Background concentrations: PM_{10}=[20 µg/m^3]; $PM_{2.5}$=[10 µg/m^3]; O_3=[8 ppb]; SO_2=[2 ppb]; NO_2=[5 ppb]
NA = estimate Not Available

Table 5: Risk Estimates for Hospital Admissions – 1997
Annual Incidence (% of total cases)
(95% Confidence Interval)

Pollutants	Health Effects	Population Evaluated	Man-Made Pollution*	
			PM_{10}	$PM_{2.5}$
Greater Tel Aviv				
Particulate Matter	Respiratory – All causes	≥65 (PM_{10}) All ages ($PM_{2.5}$)	**800** (6%) (470-1110)	**370** (2%) (50-620)
Particulate Matter	Cardiovascular – Selected causes†	≥65	**1220** (3%) (660-1760)	**NA**
Ozone	Respiratory – All causes	≥65	**1120** (8%) (330-1850)	
Sulfur Dioxide	Respiratory – All causes	All ages	**1000** (6%) (350-1610)	
Nitrogen Dioxide	Respiratory infections‡	All ages	**620** (4%) (250-970)	
Greater Ashdod				
Particulate Matter	Respiratory – All causes	≥65 (PM_{10}) All ages ($PM_{2.5}$)	**NA**	**NA**
Particulate Matter	Cardiovascular – Selected causes†	≥65	**NA**	**NA**
Ozone	Respiratory – All causes	≥65	**180** (12%) (50-290)	
Sulfur Dioxide	Respiratory – All causes	All ages	**220** (6%) (80-360)	
Nitrogen Dioxide	Respiratory infections‡	All ages	**60** (2%) (20-90)	

* Background concentrations: PM_{10}=[20 µg/m^3]; $PM_{2.5}$=[10 µg/m^3]; O_3=[8 ppb]; SO_2=[2 ppb]; NO_2=[5 ppb]
NA = estimate Not Available
† For both cities, this is likely an underestimate of risk. This estimate was limited to International Classification of Disease (ICD) Codes: 410, 411-414, 427. The original study (Schwartz, 1999) included ICD Codes: 390-429.
‡ For both cities, the baseline population was available only for ICD codes: 460-519 (all respiratory causes); number of respiratory infections (ICD codes: 464,466, 480-487, 494) was estimated at ~ 55% of total admissions based on Burnett et al., 1997 and 1999.

Table 6: Risk Estimates for Other Health Effects – 1997
Annual Incidence (% of total cases)
(95% Confidence Interval)

Pollutants	Health Effects	Population Evaluated	Man-Made Pollution* Only	
			PM_{10}	$PM_{2.5}$
Greater Tel Aviv				
Particulate Matter	Respiratory Symptoms	Ages 7-14	**27140** (19%) (14140-36520)	**19470** (14%) (8110-28170
Ozone	Impaired Lung Function with Symptoms	All ages	**NA**	
Sulfur dioxide	Asthmatic Response	All ages	(1%)	
Greater Ashdod				
Particulate Matter (PM_{10})	Respiratory Symptoms	Ages 7-14	**NA**	
Ozone	Impaired Lung Function with Symptoms†	All ages	**<1490**	
Sulfur Dioxide	Asthmatic Response	All ages	(1%)	

* Background concentrations: PM_{10}=[20 µg/m^3]; $PM_{2.5}$=[10 µg/m^3]; O_3=[8 ppb]; SO_2=[2 ppb]; NO_2=[5 ppb]
NA = estimate Not Available
† The risk estimates for impaired lung function with symptoms from ozone are presented without adjusting for personal exposure. Indoor concentrations of ozone are typically much lower than outdoor levels because ozone is a highly reactive gas. According to Ozkaynak (1999) and Lee et al. (2002), typical indoor ozone levels in U.S. homes range around 10 percent to 30 percent of outdoor concentrations, depending on presence of air conditioners and other indoor source or ventilation characteristics. On the other hand, indoor/outdoor ozone ratios in residences with open windows could be much greater, or around 0.7 (Lee et al., 1997). Unfortunately, in the absence of Israeli-specific data on ozone penetration factors into local homes (which are expected to be quite different than in the United States) and time spent outdoors by different individuals, it is impossible to develop at this time a reliable time-weighted personal exposure factor, based on ambient ozone measurements alone, that can be used to adjust impaired lung function risk estimates presented here using the ambient monitoring data collected in Israel. Moreover, the available C-R relationship for ozone and impaired lung functions with symptoms suggests a threshold below which the C-R relationship is zero, which further complicates this analysis. Thus, the calculations shown in this report, without employing a personal exposure adjustment factor, should be considered upper bound risk estimates for the projected lung function impairments in Israel.

Mortality

PM and O_3 were evaluated for mortality risks. Data for SO_2 and NO_2 related mortality were considered inadequate. Both PM and O_3 are associated with significant risks. Risk estimates suggest that PM risks from anthropogenic sources are 3 percent of the total annual deaths for short-term PM exposures and 8 percent of total annual deaths for PM long-term exposures.[11] This translates to hundreds of PM attributable deaths per year in Greater Tel Aviv (95 percent confidence intervals (CI) = 110-280 for short-term exposures; 370-980 for long-term exposures). Ozone attributable risks from anthropogenic sources are approximately 1 percent of the total annual deaths. This roughly translates to dozens of O_3 attributable deaths per year in Greater Tel Aviv (CI = 30-150) and about 15 additional deaths per year in Greater Ashdod (CI = 10-30).

Considerable controversy exists about reported associations between O_3 and mortality. In general, the controversy revolves around the potential confounding effects of PM. In an attempt to shed additional light on this issue, EPA evaluated in detail four studies that controlled for PM as a confounder. In three of the four studies elevated relative risks for O_3 exposures and mortality were observed.[12] Considering (1) the outcome of this evaluation, (2) the importance of the health effect, and (3) a desire to more broadly compare the possible public health risks posed by O_3 relative to PM, we chose to include O_3-associated mortality in this analysis . This comparative assessment suggests that if O_3 causes increased mortality, the O_3-related effect is relatively small compared to PM-associated mortality. A reasonable alterative to this approach would have been to not evaluate the association between O_3 and mortality and to await future studies of this issue. To date, EPA has chosen this latter approach. Additional study of this issue is clearly needed.

Hospital Admissions

Particulate matter, O_3, SO_2, and NO_2 were evaluated relative to increased hospital admissions. Hospital admissions for all respiratory causes (PM, O_3, SO_2), respiratory infections (NO_2), and selected cardiovascular causes (PM) were considered. Increased hospital admissions for these causes have been most strongly linked to air pollution. As was noted earlier, substantial evidence implicates PM and O_3 exposures as causes of increased hospital admissions. A few well-conducted studies have also implicated SO_2 and NO_2 exposures in increased hospital admissions, but the weight of evidence is not great at this time. Hospital admissions for NO_2 were restricted to respiratory infections (versus all respiratory causes) because the evidence for other causes was considered inadequate.[13] For comparative purposes, estimated hospital admission risks for all four pollutants are reported here. It should be noted that the uncertainties associated with SO_2- and NO_2- related hospitalizations are considerably greater than the uncertainties associated with PM and O_3.

PM, O_3, SO_2, and NO_2 are all associated with substantial risks of increased hospital admissions. Hundreds to thousands of increased annual hospital admissions are estimated for Greater Tel Aviv. Tens to hundreds of air pollution attributable admissions are estimated in Ashdod. Risks range from 2 percent of total respiratory infection admissions attributable to NO_2 (Ashdod) to 12 percent for all respiratory causes for O_3 (Ashdod). In considering the relative estimated risks for PM_{10}, O_3, SO_2, and NO_2, it should be noted that the C-R relationships for PM and O_3 are based on a subset of the population, i.e., risks to individuals older than 65; C-R

[11] Long- and short- term PM exposure analyses are considered to be different analyses of the same pollutant-related effect and should not be added together. To do so would double count attributable deaths. Studies of long-term exposures are believed to capture more fully pollutant-related deaths, as compared to studies of short-term pollutant exposures. Studies of long-term exposures are, however, more difficult and, consequently, fewer such studies are available. For this reason, most of the studies included in this report rely on short-term exposure characterizations, usually ≤24 hours.

[12] For additional discussion, see: EPA's "Benefits and Costs of the Clean Air Act, 1990 to 2010"(1999), page D-19.

[13] There are two aspects of risk that must be considered. One aspect is called "weight of evidence" and refers to the quality and quantity of the data suggesting a causal relationship between a pollutant and an effect. Weight of evidence is a qualitative characterization of one's confidence in the link between a pollutant and an effect. It is weight of evidence that is discussed in this paragraph. The second aspect of risk is the quantitative estimate that results from evaluation of the C-R relationship and exposure. This aspect of risk is discussed in the next paragraph. The importance of these two aspects of risk is evident in the following discussion of hospital admission and highlighted in Figure 6. In Figure 6, the respiratory admissions for all pollutants evaluated are similar and range from 5-6 percent for PM_{10}, O_3, SO_2, and NO_2. The amount and quality of evidence linking pollutant exposure to increased health effects, however, is much stronger for PM_{10} and O_3 than for SO_2 and NO_2. Hence, the risks for hospital admissions shown in Figure 7 would be considered greater for PM_{10} and O_3 than for SO_2 and NO_2 even though the quantitative estimates are similar.

relationships for SO_2 and NO_2 are based on the total population. These differences are based on differences in the design of studies from which the C-R relationships were taken. It is reasonable to expect that PM and O_3 studies of risks to the total population would yield higher numbers.

Less Severe Health Effects

PM, O_3 and SO_2 were evaluated for selected less severe health effects. Each pollutant was evaluated for one characteristic, similar severity effect. The data for NO_2 effects were considered inadequate for evaluation. The risk estimates for the three ranged from hundreds to tens of thousands of additional annual cases of adverse health effects. One to 19 percent of the total number of cases occurring were attributable to air pollution. Estimated incidences in Greater Tel Aviv were: approximately 27,000 annual cases (95 percent Confidence Interval (CI) ≈ 14,000-37,000) of lower respiratory effects in children (cough, chest pain, phlegm, wheeze) associated with PM and 3,400 or fewer annual cases of increased asthmatic response associated with SO_2.

The exposure assessment for SO_2 very short-term exposures (e.g., 5 minutes) is complex. The "at-risk" population is exercising asthmatics. The risk assessment, as conducted here, calculates the incidence assuming that a person with asthma is engaged in moderate to heavy physical activity at the time and place that a "peak" exposure occurs. In other words, the assessment more reflects the occurrence of a concentration that could be expected to cause an effect independent of the likelihood that a susceptible person would be present when the peak exposure occurred. More precise estimates of risk would require additional information on time-activity and exposure patterns. Consequently, these SO_2 risks will likely be overestimates and are more uncertain than are the assessments for the other pollutants and exposure durations. In addition, if 5-minute peak exposures exceed 3.0 ppm, the entire population is at risk of experiencing bronchioconstriction. Review of the Israeli monitoring data suggested that such concentrations are unlikely over a wide geographic area.

Ozone-related cases of impaired lung function were not evaluated in Greater Tel Aviv. Estimated incidences in Greater Ashdod were less than approximately 1490 annual cases of impaired lung function with symptoms associated with O_3,[14] and 750 (CI= 620-940) or fewer annual cases of increased asthmatic response associated with SO_2.

Summary

Comparison of Risks Across Pollutants

Figure 6 highlights the cross-pollutants comparison of the risks presented in Tables 4, 5, and 6. PM and O_3 appear to be associated with higher risks, more health effects, and greater confidence in the data relative to SO_2 and NO_2. Evidence for SO_2- related health effects is greater than for NO_2. PM appears to pose greater risks than O_3. The evidence associating PM and O_3 with mortality, hospital admissions, and the less severe health effects is substantial. The evidence linking SO_2 and NO_2 with mortality is considered inadequate at this time. Evidence linking SO_2 and NO_2 with hospital admissions is considered limited for SO_2 and very limited for NO_2. Potential risks for increased hospitalization are presented for all four pollutants, however, for purposes of comparison. The data linking PM, O_3 and SO_2 to less severe health effects are substantial. Data for NO_2 and less severe health effects were considered inadequate. Data on indoor O_3 concentrations and time spent indoors will be necessary for a more accurate estimate of less severe health effects from O_3. Continued attention to evolving data is warranted.

[14] The estimated risk for impaired lung function with symptoms from ozone is provided without employing a personal exposure adjustment factor, and therefore should be considered an upper bound risk estimates for the projected lung function impairments in Israel. See footnote to Table 6 and Appendix D for additional information on this risk estimate.

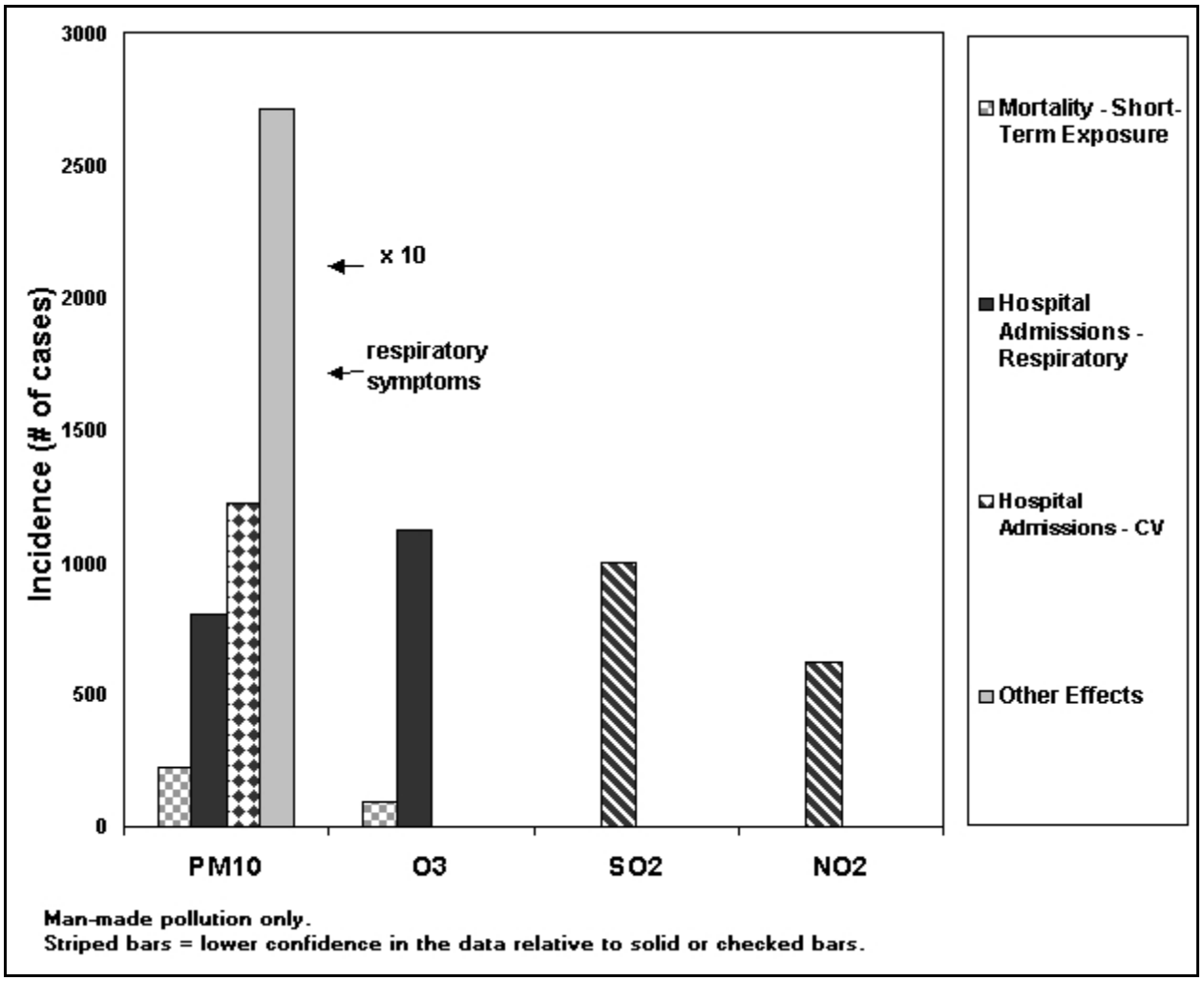

Figure 6: Comparison of Pollutants & Selected Health Effects
Annual Incidence - 1997
Greater Tel Aviv

PM_{10} vs. $PM_{2.5}$

PM is a broad class of chemically and physically diverse substances spanning several orders of magnitude in size (see Appendix B for more details). PM is often divided into two categories based on aerodynamic diameter: coarse ($PM_{10} \leq 10$ microns) and fine ($PM_{2.5} \leq 2.5$ microns). These categories are generally associated with different pollution sources. A substantial component of PM_{10} is dust from the earth's crust. A substantial component of $PM_{2.5}$ originates from combustion sources, such as vehicle emissions and power generation. The association of health effects with $PM_{2.5}$ is clear. The role of PM_{10} without the $PM_{2.5}$ fraction is more controversial, i.e., health effects associated with PM_{10} could be due to the $PM_{2.5}$ fraction, or the $PM_{10-2.5}$ fraction may independently cause health effects. Figure 7 compares risks associated with PM_{10}, which includes the $PM_{2.5}$ fraction, and risks associated with the $PM_{2.5}$ fraction alone. The data presented in Figure 7 (studies used in this project) suggest that the $PM_{2.5}$ fraction is responsible for the majority of risks observed when PM_{10} is used as a measure of PM concentration. Risk estimates for PM_{10} and $PM_{2.5}$ should be viewed as providing alternative estimates of PM risks. The risk estimates for the two different measures of PM should not be added together. Attention to evolving information on the role of $PM_{10-2.5}$ is warranted.

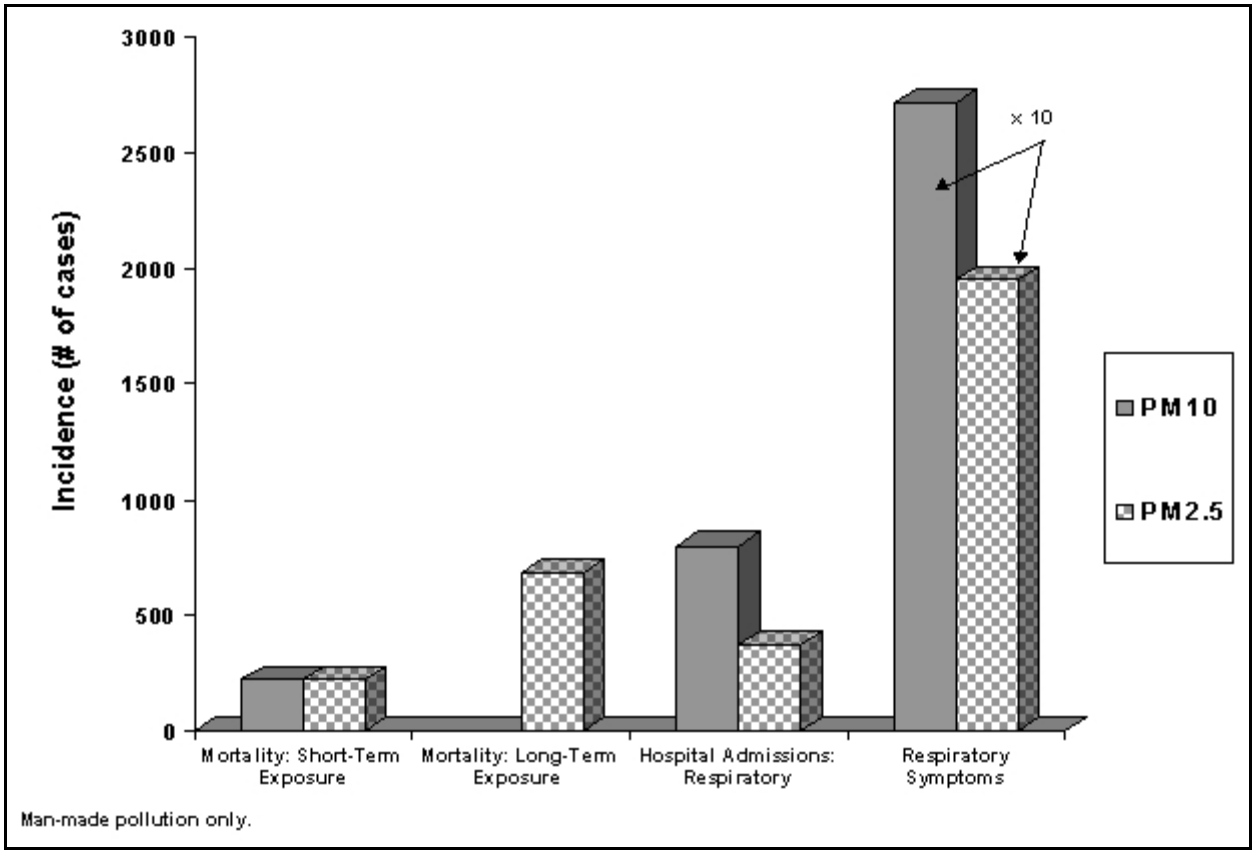

Figure 7: PM_{10} vs. $PM_{2.5}$ Risks
Annual Incidence - 1997
Greater Tel Aviv

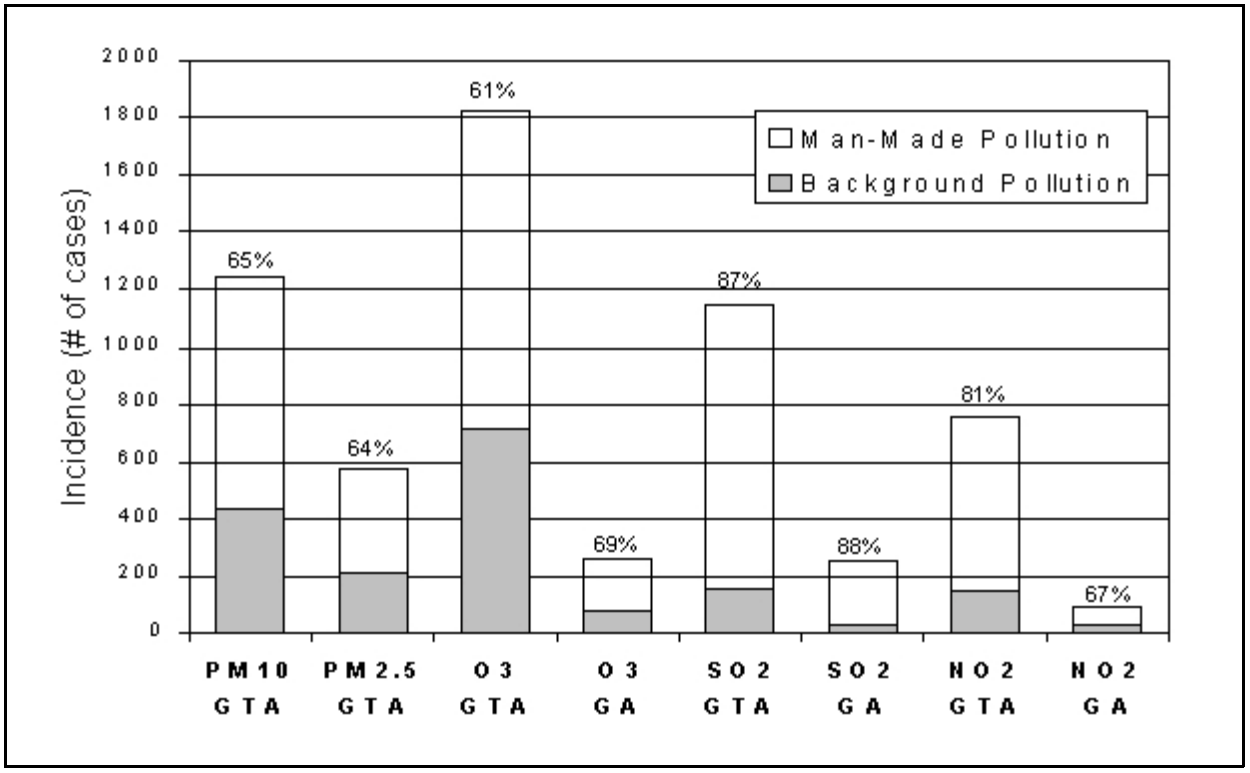

Figure 8: Man-Made vs. Background Pollution
Annual Incidence - 1997
Hospital Admissions - Respiratory Causes
Greater Tel Aviv (GTA) and Greater Ashdod (GA)
Percentage (%) are the percent of the incidences associated with the man-made pollution.
PM hospitalization (respiratory causes) risk estimates are not available for GA.

Anthropogenic Pollution vs. Background Pollution

An important issue related to air pollution is that of anthropogenic vs. background pollution. The "background" concentration is defined as the concentration of a pollutant present in the ambient air from natural sources.[15] For this project, risks associated with both man-made and background pollution were estimated (for details, see Appendix D: Results). Man-made pollution is always the most significant contributor to risks, although contributions vary with pollutant.

Estimates of man-made contributions to mortality risks range from 60 to 63 percent of total pollution risks (man-made plus background) for O_3 and PM. Figure 8 shows hospital admission risks associated both with man-made pollution and background pollution in Tel Aviv and Ashdod. The percentages of man-made pollution risk is similar in both cities, although there are significant differences in the risk estimates. Estimates of man-made contributions to total pollutant-related hospital admission risks (respiratory causes) in Greater Tel Aviv are 65, 61, 69, 87, and 81 percent for PM_{10}, $PM_{2.5}$, O_3, SO_2, and NO_2, respectively. Estimates of man-made contributions of total pollutant-related hospital admission risks (respiratory causes) in Greater Ashdod are 61, 88, and 67 percent for O_3, SO_2, and NO_2, respectively. PM risk estimates of hospital admissions from respiratory causes for Greater Ashdod are not available. It should be noted that from the public health perspective, the source of the pollution does not matter; steps can be taken to mitigate risks posed by background as well as man-made pollution.

[15] Because of their unusually high PM contributions, severe windstorm days were eliminated from all calculations.

Uncertainties

This section highlights some of the uncertainties that are embedded in the risk assessment. Table 7 lists important uncertainties and indicates the direction that the uncertainty is believed to bias the assessment, if known. Additionally, the sensitivity of the risk estimates to a few selected uncertainties was investigated. This sensitivity analysis is intended to provide a general sense of the impacts that assumptions can have on the risk assessment results, rather than a thorough characterization of the uncertainties. As noted earlier, the judgments, or science-policy choices, necessary to conduct a risk assessment can have considerable influence on the results.

Table 7: Uncertainties in Risk Assessment

Bias	Uncertainties in Exposure Assessment	Analysis
-/+	Contribution of naturally occurring background to ambient concentration.	Sensitivity analysis
-/+	Missing data due to systematic calibration of equipment.	Qualitative
-	Elimination of dust storm days from analysis.	Qualitative
-/+	Number of monitors varies between years.	Qualitative
-/+	Limited number of monitoring stations for $PM_{2.5}$.	Qualitative
-/+	$PM_{2.5}$ calculated as percent of PM_{10}.	Qualitative
-/+	Use of area-wide concentrations as indicator of exposure.	Qualitative
+	No indoor air concentration data for O_3.	Qualitative
-/+	No time-activity patterns.	Qualitative
-/+	Inadequate source inventory.	Qualitative
Bias	**Uncertainties – Health Assessment**	**Analysis**
-	Subset of air pollutants, health effects, and exposure durations used.	Qualitative
-/+	Predominant use of studies conducted in North America.	Qualitative
-/+	Study choice in characterizing the concentration - response relationships.	Sensitivity analysis
-/+	Variability around the concentration - response slope estimate (β).	Sensitivity analysis
-/+	Limitation in health data.	Qualitative
-/+	Non-accidental mortality data for 1998-1999 not available. (Calculations based on 1995-1997 data.)	Qualitative
-	Israel Ministry of Health data estimated to cover about 80 percent of the total incidence.	Qualitative
-	Israel Ministry of Health ICD classification did not always correspond exactly to C-R study ICD codes.	Qualitative
-/+	Percentage of the population with selected, less severe effects was calculated using U.S. data.	Qualitative
-	Risks calculated only for sub-populations evaluated in original C-R studies; estimates not extended to the total population.	Qualitative
-/+	Threshold vs. nonthreshold public health responses unknown	Qualitative

(-)Indicates likely underestimate of risk.
(+)Indicates likely overestimate of risk.
(-/+) Unknown or inconsistent direction of bias.

The impacts of selected uncertainties are shown on the estimated risk for $PM_{2.5}$ long-term mortality in Figure 9. The intent of Figure 9 is to illustrate to those unfamiliar with risk assessment how single assumptions or scientific judgments can impact risk estimates (i.e., the study choice, selection of C-R relationships, and the choice of ambient background concentrations, the first three bars) and how individual assumptions combine to further increase the uncertainty in the risk estimates (fourth bar). This example is restricted to risks estimated for mortality associated with long-term $PM_{2.5}$ exposures and to three important assumptions that have been made in the Israel assessment. It is important to realize there are many such assumptions embedded in any risk assessment. Figure 9 is shown as an example of the importance of considering uncertainties in any risk assessment and is in no way a thorough characterization of the uncertainties in the Israel assessment.

Individual uncertainties shown in Figure 9 are: (1) health-study choice, (2) variability around the C-R coefficient (also called slope estimate or beta, "β"), and (3) background pollutant levels. Each of these uncertainties and their underlying assumptions is briefly discussed.

- ***The first bar in Figure 9 illustrates how study choice impacts risk estimates.*** There were two reasonable studies that could have been used in this analysis of PM mortality and long-term exposures:

Dockery et al. (1993), and Pope et al. (1995). The Pope study was chosen for use in our analysis because it best met our selection criteria, i.e., a newer and larger study. The Pope et al. study included over half a million adults in more than 151 cities versus the Dockery et al. study of 8,111 adults in six cities. Also, the Pope study was designed to address some issues that had been raised by Dockery et al. (US EPA, 1996b, pages V14-15). The Dockery et al. study, however, is a reasonable alternative for estimating risks. Dockery et al. estimated a 26 percent higher average mortality in a PM-exposed population than would be expected in a similar but unexposed population. In contrast, Pope et al. estimated a 17 percent higher average mortality than expected. The first bar in Figure 9 shows how use of one study versus the other study would yield different estimates of relative risk. In this particular case, the risks presented in the Israel assessment for PM-associated mortality are lower than would have been estimated if the Dockery et al. study had been used.

- ***The second bar in Figure 9 illustrates how variability in the estimate of the C-R coefficient in a single study can impact risk estimates.*** The second bar shows the statistical variability in the estimate of the C-R coefficient (95 percent confidence intervals) from one single study (Pope et al., 1995). Ninety-five percent confidence intervals indicate that, statistically speaking, one is 95 percent confident that the "true" C-R coefficient lies within the range indicated by each end of the second bar in Figure 9. In this specific case, the Pope et al. study suggests that "true" increase in mortality risks is very likely to be between 9 and 26 percent greater in a PM-exposed population than in a similar unexposed population; also there is an equal probability that the "true" risk could be higher or lower than the mean. The possibility that the "true" risk is outside of this range also cannot be eliminated. The Israel comparative risk assessment presents both the mean and the range of estimates derived from the upper and lower 95 percent confidence intervals (see Table D as an example, especially Table D1-B for the variability shown in Figure 9 - $PM_{2.5}$ associated mortality, annual exposures, annual incidence 1997.) The approach taken in the Israel assessment is that presenting the means and the ranges of risk provide the users of the risk assessment the greatest amount of information. Discussing the estimated risks in terms of ranges also helps to avoid an unrealistic sense of precision in the risk estimates. An alternative approach to risk characterization that has been used elsewhere is to use the upper 95 percent estimate of risk. In this example that would be the 26 percent estimate from the Pope study. This type of assessment generally describes risks as being equal to or less than the 95 percent upper confidence interval of the risk estimate. This approach assumes that it is better to present a reasonable worst-case estimate of risk and avoid potentially underestimating public risks. This latter approach in effect builds a margin of safety for public health into the risk assessment. An argument against this latter approach is that degree of public health protection is a component of risk management rather than risk assessment.

- ***The third bar in Figure 9 illustrates how the choice of background (also called naturally occurring) levels of pollution impacts risk estimates.*** In the Israel assessment, risks associated with both total pollution and man-made pollution were evaluated. To estimate the risks associated with man-made pollution, an estimate of background is deducted from the exposure estimate prior to estimating risks. The "true" background level is difficult to determine. It varies with location, seasons, weather conditions, etc. Additionally, in Israel, few monitors are located in areas completely unaffected by man-made pollution. How the background levels used in the Israel assessment were determined is discussed in Appendix B. What is presented in the third bar of Figure 9 are three concentrations of $PM_{2.5}$ that were considered reasonable for use as background pollutant concentration levels, and how these choices impact the estimates of risk.

- ***The fourth bar in Figure 9 illustrates how the three individual uncertainties discussed above may interact to further increase overall uncertainty and impact risk estimates.*** The fourth bar illustrates the risk estimates if one uses reasonable alternatives from each of the preceding individual examples and combines them in a manner that emphasizes the

maximum and minimum estimates of risk that could be calculated using alternative, reasonable assumptions. It can be seen from this simple example that impact on estimated risks can be large.

Each individual uncertainty evaluated could account for a two-fold difference in the risk estimates, while the combined uncertainties could make a ten-fold difference in the risk estimates. When the possible impact of all uncertainties is considered, the difference between estimated and actual risk could be significantly greater. Ideally, a more complex analysis of all of the important uncertainties for each pollutant and health effect would be conducted and presented as an integral part of the risk assessment. Such an uncertainty assessment was outside the scope of this effort. Additional details of this analysis are presented in Appendix C: Health Assessment.

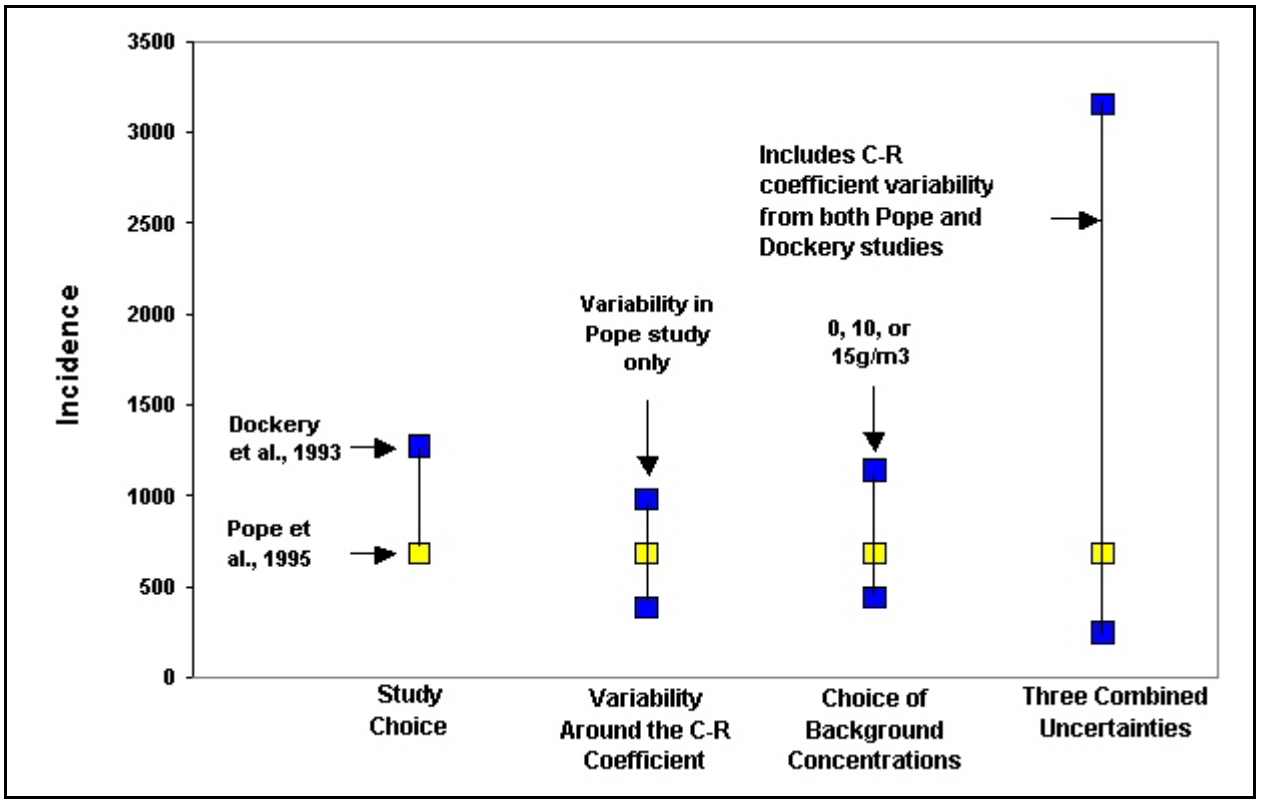

Figure 9: Effects of Several Uncertainties on Mortality Risk Associated With Long-Term $PM_{2.5}$ Exposures in Greater Tel Aviv - 1997
Yellow (light colored) squares represent the data used in this risk assessment; blue/purple (dark colored) squares represent reasonable, alternative choices.

A Comparative Assessment of Air Pollution Public Health Risks in Two Israeli Metropolitan Areas: 1995-1999

5. CONCLUSIONS AND RECOMMENDATIONS

Conclusions

The comparative risk assessment suggests significant public health risks are posed by air pollution in Israel. The assessment focused on the Tel Aviv and Ashdod regions, with a combined population of approximately 1.2 million people. Four pollutants were evaluated: PM, O_3, SO_2, and NO_2. Risks were estimated for mortality, hospital admissions, and selected less severe health effects. Important conclusions are:

1. Estimated risks in Greater Tel Aviv, attributable to the evaluated air pollutants, are hundreds of additional deaths per year, thousands of additional hospital admissions, and tens of thousands of less severe health effects. Risk levels in Ashdod are generally similar, but the incidence (number of cases) is approximately five-fold lower due to Ashdod's smaller population.

2. Significant risks are posed by exposures below current Israeli ambient air quality standards.

3. Both short- and long-duration exposures appear to produce health effects.

4. At particular risk are individuals older than 65, children, and individuals with pre-existing diseases.

5. There are some differences in apparent risks between the two regions. The basis for these differences is unclear but may result from differences in amounts and sources of pollution, with concomitant differences in population exposures. Major sources of air pollution in Tel Aviv and Ashdod are transportation and power plant emissions. Ashdod sources include a petrochemical facility, metal recycling industries, agriculture-related industries, and a large deep-water port.

6. Pollutants of concern in descending order are: PM, O_3, SO_2, and NO_2. Concern is substantially greater for PM and O_3 based on the magnitude of the relative risks, the number of health effects associated with each pollutant, and the degree of confidence in the underlying data.

The risk assessment focuses on health effects with human evidence linking the pollutant in question to the health effects of concern. The evidence for this association comes from multiple sources that are consistent and coherent in their findings,[16] suggesting a causal role for ambient pollution in contributing toadverse health effects. It is possible, however, that a pollutant thought to be the causative agent might be acting as a surrogate for an unevaluated pollutant or pollutants, or other factors that are the true causative agents. Alternatively, it has been suggested that air pollutant effects are caused by exposure to the mixture of pollutants and that risks cannot be adequately apportioned on a pollutant-by-pollutant basis. Current methodologies do not allow for resolution of these issues. Overall the health risks to individuals from air pollution are relatively small, the overall public health consequences here appear considerable.

Despite the consistency and coherency in the data with respect to health effects, significant uncertainties exist. The major uncertainties associated with the risk analyses are highlighted. Due to the many sources of uncertainty in the analyses presented here, the risk estimates should not be interpreted as precise measures of risk. The risk assessment can, however, provide useful information relevant to evaluating research priorities and risk management options.

[16] In other words, the majority of studies are in agreement in terms of what health effects are associated with specific pollutants and the approximate magnitude of the responses (consistent). In addition, there is a logical link between the various health outcomes and what is known about the underlying biological mechanism (coherent). These two attributes greatly increase the level of confidence in the data.

Recommendations

The joint working group makes the following recommendations:

1. Address $PM_{2.5}$ pollution and mitigation measures in the Tel Aviv and Ashdod regions to reduce $PM_{2.5}$ emissions from transportation and industry.

2. Address O_3 pollution and mitigation measures in the Tel Aviv and Ashdod regions to reduce O_3 precursors (i.e., volatile organic compounds and nitrogen oxides).

3. Evaluate year to year variations in monitored ambient concentrations. Trend analyses should be conducted to determine if pollution levels are increasing over time. Ashdod warrants particular attention.

4. Apply successful mitigation measures to other geographic areas of Israel with similar pollution levels.

5. Initiate public-health-based mitigation strategies, e.g., public health warnings for high pollution episodes.

6. Improve source apportionment for $PM_{2.5}$ and ozone precursors.

In addition, the workgroup recommends new data gathering and analyses to:

7. Expand monitoring for $PM_{2.5}$ and other important air pollutants for which monitoring data are not available, such as carbon monoxide, lead, and other air toxics.

8. Estimate indoor ozone concentrations and personal exposures (e.g., time-activity analyzes) to reduce the uncertainty associated with ozone risk estimates.

9. Consider the need for additional Israel-specific health data.

10. Update this assessment as new information warrants.

APPENDIX A:

REGULATION AND MONITORING OF AIR POLLUTION IN ISRAEL

Contents

This appendix provides a brief overview of Israel's current air pollution laws and regulations, the Israel Ministry of the Environment's air quality program, and Israel's ambient monitoring infrastructure. Emissions estimates and summaries of air quality data for the Tel Aviv and Ashdod study areas are found in Appendix B: Exposure Assessment.

Israel's Laws and Regulations

Israel employs a variety of national, regional, local, and facility-specific legal and policy tools for regulating air pollution. The main law for air pollution control in Israel is the Abatement of Nuisances Law (1961).[1] The law authorizes the Minister of the Environment to promulgate regulations defining what constitutes "unreasonable air pollution," e.g., ambient standards, and also empowers the Minister to issue "personal decrees." These personal decrees can be issued against specific polluters to require compliance with emissions standards, monitoring, or any other action necessary to reduce the nuisance. For example, some of Israel's power generation stations that are significant sources of air pollution are required, through personal decrees, to use low sulfur content fuels and other air pollution control measures. The Abatement of Nuisances Law also requires that any permit issued by any Israeli government authority (i.e., national or local) be in compliance with regulations promulgated pursuant to this law.

Ambient air quality standards were first issued under the Abatement of Nuisances Law in 1971 and revised in 1992.[2] The regulations provide maximum and annual and/or hourly average ambient air concentration levels for Ozone (O_3), sulphur dioxide (SO_2), carbon monoxide (CO), nitrogen oxides (NO_x), sulphate ($SO_{4)}$), settling dust, phosphate, suspended particulate matter, respirable particulate matter (PM_{10}), three metals in particulate matter, and eight air toxins.[3] The Israel ambient standards for SO_2, NO_2, CO, O_3, PM_{10}, and $PM_{2.5}$ considered in this study are shown in Table A-1, together with standards and guidelines from other states and the World Heath Organization (WHO).

[1] Israel Ministry of Foreign Affairs, 1994. "Abatement of Nuisances Law, 1961 (English Translation)," http://www.israel.org/mfa/go.asp?MFAH0aww0.

[2] Gabbay, S., 1998. "The Environment in Israel," Israel Ministry of the Environment, Jerusalem, Israel, 85-86.

[3] Israel Union for Environmental Defense, 1993. "Israel's Environmental Laws," Israel Ministry of the Environment, Jerusalem, Israel, C6-C8.

Table A-1: Ambient Air Quality Standards and Guidelines in Israel and Elsewhere*

Pollutants	Duration	Israel	USA - EPA	USA - California	WHO	Germany	Europe
SO_2 µg/m^3	½ hour	500				1000	
	Hour			655			350
	24 hour	280	365	105	125	300	125
	Year	60	80		50	140	
NO µg/m^3	½ hour	940 for NO_x				200	
	Hour			470	200		200
	24 hour	560 for NO_x				100	
	Year		100		40	80	40
CO mg/m^3	½ hour	60			60	50	
	Hour		40	23	30		10
	8 hour	11	10	10	10		
	Year					10	
O_3 µg/m^3	½ hour	230				120	
	Hour		235	180			180
	8 hour	160	157		120		
	Year					50	
PM_{10} µg/m^3	24 hour	150	150	50	**	200	50
	Year	60	50	20	**	100	30
$PM_{2.5}$ µg/m^3	24 hour		65	65			
	Year		15	12			

Conversion factors: 1 mg/m^3 SO_2=0.38 ppb; 1 mg/m^3 NO_2=0.53 ppb; 1 mg/m^3 CO=0.87 ppm; 1mg/m^3 O_3=0.51 ppb

* It should be noted that different countries have different forms of the standards, thereby affecting their actual stringency.

** WHO guidelines are set at concentrations below which no or little public health risks are thought to occur. All concentrations evaluated to date for particulate matter appear to pose some risk to public health. Consequently, no WHO guideline value for particulate matter has been set.

Additional regulations under the Abatement of Nuisances Law include a prohibition of black smoke emissions from facilities (1962) and vehicles (1963), a prohibition on the use of heavy fuel oil for heating systems (1972), regulations preventing "unreasonable air and smell pollution" from solid waste disposal facilities (1990), and regulation of dust emissions and corrective measures from quarries (1998).[4] In addition to the Abatement of Nuisances Law, Israel has several other laws for controlling air pollution. The Licensing of Business Law (1968) allows for the provision of special environmental conditions with business licenses (e.g., emission/discharge standards) for individual facilities. The Operation of Vehicles (Engines and Fuel) Law (1960) authorizes the Minister of Finance, in consultation with Minister of Transportation, to regulate fuel and engine types. The Planning and Building Law (1965) requires an Environmental Impact Statement under regulations developed in 1982 for all new buildings and land use. This law has been used successfully to restrict emissions from planned installations by requiring best available technology-based emissions standards[5].

The Ministry of Transportation's regulations under the Traffic-Ordinance (1961) prohibit registering vehicles that do not conform with emission standards. Gasoline vehicles must be tested annually for compliance with CO emissions standards. Diesel vehicle exhausts are tested for opacity. The Ministry of the Environment also conducts spot checks of vehicles' compliance with emission standards. The Public Health Ordinance

[4] Gabbay, S., 1998. "The Environment in Israel," Israel Ministry of the Environment, Jerusalem, Israel, 86.

[5] Israel Ministry of Foreign Affairs, 1999, Israel's Air Resources Management Program, http://www.israel.org/mfa/go.asp?MFAH00ib0.

(1940) authorizes the Ministries of Health and the Environment to implement measures to prevent environmental nuisances affecting public health. At a local level, municipalities and associations of towns have enacted bylaws for pollution prevention and for collecting monitoring fees.[6] A significant milestone was the enactment of the 1996 amendments to the Prevention of Environmental Nuisances (Civil Action) Law (1992) which provide legal standing for citizens and citizens' environmental groups against environmental hazards.[7]

Israel Ministry of the Environment's Air Quality Program

Despite these laws, air quality remains a significant problem in Israel. In 1994, "to bring about significant improvements in air pollution abatement and prevention," the Ministry of the Environment began implementing a "comprehensive new program for the management of air resources."[8] The main components of the Air Resources Management Program are:

- Prevention of air pollution through the integration of environmental considerations and physical planning, monitoring, and intermittent control systems.
- Legislation and enforcement, including ambient and emission standards.
- Improvement of fuel quality.
- Research.
- International cooperation.
- Individual treatment of pollution sources.
- Reduction of pollutant emissions from motor vehicles.

In addition, the Ministry of the Environment has considered developing a comprehensive Clean Air Act to provide additional statutory tools to improve air quality.

The initial intent of Israel's air resources program was to focus on three major areas: developing emission standards for stationary sources, reducing mobile source emissions, and deploying an air quality monitoring network. The Ministry of the Environment drafted and proposed strict stationary source emissions standards in 1994, based on the German TA Luft guidelines and U.S. EPA 40 CFR 60 regulations.[9] The Ministry of the Environment intended to promulgate these standards in 1996, but they still remain a draft today. Instead, on January 21, 1998, the Ministry of the Environment entered into a "covenant on air pollution abatement" with the Manufacturers Association of Israel.[10] The covenant provides a framework for cooperative implementation of the draft emissions standards according to an established time frame.

To curb vehicular emissions, beginning in 1994 the Ministry of Transportation required catalytic converters on all imported gasoline-powered motor vehicles (i.e., all new motor vehicles, since Israel does not manufacture automobiles). In addition, the Ministry of Infrastructure required a gradual phase-out of lead from gasoline, planned to be completed in 2003.

Israel's Ambient Air Quality Monitoring Network

The State of Israel faces air quality challenges similar to those of most of the developed countries in the world. Some of the problems are even more intense in Israel as a result of its unique geographic and economic features. Most of the country's economic activities take place in a narrow strip along the Mediterranean coast which extends 65 km from Ashdod in the south to Netanya in the north and 10 km from the coast line eastwards. About 2.6 million people (approximately half of Israel's population) live in this densely populated area of 650 km^2, with a standard of living that is generally higher than that of the rest of the country. In addition, more than 60 percent of the total number of vehicles (more than 1,600,000) travel in this area each day and most of the energy production facilities of Israel (five major power plants with total capacity of 7000 MW) are found here. Therefore, most of Israel's air pollution

[6] Gabbay, S., 1998. "The Environment in Israel," Israel Ministry of the Environment, Jerusalem, Israel, 86-87.

[7] Israel Ministry of the Environment, 1998. Environmental Legislation and Enforcement, http://www.environment.gov.il/Eng-site/Legislation_and_Enforcement.html.

[8] Israel Ministry of Foreign Affairs, 1999. Israel's Air Resources Management Program, http://www.israel.org/mfa/go.asp?MFA00ib0.

[9] Graber, M., Summer 1995. Transition to Clean Technologies Through Implementation of Emission Standards, "Israel Environment Bulletin," Israel Ministry of the Environment, Vol. 18, No. 3, 21-24.

[10] Israel Ministry of the Environment, Winter 1998. Covenant on Air Pollution Abatement, "Israel Environment Bulletin," Vol. 21, No. 1, 8-12.

emissions occur in a very small area. Moreover, the most frequent trade winds and the afternoon sea breeze are westerly, transporting primary and secondary air pollution eastward (inland).

Until recently, air quality monitoring in Israel was carried out locally, operated by municipalities, associations of towns for environmental protection, and the Israel Electric Company (IEC). Five separate networks exist that monitor power plants, ensuring adherence to SO_2 ambient air quality standards. They also have an alert and response system to enable switching to low sulfur fuel oil during poor dispersion conditions. To tackle additional air quality problems such as transportation NO_x emissions and photochemical smog formation and transport, the Israeli Ministry of the Environment decided to establish a national air quality monitoring network, or MANA (acronym transliterated from Hebrew).

The existing networks in the following cities are operated by associations of towns for environmental protection: Haifa, Hadera, Ashdod, and Ashkelon. The IEC operates its network in these regions as well as in the Tel Aviv metropolitan area. In addition to this primarily population-oriented monitoring, source-oriented monitoring is carried out by the IEC at small power plant gas turbines and by the Ports and Rails Authority in the Eilat port. MANA stations are mainly population-oriented, but there are transportation-oriented stations located at a pedestrian level on the main streets in some cities. Figure A-1 shows a map of the MANA monitoring stations and networks.

Figure A-1: Map showing Israel Ministry of the Environment Ambient Air Quality Monitoring Network (MANA)

APPENDIX B:

ASSESSMENT OF EXPOSURES TO AMBIENT CONCENTRATIONS

Contents

Tables

Figures

The U.S. National Research Council identifies four steps for human health risk assessment: hazard identification, concentration-response assessment, exposure assessment, and risk characterization.[1] This appendix describes the specific approach used for conducting the exposure assessment for air pollution in the Tel Aviv and Ashdod study areas. Exposure assessment involves specifying the population(s) that might be exposed to the agent(s) of concern, identifying the routes through which exposures can occur (e.g., air, food, or drinking water), and estimating the magnitude, duration, and timing of the doses that people might receive as a result of their exposures.[2] As described previously in this report, personal or population exposures are assumed to be the same as ambient pollutants concentrations measured at fixed-site monitors. In other words, to maintain consistency with available air pollution epidemiology data, ambient concentrations are used as surrogates for actual personal exposures.

The selection of pollutants for this study, particulate matter (PM), ozone (O_3), sulfur dioxide (SO_2), and nitrogen dioxide (NO_2), was based, in large part, on concerns raised by the results of ambient air quality data. These data were developed through continuous ambient air monitoring that has been conducted for the past 20 years by means of monitoring networks located in the study areas of Greater Tel Aviv and Ashdod. Unlike some other comparative risk assessments, sophisticated modeling of monitored data was not necessary because of the extensive ambient air quality monitoring systems operating within the study areas.[3]

This appendix provides information about the sources (Source Characterization) and emissions (Emission Inventory Estimations) of these pollutants, the monitoring of their concentrations in the ambient air of the study areas (Ambient Air Quality Monitoring), and a discussion about the exposure data, including uncertainties (Exposure to Air Pollution in Tel Aviv and Ashdod).

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[1] National Research Council (1994) pages 26-27.

[2] ibid.

[3] For an example of such modeling, see USEPA, 1997.

SOURCE CHARACTERIZATION

Sources of pollution in the study areas include:

- A 528 megawatt and a 1,200 megawatt low sulfur, heavy-fuel-oil-fired power plant located on the coastline in North Tel Aviv and North Ashdod, respectively;
- A large refinery located near the power plant in North Ashdod;
- Numerous medium and small high sulfur heavy-oil-fired industrial boilers serving a variety of industrial processes in both areas;
- A large urban street network in Tel Aviv through which an estimated 500,000 vehicles travel each day;
- A complex transportation network in Ashdod, involving automobiles, many heavy trucks, and a seaport.

The main pollutants emitted from the power plants are SO_2, NO_x, and PM resulting from combustion of heavy fuel oil. The main emissions from vehicles are carbon monoxide (CO), NO_x, PM, SO_2, and organic compounds. As of 1993, all new gasoline cars imported to Israel must be equipped with catalytic converters, which leads to lower emissions of CO, NO_x, and organics.

Ashdod

In addition to automobile traffic, sources of Ashdod air pollution include truck transportation, a power plant, a petrochemical facility, metal recycling industries, agriculture-related industries, and a large deep-water port on the Mediterranean Sea.

Stationary Sources: The most significant emission source is the 1,200 MW "Eshkol" power plant, consuming around 1.2 million tons of various quality (0.5, 1 and 2 percent sulfur) heavy oil fuel per year. The second largest source is the oil refinery, which consumes heavy oil fuel and fuel oil gas. A major fraction of the pollutants from the refinery is emitted during the process of production and not as a result of fuel consumption. There are also numerous smaller sources, such as Agan Chemicals, asphalt and concrete batch plants, and a variety of other factories, some of which have combustion units. To the east of the city there is another smaller industrial zone, occupied by a few factories with combustion units. In addition to the criteria pollutants, these two industrial zones emit heavy metals and organic compounds.

The power station's stacks are about 150 meters high; the refineries' stacks are about 80 meters high; and all the other stacks are lower. The fact that major emissions are released high above the city, combined with the local meteorology, greatly influences the dispersion of the plumes and the ambient pollutant concentrations detected at the monitoring stations. For example, the highest concentrations of SO_2 were detected in the Nir Galim area, located southeast of the northern industrial zone and downwind of the major stacks in the primary wind direction.

Mobile Sources: Approximately 100,000 private cars and 5,000 trucks and heavy trucks are registered in Ashdod and its surroundings. Traffic is augmented by a major road junction between the northern and southern parts of the country located in the study area. A major south-north transportation route (Road No. 4) is located about 10 km east of Ashdod, and an east-west road (No. 41) connects the Gedera area to Ashdod. A main route leads from the junction of these roads to the city of Ashdod and to a national cargo seaport. Trucks use this road heavily. Ships, working vehicles, and the port itself emit particulate matter and combustion-related pollutants (SO_2, NO_x, and CO).

Greater Tel Aviv

The main sources of air pollution emissions in Tel Aviv are mobile sources (automobiles, buses, two-wheeled vehicles), the Reading Power Plant, and a number of small industrial facilities and factories.

Stationary Sources: Located on the coast in the northern section of the city at the mouth of the Yarkon River, the 528-megawatt Reading Power Plant is the dominant stationary source in Tel Aviv. The plant burns heavy residual [#6] oil. Since 1993, the maximum sulfur content of fuel oil burned in the plant has not exceeded 1 percent. This has resulted in a marked improvement in SO_2 air quality.

Mobile Sources: Each day, an estimated 460,000 motor vehicles travel within the metropolitan area of Tel Aviv and its neighboring cities. Of these, 70,000 vehicles are diesel, including all of the region's taxi, bus, and shuttle bus fleets. Given the long hours of operation of public transport vehicles, their contribution to air pollution in the region is significantly higher than the average vehicle.

EMISSION INVENTORY ESTIMATES

Emission estimates and air quality monitoring results have contributed to concerns about air pollution in Israel. Estimated emissions of selected air pollutants show a variety of patterns (Figure B-1). Emissions trends in Israel are influenced by various factors, including rapid population growth and changing consumption patterns, increasing electrical demand and production, lowering the sulfur content of fuel oil consumed by power stations, increasing numbers of vehicles and miles traveled, the introduction of catalytic converters, and the introduction of private diesel vehicles. As a result of these influences, the relationship between emissions, air quality, and human health risk is not straightforward.

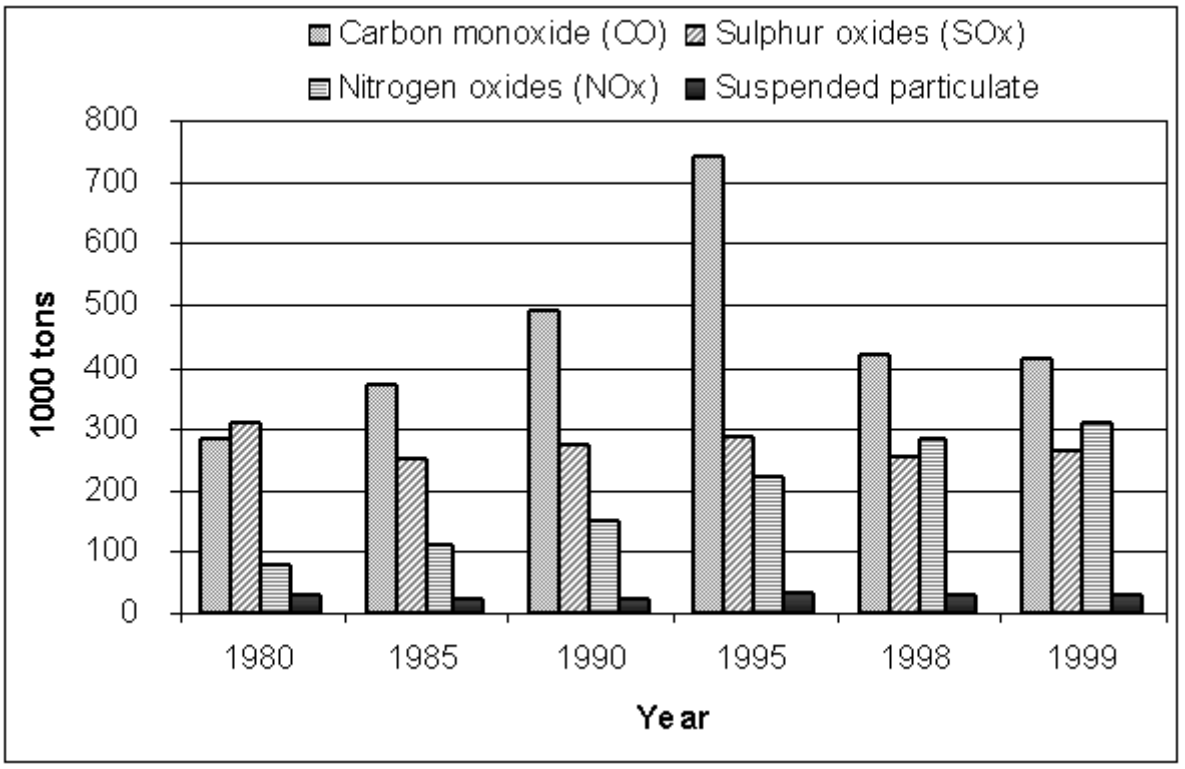

Figure B1: Estimated Annual Emissions of CO, SO_x, NO_x, and Suspended Particles in Israel
The chart illustrates the estimated annual emissions in Israel from 1980-1999. Data are from the Israel Bureau of Statistics, 2001, Statistical Abstracts of Israel 2000, http://www.cbs.gov.il/shnanton51.

Estimated inventories of daily emissions from stationary and mobile sources in Ashdod and Tel Aviv are shown in Tables B-1 and B-2, respectively.

Table B-1: Estimated Inventory of Daily Emissions in Ashdod (ton/day)

Sources	PM	SO_2	NO_x	CO
Stationary				
Power Plant	5.5	75	85	NA
Industry (including refineries)	0.8	28	6	NA
Mobile	0.6	0.6	2.6	50

Table B-2: Estimated Inventory of Daily Emissions in Greater Tel Aviv (ton/day)

Sources	PM	SO_2	NO_x	CO
Stationary				
Power Plant	4.3	31.7	9	1
Industry	0.1	1.8	0.1	0.02
Mobile	2.1	0.4	12.3	265

As illustrated in Tables B-1 and B-2, industrial sources are thought to be responsible for the majority of air pollution emissions in the Ashdod area. In Tel Aviv, mobile sources likely contribute the largest amount of NO_x and CO, while the power plant is considered the major pollution source for SO_2 and PM. Calculations of stationary-source emission estimates for the Ashdod study area are based on stack measurements using data from the year 2000, and the largest factories were taken into account. Emissions from stationary sources in Tel Aviv are estimated based on stack measurements and fuel consumption.

Mobile-source emission estimates for Ashdod are based on a municipal car registration list and assumptions made for the average distance traveled per vehicle. Mobile source emission estimates for Tel Aviv are based on Israel Central Bureau of Statistics (CBS) 1996 survey data concerning commuting habits within the Tel Aviv metropolitan area, emission factors given by the Israel Ministry of Environment, and calculations made by the Tel Aviv Municipality. In determining emission factors, low speed driving was assumed for both areas and year-group. Data were combined according to the data available for the vehicle fleet age distribution. Table B-3 shows the calculated emission factor for private cars in Tel Aviv and Ashdod. Table B-4 shows the emission factors calculated for diesel vehicles.

Table B-3: Emission Factors Used for Private (Gasoline) Cars (g/km)*

Age Group	Ashdod		Tel Aviv				
	NO_x	CO	NO_x	CO	SO_2	TSP	HC
1993 and on	0.11	15.50	0.11	15.50	0.03	0.01	3.50
1988 - 1992	* 0.80	* 50	1.08	37	0.03	0.01	3.50
Before 1988	* 0.80	* 50	0.80	50	0.03	0.01	3.50

* In Ashdod, data were not available to differentiate between car ages prior to1992.

Table B-4: Emission Factors Used for Diesel Vehicles (g/km)*

Vehicle Class	NO_x	PM	CO	SO_2	HC
Trucks up to 4 tons	1.43	0.3	1.17	0.08	0.38
Trucks above 4 tons	16.9	1.18	3.51	0.25	2.34
Taxi	0.72	0.27	0.89	0.06	0.23
Bus	20	1.87	6.25	0.24	4.87
Motorcycle > 50 cm^3	0.3	0.13	20	< 0.01	6.08
Motorcycle < 50 cm^3	0.05	0.04	10	0.01	3.15

* No professional travel was accounted for in the CBS survey, so trucks above 4 tons were not represented in the calculations, and all trucks were assumed to be small.

METEOROLOGY

Meteorology is one of the most important factors influencing air pollution and driving atmospheric dispersion patterns. The Greater Tel Aviv and Ashdod areas are located on the coastline of the Mediterranean Sea, so they are strongly influenced by the sea breeze. In Ashdod, the primary wind directions (annually) are north to northwest during the day and east to southeast during the night. In Tel Aviv, the primary wind directions are west to northwest during the day and east to southeast during the night. Boundary layer depth is variable and sometimes bounded by ground inversion, a situation that leads to high pollutant concentrations near the ground, especially during the cold winter months.

AMBIENT AIR QUALITY MONITORING IN TEL AVIV AND ASHOD

The Ministry of the Environment, the Israel Electric Company, and the local environmental agencies operate ambient air quality monitoring networks in the Tel Aviv and Ashdod study areas. The monitoring stations that make up these networks, locations of the stations, and the pollutants monitored are summarized in Table B-5 and illustrated in Figures B-2 and B-3.

Table B-5: Monitoring Networks in Tel Aviv and Ashdod (X indicates parameter is monitored)

No.	Station Name	City	O_3	TSP	PM_{10}	$PM_{2.5}$	NO_2	SO_2	CO	Wind Speed and Direction
Tel Aviv Area										
1	Yad Lebanim	Ramat Gan			X	X		X		
2	Shikun Bavli	Tel Aviv						X		
3	Mechabei Esh	Ramat Gan	X				X	X		X
4	Bitzaron	TA						X		X
5	Bnei Beraq	Bnei Beraq						X		
6	Tahana Merkazit	Tel Aviv	X		X	X	X	X		X
7	Antokolski	Tel Aviv	X			X	X	X		
8	Tel Baruch	Tel Aviv						X		X
9	Shikun Lamed	Tel Aviv			X			X		
10	Tipat Halav	Tel Aviv					X		X	
11	Amiel*	Tel Aviv					X		X	
12	Ironi Dalet*	Tel Aviv					X		X	
13	Ironi TZ*	Tel Aviv					X		X	
14	Yad Avner*	Tel Aviv	X		X		X	X	X	X
15	Remez*	Benei Beraq					X		X	
Ashdod Area										
1	Egude	Ashdod	X			X	X	X		X
2	Gan-Yavne	Gan Yavne					X	X		X
3	Gedera	Gedera	X				X	X		X
4	Hevel-Yavne	Hevel-Yavne					X	X		X
5	Benaya	Benaya					X	X		X
6	Rova Yud	Ashdod	X			X	X	X		X
7	Rova YA	Ashdod	X				X	X		X
8	Ner-Galim	Ner-Galim						X		
9	Gan-Darom	Gan-Darom						X		
10	Kvzat-Yavne	Kvzat-Yavne	X				X	X		X
11	Kfar-Aviv	Kfar-Aviv						X		
12	Yavne	Yavne	X		X		X	X		X
13	Yesodot	Yesodot		X						

*Station began operation in 1999

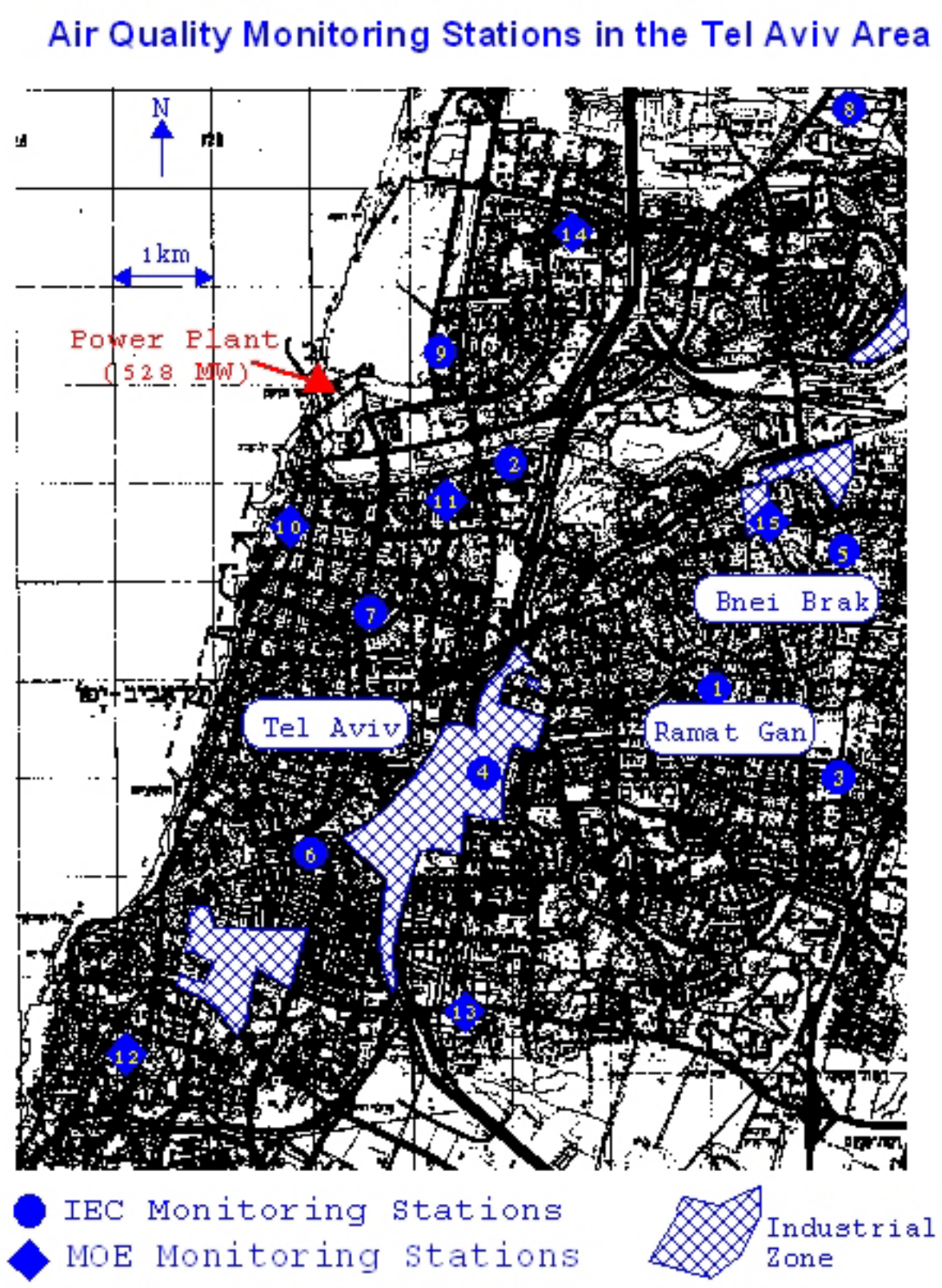

Figure B-2: Map Showing Tel Aviv Study Area
The Tel Aviv study area includes the municipalities of Tel Aviv, Benei Brak, Halon, Givatayim, and Ramat Gan. Also shown are the locations of major stationary emission sources (power plant and industrial zones), major traffic arteries, and ambient air monitoring stations operated by the Israel Electric Company and the Israel Ministry of the Environment.

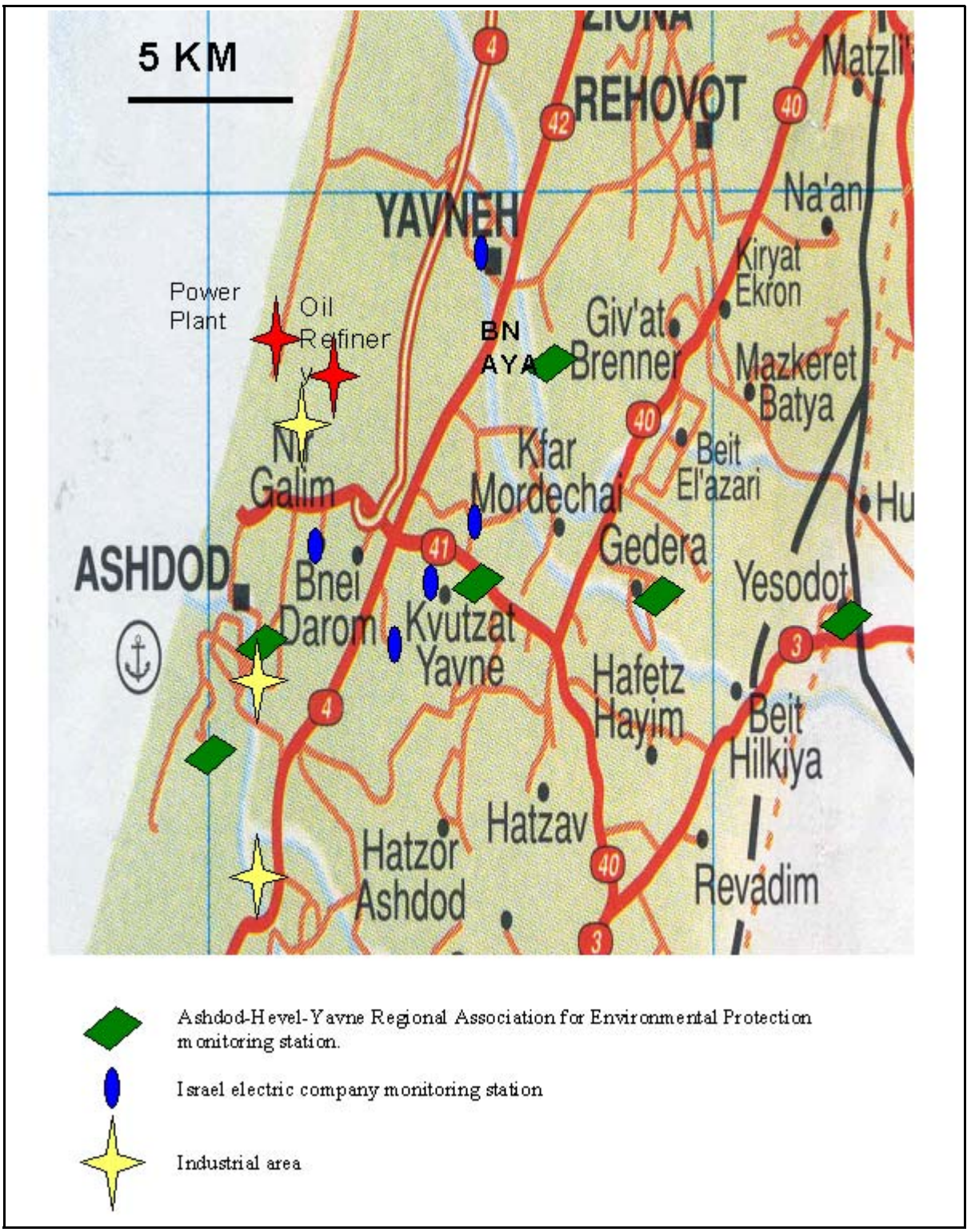

Figure B-3: Map Showing Ashdod Study Area
The Ashdod study area includes the city of Ashdod and the towns of Bnaya, Gedera, and Gan-Yavne. Also shown are the locations of major stationary emission sources (sea port, oil refinery, power plant and industrial areas), major traffic arteries, and ambient air monitoring stations operated by the Ashdod-Havel-Yavne Regional Association for Environmental Protection and the Israel Electric Company.

Ambient air monitors, complying with standard U.S. EPA ambient monitoring methods, are located in each of the monitoring stations listed in Table B-5 and continuously monitor the concentrations of the pollutants indicated. Except for stations 10 to15, all stations were in operation during the 1995-1999 study period. All stations operating during the study period were located at a height of about 10 meters above ground; therefore, the measurements reflect the general air quality in the area and are not directly influenced by traffic emissions in the immediate vicinity of the station.

Measurement data are recorded and stored each half-hour by instrumentation connected to the monitors. Data are then transferred by telecommunications systems to computer terminals located at the Israel Ministry of Environment monitoring centers in Ramle and Tel Aviv, at the Tel Aviv Municipality environmental department, and at the offices of the Ashdod-Havel-Yavne Regional Association for Environmental Protection. The data are processed by these agencies to obtain averaged data in the formats required for comparison to ambient air quality standards.

Particulate Matter

Figure B-4 shows the ambient concentration of particulate matter less than 10 microns aerodynamic diameter (PM_{10}) in several Israel cities. Concentrations consistently approach or exceed the Israel annual ambient standard of 60 µg/m^3 for PM_{10}, although there are relatively few exceedences of the 150 µg/m^3 daily standard. A key factor in the dispersion of particulate matter in the region is the meteorological systems that create dust storms originating in the deserts of North Africa and the Arabian Peninsula.

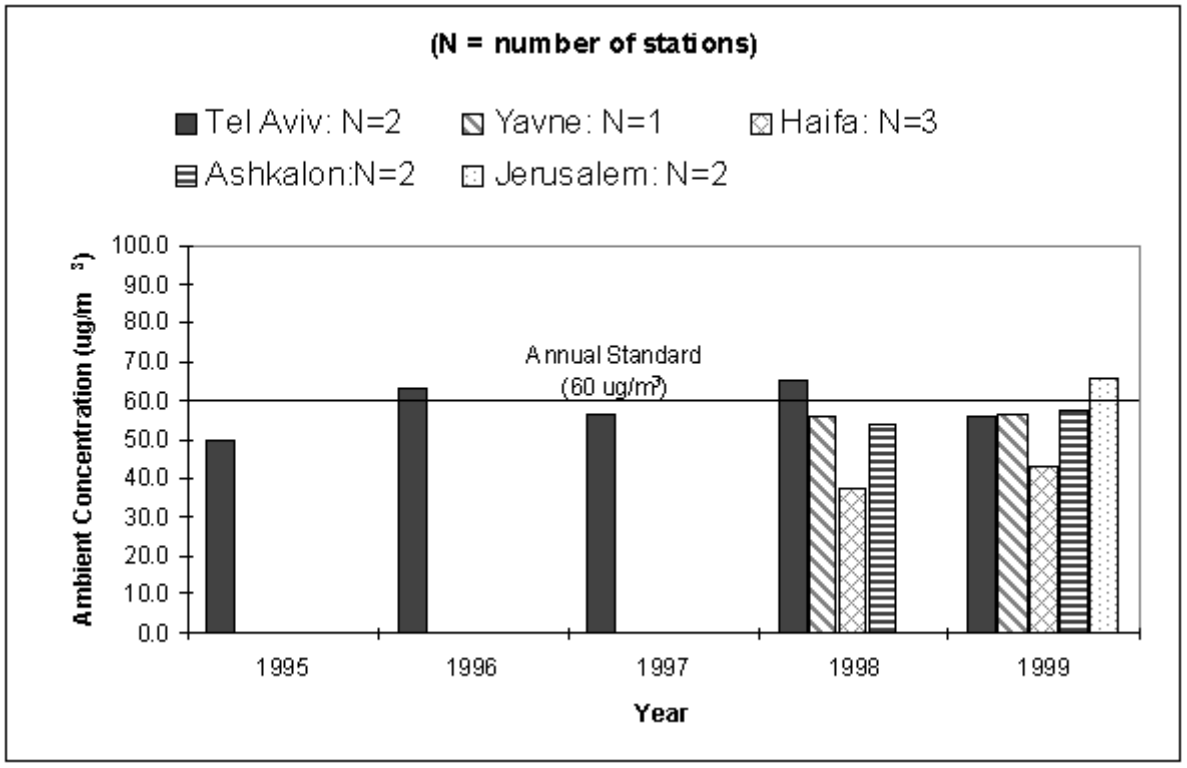

Figure B-4: Annual Average PM_{10} Concentrations from Ambient Air Quality Monitoring Stations in Israeli Cities
Tel Aviv is located in the central coastal plain, Haifa on the north coast, Ashkelon on the south coast, Jerusalem in the eastern central region, and Yavne about 30 km southeast of Tel Aviv. With the exception of Yavne, the annual average concentration is calculated from the mean of multiple stations (N) located within the area. Annual averages for 1995-1997 are only available for Tel Aviv. Data are from Israel MoE.

Nitrogen Oxides

The majority of the exceedences of the Israel nitrogen oxide (NO_x) standards are thought to be due to transportation emissions. As shown in Figure B-5, the 940 µg/m^3 half-hour standard can be exceeded several hundreds of times per year at Israel Electric Company (IEC) monitoring stations in the Tel Aviv metropolitan area. The NO_x concentrations to which motorists and pedestrians are exposed are several times greater than those to which the general public is exposed. These higher exposures are indicated by the number of times the half-hour NO_x standard is exceeded at MANA** transportation monitoring stations that are located at street level of major traffic arteries. Most of the NO_x exceedences occur during the winter and during the change of seasons.

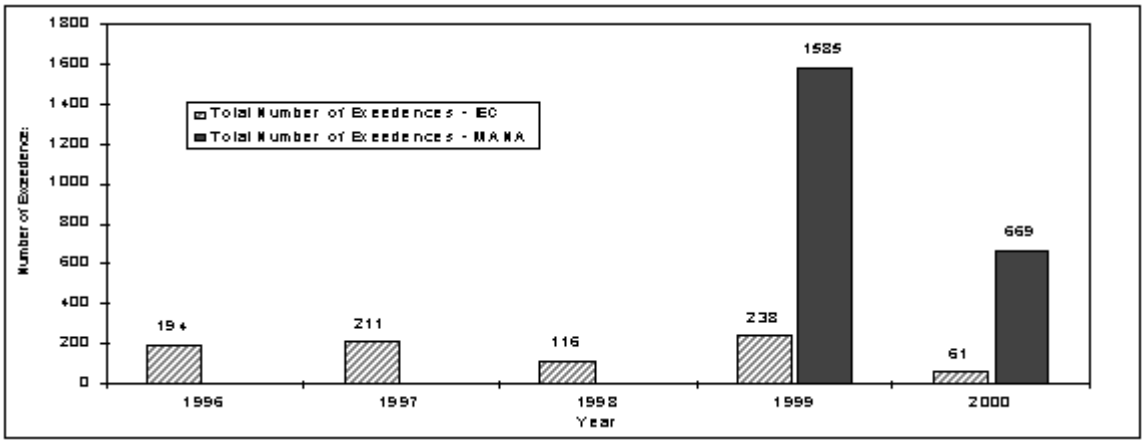

Figure B-5: Exceedences of NO_x Half-Hour Standard in Tel Aviv, Israel
The large number and variability of exceedences of Israel's half-hour NO_x standard are thought to reflect the impact of emissions from transportation (MANA stations located at street level along major traffic arteries) and power generation, as reflected by Israel Electric Company (IEC) stations. Data are from the Israel Ministry of the Environment.

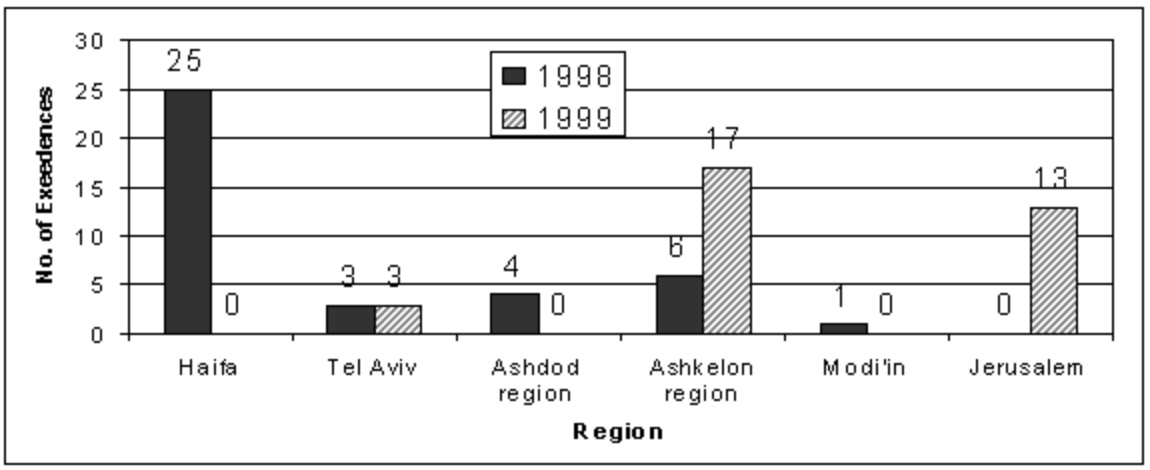

Figure B-6: Total Exceedences of the Half-Hour Israel Standard for Ozone (O_3) from Several Regions in Israel for 1998-1999 Annual (1998-1999) and regional variability in O_3 concentration in Israel are illustrated by differences in the number of times concentrations exceed the Israel half-hour ambient standard. Data are from Israel MoE.

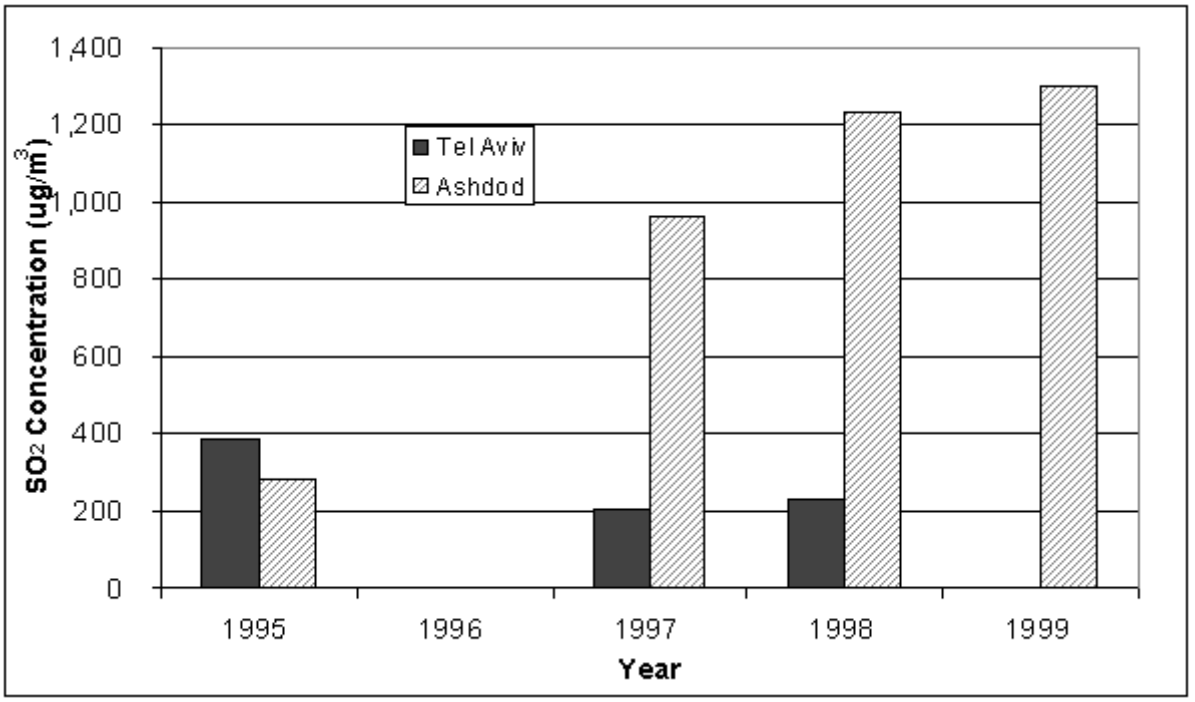

Figure B-7: Annual SO_2 Maximum Half-Hour Concentrations in Tel Aviv and Ashdod, Israel
Ambient annual (1995-1999) maximum half-hour SO_2 concentrations in Tel Aviv and Ashdod are shown. The Israel standard of 500 ug/m^3 is regularly approached or exceeded in Ashdod, but not in Tel Aviv. Data are not available for 1996. Data are from the Israel Bureau of Statistics, 2001, Statistical Abstracts of Israel 2000, http://www.cbs.gov.il/shnaton51.

Ozone

Figure B-6 illustrates the number of exceedences of the Israel half-hour standard of 230 µg/m^3 for ozone (O_3). O_3 is a secondary pollutant that is formed as a result of chemical reactions among pollutants exposed to solar radiation in the atmosphere. O_3 formation is very dependent on local meteorological conditions and, therefore, shows significant annual and seasonal variability. The highest O_3 concentrations occur during the hot summer months when solar radiation is greatest. O_3 also displays regional variability with the highest concentrations often occurring at distances from the sources of the O_3 precursors (e.g., as occurs in Jerusalem), or where local topography provides conditions for entrapped air masses (e.g., Haifa).

Sulfur Dioxide

Figure B-7 shows ambient annual maximum half-hour sulfur dioxide (SO_2) concentrations from monitoring stations in the greater Tel Aviv and Ashdod areas. Due to the lowering of the sulfur content in heavy fuel oil used in power plants and in diesel fuel and the use of emission control technology in coal-operated power plants, SO_2 concentrations in Tel Aviv seldom exceed the standards. Despite these efforts, the SO_2 half-hour standard was exceeded in Ashdod 10 times in both 1998 and 1999.

Exposure to Air Pollution in Tel Aviv and Ashdod

Ambient air quality monitoring data were used to approximate exposure to air pollutants in the Tel Aviv and Ashdod study areas. Summaries of the monitoring results in the study areas of Greater Tel Aviv and Ashdod are presented in Table B-6 as combined averages for all monitoring stations in each study area, for the years 1995 to 1999. Data are only presented for the years in which risks are calculated.

Sources for monitoring data are the Ashdod-Havel-Yavne Regional Association of Towns for Environmental Protection, the Tel Aviv Municipal Environmental Division, and the IEC monitoring stations. Methods for data acquisition differ among systems and regions, and modifications are made on a regular basis. In general, most pollutants are measured every half-hour. These half-hour measurements are calculated to give one-hour and 24-hour averages. In no case where greater than 75 percent of the data were missing or otherwise determined to be questionable was the average concentration calculated. Thus, if 75 percent of the half-hour averages were not measured, neither the hourly nor daily average concentrations were calculated. In addition to missing values, data were also reviewed for bias. For example, in some cases there are "0" values that represent a null. These "0" values were not included in calculations. Every data set for every pollutant, from all stations, for each year, was reviewed to identify gaps and inconsistencies.

Table B-6: Monitoring Results in Greater Tel Aviv Study Area ($\mu g/m^3$)

	O_3 Annual Average	**O_3** Annual Average of 8-hour Daily Maximum	**$PM_{2.5}$*** Annual Average (estimated as 50% of PM_{10})	**PM_{10}** Annual Average	**NO_2** Annual Average	**SO_2** Annual Average	**SO_2** Annual Average of 5-minute Peak (2.5 x hourly)	**SO_2** Annual Average of Daily 1-hour Maximum
Israeli Standard	No Standard	160	No Standard	60	No Standard	60	No Standard	280
US NAAQS				50	100	80		365
Tel Aviv Study Area								
No. of Stations	3	3		2	3	9	9	9
1995	33	Not Measured	24	47	72	20	50	43
1996	35	Not Measured	28	56	73	16	39	35
1997	40	60	28	56	49	15	37	40
1998			32	65				
1999			28	56				
Ashdod Study Area								
No. of Stations								
1995	34	57	Not Measured	Not Measured	18	7	17	25
1996	45	71	Not Measured	Not Measured	28	8	20	26
1997	46	67	Not Measured	Not Measured	27	11	28	42
1998			28	56				
1999			28	56				

* $PM_{2.5}$ concentrations were not measured. They are estimated as 50 percent of PM_{10} concentrations. Although no $PM_{2.5}$ sampling and measurement instruments were available at any of the monitoring stations in either the Tel Aviv or Ashdod regions during 1995 - 1997, concurrent sampling of $PM_{2.5}$ and PM_{10} from stations in Tel Aviv since 2001 support the validity of these estimates.

DISCUSSION OF EXPOSURE MONITORING RESULTS

Ashdod: Average annual O_3 concentrations increased during the 3-year period studied, from 34 µg/m^3 in 1995 to 45 in 1996 and 48 µg/m^3 in 1997. Annual average NO_2 concentrations increased from 18 µg/m3 in 1995 to 28 and 27 µg/m^3, in 1996 and 1997, respectively. This may indicate an increase in emissions from mobile and stationary sources, although differences in meteorological conditions cannot be ruled out as an influencing factor.

Tel Aviv: Annual average concentrations of SO_2, NO_2, and O_3 from the individual monitoring stations were used to create maps of air pollution levels in the Tel Aviv area. From these maps city-wide differences in concentrations could be observed. It was not possible to characterize the spatial distribution for PM_{10} because there were not enough stations measuring PM_{10} to provide significant geographical variation.

In the Tel Aviv area, the highest annual average SO_2 concentrations (16 - 21 µg/m^3) were measured in the southern half of the area, while the concentrations measured in the northern half were about one third lower (10 - 15 µg/m^3). For annual average NO_2 concentrations, the trend was reversed and more moderate, i.e., the concentrations measured in the northern and central areas averaged approximately 52 µg/m^3, while the southern half was 47 µg/m^3. For O_3, the north-south trend was similar to SO_2, i.e., the maximum 1-hour average concentrations measured in the northern and central areas averaged approximately 215 µg/m^3, while in the southern half an average value of 260 µg/m^3 was recorded.

Uncertainties

Representative measurement data were not available for many sections of the areas studied. For risk calculations, the composite average measurements for all the stations were taken as representing the exposure throughout each area. Thus, the various monitored and non-monitored areas were assigned the average value for the entire area, resulting in relatively "non-polluted" areas receiving higher-than-expected exposure levels and relatively "polluted" areas receiving lower-than-expected exposure levels. For NO_2 and O_3, this meant that it was necessary to extrapolate to the entire Tel Aviv area based on measurements performed at only three stations, and for PM_{10} from measurements at only two stations. For Ashdod, only three monitoring sites were available for PM. Thus, the area-wide exposure levels for these pollutants are based on a relatively minimal amount of actual measurement that covers only a small portion of the areas studied.

Instruments for measuring $PM_{2.5}$ were not available at any of the monitoring stations in both the Tel Aviv and Ashdod areas during the study period of 1995 to1999. Thus, $PM_{2.5}$ was not monitored but was calculated based on the assumption that 50 percent of the PM_{10} is $PM_{2.5}$. The assumption was supported by statistical comparison of PM_{10} and $PM_{2.5}$ measurements in Ashdod area. In addition, concurrent monitoring of $PM_{2.5}$ and PM_{10} at stations in Tel Aviv since 2001 support the validity of these estimates.

A substantial uncertainty centers on natural versus man-made sources of pollution. Natural sources contribute to pollution in the atmosphere. This is particularly true for fine and coarse particles. The "background" concentration is defined here as the concentration of a pollutant present in the ambient air that is caused by natural sources. In this document, risks are calculated for both man-made and for combined man-made and background pollution (see Appendix D: Results).

There is considerable uncertainty regarding appropriate background concentration, and this is an important area for subsequent study in Israel. For example, background levels of PM vary by geographic location and season. The natural component of the ambient concentration arises from physical process of the atmosphere (e.g., wind-blown dust) as well as emissions from natural sources (e.g., emissions from vegetation). The exact magnitude of this natural portion for a given geographic location cannot be precisely determined; it is difficult to distinguish from the long-range transport of man-made particles and gases.

The range of expected background concentrations on a short-term basis is much broader. Specific natural events such as wildfires and dust storms can lead to very high levels of PM, even higher than those concentrations observed in polluted urban atmospheres. Disregarding such large and unique events, some estimate of the range of "typical" background on a daily basis can be obtained from reviewing multi-year data and special studies.

In determining background concentrations for this comparative risk assessment, several inputs and possible values were considered, including:

- Minimum levels observed.
- Measurements from remote and relatively unpolluted areas.
- Background levels taken from literature and other countries.
- Lowest 1 and 5 percentiles of measured concentrations.
- Concentrations measured during midnight to 5 a.m. (relatively low production in facilities and low transportation).
- Measurements likely to be influenced from industrial sources were omitted.

In general, background concentrations were determined using best professional judgment and are considered as a best estimate given available information. For the purpose of estimating the background concentration in this study, the lowest short-term concentration (1-hour, except 24 hours for PM) measured in a calendar year was selected as the background concentration, even though it was not necessarily entirely the result of emissions from natural sources. A summary of the background concentrations used in this risk assessment is found in Table B-7.

Table B-7: Background Concentrations

Pollutant	Concentration
PM_{10}	20 µg/m^3
$PM_{2.5}$ (estimated as 50% of PM_{10})	10 µg/m^3
O_3	8 ppb
SO_2	2 ppb
NO_2	5 ppb

APPENDIX C:

HEALTH ASSESSMENT

Contents

Tables

Figure

The U.S. National Research Council identifies four steps for human health risk assessment: hazard identification, concentration-response (C-R) assessment, exposure assessment, and risk characterization.[1] This appendix describes the specific approach used for conducting the hazard identification and C-R assessment for air pollution in the Tel Aviv and Ashdod study areas.

Hazard Identification

Hazard identification entails identification of the pollutants that are suspected to pose health hazards, a description of the specific form of toxicity (such as respiratory toxicity or cancer) that can be caused by the pollutant of concern, and an evaluation of the conditions under which these forms of toxicity might be expressed in exposed humans.[2] Information for this step is typically derived from epidemiological, clinical, and/or animal studies. There is a large body of health information on the air pollutants addressed in this report. The pollutants evaluated in this study are known to cause a variety of health effects in humans at ambient levels. These effects have been observed in many studies and at many locations around the world.[3] Table C-1 identifies human health effects associated with the pollutants and the subset of effects for which risks are quantified.

[1] National Research Council, 1994, pages 26-27.

[2] Ibid.

[3] WHO (1999) page 32; USEPA (1997) pages 6-12.

Table C-1: Human Health Effects of Selected Pollutants

Pollutant	Effects Quantified for the Israeli Project	Additional Effects Quantified Elsewhere	Unquantified	Other Possible Effects
Particulate Matter	Mortality Hospital admissions - respiratory and cardiovascular Respiratory symptoms	ER visits - asthma Worse asthma status Lower and/or upper respiratory illness Restricted activity days Days of work lost	Decreased lung function	Infant mortality Chronic lung diseases other than bronchitis Lung inflammation Cancer
Ozone	Mortality Hospital admissions - respiratory Decreased lung function with symptoms	Hospital admissions - cardiovascular Adult onset of asthma ER visits - asthma Restricted activity days Chronic sinusitis and hay fever	Increased airway responsiveness Lung fibrosis Lung inflammation	Immunologic changes Chronic lung diseases Cancer
Sulfur Dioxide	Hospital admissions - respiratory Increased symptoms in asthmatics	Mortality Hospital admissions - cardiovascular Decreased lung function		Mortality Respiratory symptoms in non-asthmatics
Nitrogen Dioxide	Hospital admissions - respiratory infections	Hospital admissions - cardiovascular & other respiratory Respiratory illness/symptoms	Increased airway responsiveness	Decreased lung function Lung inflammation Immunologic changes

Air Concentrations and Health Responses

Characterizing the relation between air concentrations and health effects is generally called concentration-response (C-R) assessment. C-R assessments entail a further evaluation of the conditions under which the toxic properties of a chemical might be manifested in exposed people. Particular emphasis is placed on the quantitative relation between the exposure concentrations and the toxic response. The development of this relationship may involve the use of mathematical models. The C-R assessment may include an evaluation of variation in response, such as differences in susceptibility between young and old people.[4] An example of a C-R relationship is shown in Figure C-1. This figure illustrates the relation between short-term increases in ozone concentrations and increases in hospital admissions.

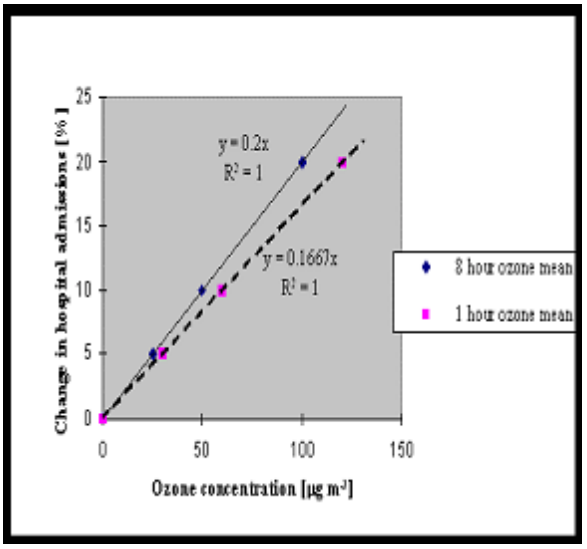

Figure C-1: Increases in Hospital Admissions for Respiratory Conditions as a Function of Ozone Concentration
The equation that calculates the line describing the relative change in hospital admissions as a function of increasing ozone concentration is shown as "y". "R^2" is the correlations coefficient, a measure of how well the line "fits" the data; a "1" is a perfect correlation. Adapted from WHO (1999).

[4] National Research Council (1994) pages 60-66, 206-210.

Selection Criteria

For this project, 13 studies or groups of studies were selected to characterize the relationships of particulate matter (PM), ozone (O_3), sulphur dioxide (SO_2), or nitrogen dioxice (NO_2) concentrations to specific health responses. The seriousness of the health responses evaluated encompasses mortality to respiratory symptoms, such as coughing and wheezing. Each of the 13 studies or groups of studies provides information similar to that seen in Figure C-1, i.e., the quantitative relationship between air pollutant concentrations and health effects. All studies used in this assessment, either epidemiologic or clinical, are based on health effects in humans. The studies were chosen using criteria outlined in Table C-2.

In spite of the wealth of data available for the studied pollutants, the ideal data sets for quantitative comparison of pollutants are not available. Studies often evaluate different subpopulations and/or exposure parameters.[5] Three examples of the difficulties of comparative risk assessment and study selection are provided:

First, at the time this analysis was conducted, there was only one available study of SO_2 and hospital admissions (all respiratory causes) that met our study selection criteria. This study used the daily 1-hour maximum as the exposure metric and the population of interest was "all ages" (Burnett, 1999). This is in contrast to the selected PM and O_3 studies that used a daily average exposure metric and the " >= 65 population." Rather than not evaluate SO_2 hospital admissions, we chose to include this study in the evaluation, although several characteristics of the study were different from the selected PM and O_3 studies.

Second, at the time of this analysis four well conducted, large epidemiology studies of ozone mortality were available that had been extensively reviewed. Two studies used 1-hour daily maximum O_3 concentration for the exposure metric and two studies used daily average O_3 concentrations for the exposure metric. It can be reasonably argued that the 1-hour ozone metric is a better exposure metric than the daily average; however, the Samet (1997) study, that used the daily average exposure metric, was chosen for use in this study primarily because it controlled for the greatest number of possible confounding air pollutants (NO_2, SO_2, particles, and carbon monoxide).

Third, at the time of this analysis there were several epidemiology studies on the respiratory effects of O_3. In the 1996 U.S. Environmental Protection Agency's assessment of O_3, the Thurston et al. (1992) study was selected as the most appropriate study of hospitalization. This and several other studies were considered for use in the Israel assessment. The Schwartz (1995) study was chosen for several reasons: (1) it conformed to the selection criteria more closely than several other available studies; (2) the Israeli data for hospital admissions for "all causes" was available, whereas the hospital admissions data for "asthma" was not readily available; (3) the comparison between O_3 and PM was considered particularly important; hence, the choice of the Schwartz (1995) O_3 study made several characteristics the same or similar for the ozone and PM comparison, i.e., methods, relevant subpopulation >= 65, exposure metric, and health effect (all respiratory causes).

These three examples illustrate the difficult scientific judgments that must be made in conducting a comparative risk assessment. A simple example of how study choice and other assumptions can impact risk estimates is discussed earlier in the document in the uncertainties portion of risk characterization.

In addition to the difficulties identified in the preceding paragraph, very similar well-conducted studies can report different C-R relationships. In such instances, a single, optimal study cannot always be identified and additional judgments must be made concerning the selection of a C-R relationship. In this analysis, a science-policy choice was made to use an approximation of the mean or central tendency of the most appropriate C-R relationships for the particular effect in question. In some cases, several studies were pooled and an estimate of the central tendency was developed.[6] In other cases, the C-R relationship estimate was taken from one study

[5] Subpopulation is defined here as a portion of the population that has different characteristics than the general population. One such characteristic can be a different sensitivity to air pollution. Subpopulations commonly evaluated in air pollution studies include the elderly, children, and individuals with preexisting diseases, such as asthma.

[6] Taken from USEPA, 1996b.

most closely approximating the mean of the possible C-R relationships. While we have attempted to present a suite of C-R relationships that typify the current peer-reviewed literature, it would also be reasonable to identify alternative C-R relationships that may result in somewhat different results.

Ideally, the impact of all-important scientific judgments would be evaluated in an uncertainties analysis. Uncertainties analyses help to characterize the sensitivity of the risk estimates to such judgments. A detailed, quantitative uncertainties analysis was outside the scope of this effort; however, an example of how such judgments can quantitatively impact the estimates of risks is provided (Figure 9).

Selected Studies

Tables C-3 through C-6 show the concentration response functions selected for use in this study. Each pollutant assessed in this study is listed together with its associated exposure duration, health effect, population or sub-population studied, the relative risk of exposed versus unexposed population, and the slope estimate (β) and the equation used to describe the C-R relationship.

Table C-2: Considerations Used in Selecting Health Studies

Peer reviewed	Only peer reviewed research was used. Studies that have undergone the extensive review associated with the U.S. regulatory process were preferred. In particular, EPA's "Benefits and Costs of the Clean Air Act" and the criteria pollutant staff papers were utilized to identify critical studies and C-R coefficients.
Study type	Studies reporting effects in humans were used exclusively. Only studies containing C-R coefficients were used in the quantitative evaluation of risks. Studies that facilitated comparisons across pollutants, and studies consistent and coherent with the larger body of literature, were preferred.
Study period	Studies examining a relatively longer period of time, and therefore having more data, are preferred because they have greater statistical power to detect effects.
Study date	More recent studies are generally preferred because of recent improvements in design and techniques. However, studies that became available after this analysis was begun in late 1999 were not included.
Study population	Studies examining a relatively large sample are preferred, although this does not exclude the possibility of studying populations that are potentially more sensitive to pollutants, e.g., asthmatics, children, or elderly.
Study location	North American studies were used exclusively in selecting the C-R coefficients due to the extensive data development, review, and documentation associated with the U.S. regulatory process. Studies conducted in Israel were also reviewed (see Appendix E: Summary of Air Pollution-Related Epidemiology Studies in Israel). No Israeli study reviewed contained the necessary C-R coefficients needed for quantitative assessment, but the Israeli studies were considered qualitatively.
Pollutants included	Studies that evaluate more pollutants are generally preferred to studies considering fewer pollutants. Studies that attend to potential co-linearity between pollutants are preferred.
Effects included	Only health effects were included (although air pollutants are known deleterious ecological effects). Health effects that could be compared across pollutants most easily were preferred. Not all health effects were evaluated.
Measures of PM	For studies of particulate matter, only studies measuring PM10 and/or PM2.5 were used. Other measures of particulate matter, such as total suspended particulate matter (TSP), coefficient of haze (COH), or black smoke (BS), were not used; evidence indicates that PM10 and PM2.5 are more directly correlated with adverse health effects than are these more general measures of PM.

Table C-3: Concentration-Response Functions for PM_{10}

Pollutants with Exposure Durations	Health Effects	Relevant Population or Subpopulation	Relative Risk per 50 ug/m³ (95% confidence intervals[7])	β (σ_β = standard error of β)	Comments
PM_{10} Short–term Exposures					
Daily averages	Short-Term Mortality[8]	All ages	1.04 (1.01, 1.07)	0.000784	Δ Nonaccidental daily mortality = $-[y_0 \bullet (e^{-\beta \Delta PM_{10}}-1)] \bullet pop$
	Hospital Admissions-Respiratory: all causes (ICD codes: 460-519)[9]	Ages ≥65	1.09 (1.05, 1.13)	0.001723	Δ Respiratory admissions = $-[y_0 \bullet (e^{-\beta \Delta PM_{10}}-1)] \bullet pop$
	Hospital Admission Cardiovascular: all causes (ICD codes: 390-429)[10]	Ages ≥65	1.04 (1.02, 1.05)	0.000737 (0.000170)	Δ Cardiovascular admissions = $-[y_0 \bullet (e^{-\beta \Delta PM_{10}}-1)] \bullet pop$
	Lower Respiratory Symptoms[11,12,13]	Ages 7-14	2.03 (1.36, 3.04)	0.0142	$\Delta LRS = -\left[\frac{y_0}{(1-y_0) \bullet e^{\Delta PM_{10} \beta}} + y_0\right] \bullet pop$ Where: y_0 = daily LRS incidence rate per person = 0.0015

[7] As in the original analyses, the 95 percent confidence intervals are provided from pooled studies and the standard error of beta is provided for single studies.

[8] Pooled relative risk estimate - 10 locations/functions: Schwartz et al., 1996a; Ito and Thurston, 1996; Pope et al., 1992; Schwartz, 1993a; Kinney et al., 1995; USEPA, 1996b, Table VI-2, pages VI 12-13; July 2, 1997 EPA memorandum - To The PM docket, From: Harvey Richmond, RE: Corrections to Risk Analysis Tables and Figures in the PM Staff Paper.

[9] Pooled relative risk estimate - 4 locations/functions: Schwartz, 1995; Schwartz, 1996; Schwartz et al., 1996b; USEPA, 1996b, Table VI-2, pages. VI 12-13 ; July 2, 1997 USEPA memorandum - To: The PM docket, From: Harvey Richmond, RE: Corrections to Risk Analysis Tables and Figures in the PM Staff Paper.

[10] Schwartz, 1999; USEPA, 1999, Table 18, page D76.

[11] Lower respiratory symptoms were defined as cough, chest pain, phlegm and/or wheeze.

[12] Risks to asthmatic children are almost twice as high as for healthy children.

[13] Schwartz et al., 1994; USEPA, 1996b, Table VI-2, pages VI 12-13, relative risk converted from odds ratio reported in original paper.

Table C-4: Concentration-Response Functions for $PM_{2.5}$

Pollutants with Exposure Durations	Health Effects	Relevant Population or Subpopulation	Relative Risk per 25 ug/m³ (95% confidence intervals)	β (σ_β = standard error of β)	Comments
$PM_{2.5}$ Long-term Exposures					
Annual median	Annual mortality[14]	Ages ≥30	1.17 (1.09, 1.26)	0.006408 (0.001509)	ΔNonaccidental mortality = $-[y_0 \bullet (e^{-\beta \Delta PM_{2.5}} - 1)] \bullet pop$
$PM_{2.5}$ Short-term Exposures					
Daily averages	Short -Term Mortality[15]	All ages	1.04 (1.02, 1.05)	0.001569	ΔNonaccidental daily mortality = $-[y_0 \bullet (e^{-\beta \Delta PM_{2.5}} - 1)] \bullet pop$
	Hospital admission All respiratory causes (ICD codes: 466, 480-482, 485, 490-493)[16]	All ages	1.15 (1.02, 1.28)	0.006	ΔAll respiratory admissions = $\beta \bullet \Delta PM_{2.5} \bullet pop$
	Lower Respiratory Symptoms[17]	Ages 7-14	1.44 (1.15, 1.82)	0.01823 (0.00586)	$\Delta LRS = -\left[\frac{y_o}{(1 - y_o) \bullet e^{\Delta PM_{2.5}\beta} + y_o} - y_o\right] \bullet pop$ Where: y_0 = daily LRS incidence rate per person = 0.0015

[14] Pope et al., 1995, 51 locations; USEPA, 1996b, Table VI-2, pages VI 12-13.

[15] Pooled relative risk estimate - 6 locations: Schwartz et al.1996a; USEPA, 1996b, Table VI-2, pages VI 12-13.

[16] Thurston et al., 1994; USEPA, 1996b, Table VI-2, pages VI 12-13.

[17] Schwartz et al., 1994; USEPA, 1996b, Table VI-2, pages VI 12-13 ; relative risk converted from odds ratio reported in original paper, USEPA, 1999, Table 18, page 77.

Table C-5: Concentration-Response Functions for Ozone

Pollutants with Exposure Durations	Health Effects	Relevant Population or Subpopulation	Relative Risk (RR)	β (σ_β = standard error of β)	Comments
Ozone					
Daily averages	Daily mortality[18]	All ages	1.02/20 ppb	0.000936 (0.000312)	ΔDaily mortality = $-[y_0 \bullet (e^{-\beta\Delta O_3} - 1)] \bullet$ pop
	Hospital Admissions- All respiratory causes (ICD codes: 460-519)[19]	Ages ≥65	1.07/10 ppb	0.00715 (0.00257)	ΔAll respiratory admissions = $-[y_0 \bullet (e^{-\beta\Delta O_3} - 1)] \bullet$ pop
Daily 8-hour maximum	Impaired Lung function with symptoms[20 20a 21]	All ages[22]	1.09/60 ppb	4.9 (see next column)	Rc(C) = (a + bC) • 100% R(C) = % population response rate as a function of C, the [O_3] in ppm. For 8 hr exposures and 15% decrement in $FEV_{1:}$ a = - 0.2087; b = 4.9 Rc(C) = 0% if (a = bC) is < 0; Rc(C) = 100% if (a+bC) is >100% Threshold ~ 0.043 ppm, hence, below this R(C) = 0.

[20a] The functions from the Argonne National Lboratory report are based on exposure-response function and not ambient concentration response. In addition, the risk assessment, as conducted here, calculates the incidence assuming that a person exposed is engaged in moderate exertion. As a consequence, the actual risk of impaired lung function is likely be lower.

[18] Samet et al., 1997; USEPA, 1999, Table 17, page 65.

[19] Schwartz, 1995 (Tacoma); USEPA, 1999, Table 17, page 67.

[20] The measure used was a 15 percent decrease in forced expiratory volume, which is generally considered to be adverse. Ozone-induced symptoms include cough, throat irritation, chest pain on deep inspirations, nausea, and shortness of breath (USEPA, 1996a).

[21] Folinsbee et al., 1988; Horstman et al., 1990; McDonnell et al., 1991; Argonne National Laboratory, 1996, Figure B.28, page 113.

[22] Original study ages 18-30 (with moderate exertion), but the C-R function can be applied to all ages. Ozone responses decrease as a function of age; hence risks will be somewhat overestimated for age greater than 30 and underestimated for ages less then 18 (USEPA, 1996a).

Table C-6: Concentration-Response Functions for Sulfur Dioxide and Nitrogen Dioxide

Pollutants with Exposure Durations	Health Effects	Relevant Population or Subpopulation	Relative Risk (RR)	β (σ_β = standard error of β)	Comments
Sulfur Dioxide					
Daily 1 hour max[23]	Hospital admissions - All respiratory (ICD codes: 493, 464, 466,480-487, 490-492, 494, 496)[24]	All ages	1.02/4.0 ppb	0.00446 (0.00293)	ΔRespiratory admissions = $-[y_0 \bullet (e^{-\beta \Delta SO_2} - 1)] \bullet pop$
5 min peak/hr estimated using 1 hr x 2.5[25]	Increased asthma symptoms[26]	All ages; Exercising asthmatics	1.2/0.6-1.0 ppm	0.00589 (0.00247)	$\Delta Symptoms = \left[\frac{1}{1 + e^{-\sigma - (SO2 \bullet \beta) - 1}}\right] \bullet pop$ σ = constant = −5.65 β = 0.00589 SO_2 = peak 5 minute conc. = Hourly SO_2 ppb • 2.5 pop = 5.61% • 1.7% (5.61% of general population are asthmatics • 1.7% of asthmatics that exercise
Nitrogen Dioxide					
Daily averages	Hospital admissions Respiratory infections (ICD codes: 464, 466,480-487, 494[27]	All ages	1.04/25.0 ppb	0.00172 (0.000521)	Δ Resp. infections admissions = $-[y_0 \bullet (e^{-\beta \Delta NO_2} - 1)] \bullet pop$ y_0 = daily hospital admission rate for respiratory infections per person = $1.56 \bullet 10^{-5}$

[23] Until recently, it has been generally thought that the effect of ambient SO_2 exposures was limited to increased asthma symptoms in asthmatics. However, a few recent, well-conducted studies suggest that health impacts of SO_2 may be broader than previously thought and include increases in respiratory disorders other than asthma.

[24] Burnett, 1997; USEPA, 1999, Table 19, page D81.

[25] The exposure assessment for SO_2 very short-term exposures (e.g., 5 minutes) is complex. The "at-risk" population is exercising asthmatics. The risk assessment, as conducted here, calculates the incidence assuming that a person with asthma is engaged in moderate to heavy physical activity at the time and place that a "peak" exposure occurs. In other words, the assessment more reflects the occurrence of a concentration that could be expected to cause an effect independent of the likelihood that a susceptible person would be present when the peak exposure occurred. More precise estimates of risk would require additional information on time-activity and exposure patterns. Consequently, these SO_2 risks will likely be overestimates and are more uncertain than are the assessments for the other pollutants and exposure durations. In addition, if 5-minute peak exposures exceed 3.0 ppm, the entire population is at risk of experiencing bronchoconstriction. Review of the Israeli monitoring data suggested that such concentrations are unlikely over a wide geographic area.

[26] Linn et al.,1987, 1988, 1990; Roger et al., 1985; USEPA, 1999, Table 19, page D81.

[27] Burnett, 1999; USEPA, 1999, Table 16, page D62.

Israeli Demographic Data

In addition to the information on the relationship between pollutant concentrations and health effects, certain demographic data are needed to estimate risks. Specifically these data describe(1) the total population and selected subpopulationsizes by age in Greater Tel Aviv and Ashdod and (2)rates of death and disease in these populations(termed here "baseline health"). The population data are used as a surrogate for the number of people exposed. The baseline health data estimate death and illness from all causes except accidents,murder, and suicide. Risks attributed to air pollution are calculated as a percentage of the baseline health data. Data for the study areas were obtained from two sources: Israel Central Bureau of Statistics andthe Israel Ministry of Health.

Population Data

Population and subpopulation data were obtained from the Israel Central Bureau of Statistics for towns and villages within the study area by age groups (all ages, 0 - 1, ≥30, and ≥65 years of age) for the years 1995 to 1999. The portion of the Israeli population that is likely to be asthmatic was estimated based on data from the United States, where 5.6 percent of the population is asthmatic. According to the Asthma Association of Israel, about 5 to 8 percent of the population are asthmatics.[28] An example of these data is shown in Table C-7. The population was characterized partially by age and disease state to match the subpopulations evaluated in the C-R studies.

[28] See: The Lungs Clinic, Rabin Medical Center (In Hebrew), URL: http://www.lung-rmc.co.il.

Table C-7: Population Data for Greater Tel Aviv and Ashdod (1997)*

Population	Greater Tel Aviv	Greater Ashdod
Total	959,543	181,328
Ages ≥65	149,393	18,648
Ages ≥30	514,418	87,523
Children, ages 7-14	142,012	26,836
Asthmatics	53,830	10,172

* Mortality data was provided by the Israel Central Bureau of Statistics. Morbidity data was provided by the Israel Ministry of Health.

Baseline Health Data

Mortality data were available from the Israel Central Bureau of Statistics for the years 1995 to1999. Mortality rates (incidence per 100,000 people) were available for 1995 to 1997. Rates were calculated based on mortality from disease (excluding suicide, murder, and accidents). For 1998 to 1999, only total mortality figures were available (including suicide, murder, and accidents). Mortality rates for these later years were adjusted using the average ratio between mortality from disease and total mortality for the years 1995 to 1997. The average was then applied to estimate mortality rates for the years 1998 to 1999. An example of these data is shown in Table C-8.

Table C-8: Baseline Health Effects (incidence/100,000 population/year)*

Health Effects	Greater Tel Aviv	Greater Ashdod
Mortality		
A. All ages	860	569
B. ≥30 years	1577	1138
Morbidity		
A. Hospitalization		
Respiratory - all causes (≥65 yrs) ICD Codes: 460-519	9264	8290
Respiratory - all causes (all ages) ICD Codes: 460-519	2512	1877
Cardiovascular - selected causes (≥65 yrs) ICD Codes: 410, 411-414, 427, 428	31,783	23,059
B. Respiratory Symptoms		
Respiratory symptoms (daily incidence in children (ages 7-14)**	0.15%	0.15%
Asthmatics	5.6%	5.6%

* Mortality data were provided by the Israel Central Bureau of Statistics. Morbidity data were provided by the Israel Ministry of Health.

**Lower respiratory symptoms: cough, chest pain, phlegm, wheeze.

The Israel Ministry of Health provided hospital admissions data for the years 1995 to 1997, grouped by the International Classification of Disease (ICD) codes (9th revision) for age and year. Diseases include: acute myocardial infarction; other ischemic heart disease; cardiac dysrhythmias and heart failure; respiratory diseases, including bronchitis, emphysema, asthma, chronic airway obstruction; pneumonia; and influenza. City-specific disease rates were used. These rates are believed to underestimate actual admissions, potentially by as much as 20 percent.[29]

The baseline incidences of the less severe health effects (asthma and respiratory symptoms) were estimated based on U.S. data. Subsequently, data from the Israel Israel Ministry of Health confirmed that asthma rates in the United Statesand Israel were similar. The similarity of respiratory symptoms incidence between U.S. and Israel in unknown.

Uncertainties

The health information presented here is largely consistent and coherent. In other words, the majority of studies are in agreement in terms ofwhat health effects are associated with specific pollutants and the approximate magnitude of the responses (consistent). In addition, there is alogical link between the various health outcomes and what is known about the underlying biologic mechanism (coherent). These two attributes greatly increase the level of confidence in the data.

Despite the consistency and coherence in the data with respect to health effects, significant uncertainties exist. These uncertainties can impact the risk estimates and interpretation of the results. The notable uncertainties are shown in Table 7: Uncertainties in Risk Assessment, which appears on page 18. Instances in which these areas of uncertainty may result in over- or under-estimations of risk are noted.

In addition to the uncertainties presented in Table 7 (page 18), important conceptual issues should benoted relative to the risk estimates. The issues tonote here are: results of increasing exposures,multiple effects, and thresholds vs. no-thresholds.

- The results of increasing exposures to most pollutants are: (1) the percent of the population affected increases (incidence), (2) the severity of the effect increases, and (3) other effects begin to occur. It is difficult to evaluate simultaneously these multiple changes as a function of concentration. Consequently, it is common to look at the incidence of a defined health effect as a function of concentration while holding the other variables constant. This is the approach taken in this report. It is important to remember, however, that as the incidence increases, it is likely that both the severity of the effect and the types of other health effects are increasing simultaneously.

- Evaluation of pollutant-related public health impacts is complicated by the fact that most pollutants can produce multiple effects in the same population. The pollutants cause both similar or dissimilar effects. This makes attribution of effects to a specific pollutant more difficult when multiple pollutants are present.

- There is controversy about the degree of risk posed by very low exposures. Historically, there has been a commonly held belief that below a certain concentration no health effect occurs. The concentration below which no effects occur is called a "threshold for effect." More recently it has been suggested that thresholds do not exist or cannot be identified in large, diverse populations. (Apparent observed thresholds can be artifacts of small sample size or crude measures of toxicity.)

In most recent epidemiological studies, no thresholds have been observed. In other words, no pollutant concentrations were studied that were not associated with health effects. The existence or absence of a threshold is important to the risk assessment because the large majority of people who are exposed to the lowest concentrations can contribute significantly to the total estimate of risk; i.e., if low-exposure individuals are below some unidentified threshold, and if a threshold is not included in the calculation, the risk will be overestimated. Alternatively, if a threshold is assumed but does not exist, the risk will be underestimated. The resolution of this controversy will not occur in the near future. For this assessment, if an apparent threshold was observed in the original study from which the C-R coefficient was derived, a threshold was assumed in the risk calculations. If no threshold was observed in the original study, no threshold was assumed in the risk calculation.

[29] Personal communication from the Israel Israel Ministry of Health.

Summary

This section has identified and discussed the key components of the health assessment:

- Health effects commonly associated with PM, O_3, SO_2, and NO_2 and a subset of health effects evaluated in this study.
- Key scientific studies quantitatively linking human health effects and air pollutant concentrations used in risk calculating.
- Demographic data used as inputs to therisk calculations included both population data and baseline health data for mortality and morbidity.
- Uncertainties.

These data, when integrated with the exposure assessment, yield estimates of air pollution public health risks and a qualitative understanding of the associated uncertainties.

APPENDIX D:

RESULTS

Contents

Tables D1, D2, and D3 summarize estimated cases of mortality (death) and morbidity (illness) attributed to four air pollutants in the Greater Tel Aviv and Greater Ashdod study areas. Each table presents the estimated mean number of cases (in **bold**), followed by the percent of total cases of the health endpoint in parentheses estimated due to that pollutant. The 95 percent confidence interval for the risk estimate is provided in the parenthesis underneath. Two different measures of particulate matter (PM), PM_{10} and $PM_{2.5}$, were also evaluated. Tables are organized by health effects. For each pollutant and specific health effect, the number of cases (incidence), the percent of baseline incidence that these cases represent, and the 95 percent confidence intervals are given.[1] Unless otherwise noted, presented risks are to the total population. The narrative in the following sections refers to the average values of the years evaluated. Risk estimates are rounded to the nearest 10.

The risks discussed below were calculated for total pollution (overall risks) (Tables D1-A, D2-A, and D3-A) and anthropogenic pollution (Tables D1-B, D2-B, and D3-B). As noted in Appendix B: Exposure, background concentration, or the concentration attributable to natural sources, was defined as the lowest monitored concentration. By subtracting background concentrations from the total monitored concentrations, man-made pollution levels were estimated.

[1] Disease has many causes. The baseline incidence is the number of cases of specific health effects from all causes. The pollution-attributed cases can be calculated as a percentage of the cases resulting from all causes. Because this calculation is expressed as a percentage of the population, it is not affected by changes in the population. Consequently, it may be used to compare risks across different years and cities with different population sizes. It is, therefore, the measure of risk preferred by many people.

RISK ESTIMATES FOR MORTALITY[2]
(Tables D1-A and D1-B)

Overall Risk from Natural and Anthropogenic Pollution (Table D1-A)

There are an estimated 1,100 and 150 cases/year of mortality (ages 30 and up) in Greater Tel Aviv and Greater Ashdod, respectively, from long-term exposures to $PM_{2.5}$. The estimated number of cases remained relatively stable during the period 1995 to 1999 for Greater Tel Aviv and the period 1998 to 1999 for Greater Ashdod. These estimated rates account for approximately 14 percent of yearly baseline mortality due to disease, ages 30 and up, in Greater Tel Aviv and Greater Ashdod (Table D1-A). PM data were not available for Ashdod in 1995 to 1997.

Annual mortality attributable to short-term exposure to $PM_{2.5}$ and PM_{10} is estimated to be 350 cases/year in Greater Tel Aviv for the years 1995 to 1999 and 50 cases/year in Greater Ashdod area for the years 1998 to 1999. This is about 4 percent of the mortality from disease in both regions. Risk estimates for $PM_{2.5}$ and PM_{10} should be viewed as alternative estimates of the total health impacts of particles on health and should not be summed.[3]

Exposure to natural and man-made ozone (O_3) is estimated to be 140 mortality cases/year in Greater Tel Aviv, 1995 to 1997. This is approximately 2 percent of baseline mortality from disease. In Greater Ashdod, pollutant-related mortality is estimated to be 20 cases/year (1995 to 1997) or 2 percent of mortality from disease.

Anthropogenic Risk (Table D1-B)

Mortality due to long-term exposure to $PM_{2.5}$ from anthropogenic sources is estimated to be 620 cases/year in Greater Tel Aviv and 90 cases/year in Greater Ashdod (1995 to 1999). This is approximately 8 percent of mortality in both regions.

In Greater Tel Aviv an estimated 180 cases/year (2 percent) in 1995 to 210-230 deaths/year (3 percent) from 1996 to 1999 are due to short term exposure to anthropogenic concentrations of PM_{10} and $PM_{2.5.}$ In Greater Ashdod, there are an estimated 30 cases/year, representing 3 percent of mortality from disease between 1998 and 1999.

Short-term exposure to man-made O_3 is estimated to be 1 percent of mortality in Greater Tel Aviv and Greater Ashdod. In Greater Tel Aviv, this was an estimated 70 cases in 1995 and 1996 and 90 cases in 1997. In Greater Ashdod, there were an estimated 10 cases in 1995 and 1996 and 15 cases in 1997.

RISK ESTIMATES FOR HOSPITAL ADMISSIONS
(Tables D2-A and D2-B)

Overall Risk from Natural and Anthropogenic Pollution (Table D2-A)

Hospitalization of people 65 and older from all respiratory causes attributable to natural and anthropogenic PM_{10} is estimated to be 1,040 cases in Greater Tel Aviv in 1995 (8 percent of total hospitalization due to respiratory causes), increasing to an estimated 1,240 (9 percent) in 1997. In Greater Tel Aviv, natural and anthropogenic $PM_{2.5}$ is estimated to have caused approximately 500 cases of hospitalization (all ages) in 1995, increasing to an estimated 590 in 1999. This represents 3 to 4 percent of hospitalizations due to respiratory causes.[4]

PM_{10} is also associated with increased hospital admissions for cardiovascular causes. Hospitalization for people 65 and older due to selected cardiovascular causes (ICD Codes 410, 411-414, 427) is estimated to have increased from 1,670 in 1995 to 1,890 in 1997 in Greater Tel Aviv. This represents approximately 4 percent of yearly hospitalization of the elderly due to cardiovascular causes.

Hospitalization of all ages from all causes of respiratory illness attributable to exposure to combined natural and anthropogenic sources of O_3 is estimated to be 1,510 cases (11 percent of total

[2] Mortality is based on deaths from disease, excluding accidents, murders, and suicides.

[3] In addition, long- and short-term PM exposure analyses are considered to be different analyses of the same pollutant-related effect and should not be added together. To do so will double-count attributable deaths. Studies of long-term exposures are believed more fully to capture pollutant-related deaths relative to studies of short-term pollutant exposures. Studies of long-term exposures are, however, more difficult; consequently fewer such studies are available. For this reason, most of the studies included in this report rely on short-term exposure characterizations. The evaluation of PM short-term exposures is included to allow comparison with these other studies.

[4] Morbidity data for 1998 and 1999 were not available; estimated percentages were assumed to be similar to previous years (3 percent).

hospitalization due to respiratory causes), increasing to an estimated 1,830 in 1997 (13 percent) in Greater Tel Aviv. In Greater Ashdod, hospitalization attributable to O_3 is estimated to have increased from 150 cases (12 percent) in 1995 to an estimated 260 cases (13 percent) in 1997.

Exposure to sulfur dioxide (SO_2) is estimated to contribute an average of 1,100 hospitalizations year (respiratory-all causes, all ages) in Greater Tel Aviv from 1995 to 1997. This represents about 7 percent of all hospitalizations in Greater Tel Aviv due to respiratory causes. In Greater Ashdod, the number of cases increased from an estimated average of 75 in 1995 to 1996 to 250 in 1997, an increase from 4 to 7 percent of hospitalizations of all ages due to all respiratory causes. It should be noted that factors such as unusual meteorology can cause idiosyncratic changes in monitored ambient concentrations and consequent risk estimates. SO_2 levels in Greater Ashdod should be watched closely to determine if the data represent a long-term increase in ambient concentrations and risks. From 1995 to 1997, there was actually an improvement in the sulfur content in the fuels used by the Eshkol power plant and the refineries. An increasing number of relatively small facilities using higher sulfur content fuels might explain the increase in SO_2 concentrations and the increase in SO_2-attributed hospitalizations.

Nitrogen dioxide (NO_2) is estimated to cause 1,100 cases (6 percent) of hospitalizations (all ages) from respiratory infections during 1995-1996, and an estimated 750 cases (4 percent) in 1997 in Greater Tel Aviv. In Greater Ashdod, estimated cases for the same health effect range from 30 to 90 between 1995 and 1997, remaining at around 2 to 3 percent of hospitalizations (all ages) for respiratory infections.

Anthropogenic Risk (Table D2-B)

PM_{10} hospitalization risk estimates (respiratory - all causes; ages $\geq$65) increased from 600 cases (5 percent) in 1995 to 800 cases (6 percent) in 1997 in Greater Tel Aviv. Hospitalizations attributable to $PM_{2.5}$ (respiratory - all causes; all ages) averaged an estimated 380 cases/year in 1996 to 1999. This represents about 2 percent of all hospitalizations. In Greater Ashdod, there are an estimated 80 hospitalizations (respiratory-all causes; all ages) from $PM_{2.5}$ for 1998 and 1999. In Greater Tel Aviv, hospitalization due to cardiovascular causes (ages $\geq$65) from PM_{10}, increased from an estimated 970 cases (2 percent) in 1995 to an estimated 1220 cases (3 percent) in 1997.

In Greater Tel Aviv, O_3 hospitalization risk estimates (all respiratory causes, ages $\geq$65) increased from 830 in 1995-1996 to 1,120 cases in 1997. This represents a 2 to 3 percent increase. In Greater Ashdod, hospitalization risks from O_3 increased from an estimated 80 cases (7 percent) in 1995 to an estimated 180 cases (12 percent) in 1997.

In Greater Tel Aviv, an estimated 1,000 cases/year (6 percent) of hospitalizations (respiratory - all causes; all ages) were due to SO_2 in the years 1995 to 1997. In Greater Ashdod, hospitalizations due to SO_2 (respiratory-all causes, all ages) increased from an estimated 60 cases (3 percent) in 1995 to an estimated 220 (6 percent) in 1997.[5]

An average estimated 960 cases of hospitalization were due to respiratory infection from NO_2 in Greater Tel Aviv in 1995-1996 (6 percent), decreasing to an estimated 600 cases (4 percent) in 1997. In Greater Ashdod, NO_2 attributed hospitalizations (respiratory infections; all ages) are estimated to be 10 (1 percent) in 1995, increasing to an estimated 60 (2 percent) in 1997.

RISK ESTIMATES FOR LESS SEVERE HEALTH EFFECTS (Tables D3-A and D3-B)

Overall Risk from Natural and Anthropogenic Pollution (Table D3-A)

In Greater Tel Aviv, in 1995, there were an estimated 36,000 cases of respiratory symptoms[6] in children (ages 7-14) from PM_{10}, increasing to almost 40,000 cases in 1999. This represents an estimated increase from 25 to 28 percent of doctor-diagnosed cases of lower respiratory symptoms in children ages 7-14. Cases of the same health effect and age group from PM_{25} is estimated to have increased from approximately 26,000 (18 percent) in 1995 to an

[5] As was noted earlier, substantial evidence implicates PM and O_3 exposures as causes of increased hospital admissions. A few well-conducted studies have also implicated SO_2 and NO_2 exposures in increased hospital admissions, but the weight of evidence is not substantial at this time. Hospital admissions for NO_2 were restricted to respiratory infections (versus all causes), because the evidence for other causes is inadequate. For comparative purposes, estimated hospital admission risks for all four pollutants are reported here.

[6] Lower respiratory symptoms were defined as cough, chest pain, phlegm and/or wheeze.

estimated 29,000 cases (21 percent) in 1999. In Greater Ashdod, the number of respiratory symptoms in the same age group from PM_{10} is estimated at approximately 8,000 cases in 1998 and 1999, representing about 28 to 29 percent of the cases in this age group. Cases for same age group and health effect from $PM_{2.5}$ in 1998 and 1999 are estimated at 6,000 cases in Greater Ashdod, which represents 20 to 21 percent of all cases in children ages 7-14.

The risk estimates for impaired lung function with symptoms from O_3 are presented without adjusting for personal exposure. Indoor concentrations of O_3 are typically much lower than outdoor levels because O_3 is a highly reactive gas. According to Ozkaynak (1999) and Lee et al. (2002), typical indoor O_3 levels in U.S. homes range around 10 percent to 30 percent of outdoor concentrations, depending on presence of air conditioners and other indoor source or ventilation characteristics. On the other hand, indoor/outdoor O_3 ratios in residences with open windows could be much greater, or around 0.7 (Lee et al., 1997).

Unfortunately, in the absence of Israel-specific data on O_3 penetration factors into local homes (which are expected to be quite different than in the United States) and time spent outdoors by different individuals, it is impossible to develop at this time a reliable time-weighted personal exposure factor, based on ambient ozone measurements alone, that can be used to adjust impaired lung function risk estimates presented here using the ambient monitoring data collected in Israel. Moreover, the available C-R relationship for O_3 and impaired lung functions with symptoms suggests a threshold below which the C-R relationship is zero, which further complicates this analysis. Thus, the calculations shown in this report, without employing a personal exposure adjustment factor, should be considered upper-bound risk estimates for the projected lung function impairments in Israel. Impaired lung function with symptoms (all ages) due to O_3 exposure in Greater Ashdod is estimated at less than approximately 700 cases in 1995, increasing to less than an estimated 5,000 cases in 1996 and 1997. This increase might be explained by two related factors. First, the C-R relationship for impaired lung functions for all ages due to O_3 has a threshold concentration of 0.043 ppm. A larger number of daily 8-hour maximums exceeding this C-R threshold were recorded in 1996 and 1997 than in 1995. Second, the annual average concentration of O_3 increased in Ashdod from 1995 through 1997. Eight-hour exposure data needed for this assessment were not available for Greater Tel Aviv.

Asthmatic responses in exercising asthmatics (all ages) in Greater Tel Aviv and Greater Ashdod from SO_2 is estimated at 1 percent or less for 1995 to 1997. As was noted earlier, the very short-term nature of an exposure of interest is very uncertain; hence, the risk estimate represents a likely maximum potential for exposures of concern.

Anthropogenic Risk (Table D3-B)

Anthropogenic PM_{10} caused an estimated 22,000 (16 percent) cases of respiratory symptoms in children ages 7-14 in 1995-1996, increasing to an estimated 27,000 (19 percent) in 1997 in Greater Tel Aviv. $PM_{2.5}$ caused an estimated 16,000 cases in 1995 to 1996, increasing to about 20,000 in 1999. This represents an increase from 11 to 14 percent in this age group. In Greater Ashdod, for the same health effect, there were an estimated 5,700 cases in 1998 and 1999 from anthropogenic PM_{10} exposure; about 20 percent of all cases in children age 7-14. $PM_{2.5}$ that originated from man-made sources in Greater Ashdod caused an estimated 5,000 cases (17 percent) in 1998 and an estimated 4,000 cases (13 percent) in 1999.

O_3 caused fewer than an estimated 20 cases of impaired lung function with symptoms in Greater Ashdod in 1995, fewer than approximately 800 cases in 1996, and fewer than an estimated 1500 cases in 1997. For Greater Tel Aviv, data for 8-hour averages of O_3 were not available. As noted earlier, these calculations, without employing a personal exposure adjustment factor, should be considered upper-bound risk estimates for the projected lung function impairments from anthropogenic O_3 in Israel.

Increased asthmatic response in exercising asthmatics (all ages) in Greater Tel Aviv and Greater Ashdod from SO_2 is estimated at 1 percent or less.[7]

Summary

Figure D1 illustrates the data presented in Table D2-B. The data in this figure clearly show several underlying patterns of exposure and risk. Upward trends are clear for O_3 in both cities. This warrants particular attention. The increase in Greater Ashdod's SO_2 levels in 1997 should be investigated further. From the perspective of air pollution levels and percentage of baseline

[7] Exercising asthmatics are considered the most sensitive sub-population, hence, the most at-risk. As noted earlier, the uncertainties in this very short-term exposures analysis are high; the risk estimate should be considered an upper bound on possible risks.

incidences, air pollution in Greater Ashdod appears more severe than that of Greater Tel Aviv for O_3 and PM; air pollution levels in Greater Tel Aviv for NO_2 and SO_2 appear worse than in Greater Ashdod in the years 1995-1996. SO_2 levels were similar in 1997. More people, however, are affected consistently in Greater Tel Aviv, due to its larger population. Trend analysis should continue and be updated as new data become available.

One of the most significant issues highlighted by the data presented here is the impact of natural and anthropogenic sources on public health risks. Both appear to impose significant burdens on public health.

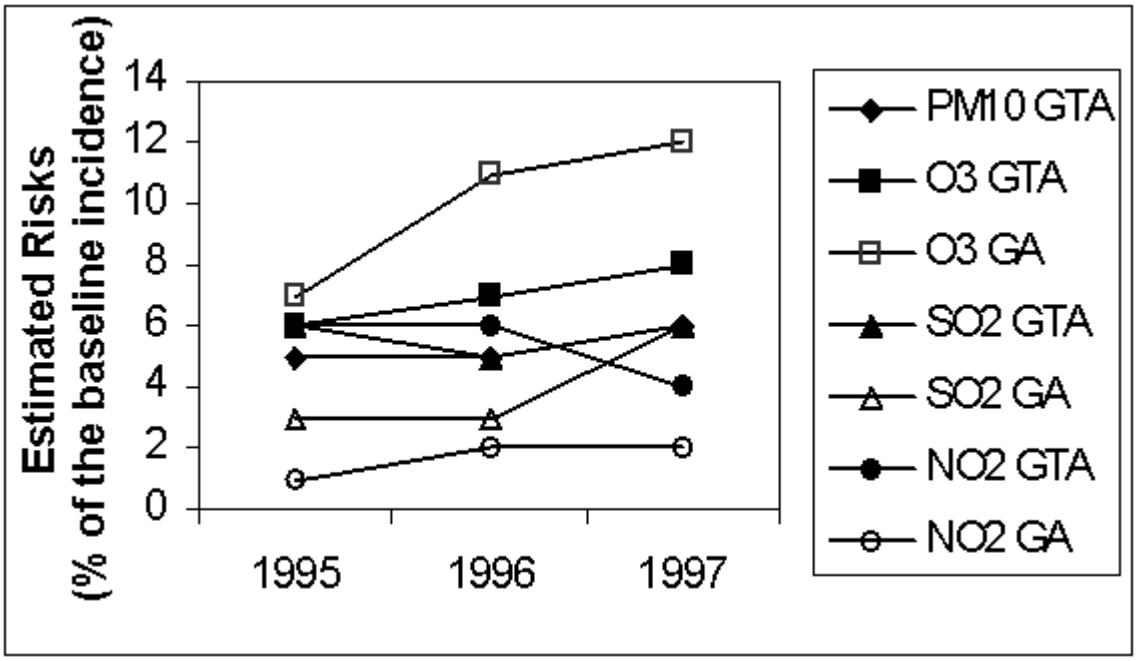

Figure D1: Comparison of Greater Tel Aviv (GTA) and Greater Ashdod (GA) Air Pollution Risks
Hospital Admissions-Respiratory Causes
Man-Made Pollution Only
1995-1997
Note: PM_{10} risk estimates are not available for Greater Ashdod.

Table D1-A: Risk Estimates for Mortality: Natural and Man-Made Pollution

(Background Concentration: 0 ug/m^3)

Pollutant	Health Effects: Mortality	Population Evaluated	Annual Incidence 1995		Annual Incidence 1996		Annual Incidence 1997		Annual Incidence 1998		Annual Incidence 1999	
			PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
Greater Tel Aviv												
Particulate Matter	Annual Exposures	≥30	**NA** (a)	**1170** (14%) (640-1660)	**NA** (a)	**920** (11%) (500-1320)	**NA** (a)	**1140** (14%) (620-1620)	**NA** (a)	**1150** (14%) (630-1630)	**NA** (a)	**1110** (13%) (600-1570)
	Short-Term Exposures	All ages	**320** (4%) (80-540)	**320** (4%) (160-400)	**340** (4%) (90-560)	**340** (4%) (180-410)	**350** (4%) (90-590)	**350** (4%) (180-430)	**390** (5%) (100-650)	**390** (5%) (200-480)	**360** (4%) (90-610)	**360** (4%) (180-440)
Ozone	Short-Term Exposures	All ages	**140** (2%) (50-230)		**140** (2%) (50-230)		**150** (2%) (50-250)		**NA** (b)		**NA** (b)	
Greater Ashdod												
Particulate Matter	Annual Exposures	≥30	**NA** (c)	**NA** (c)	**NA** (c)	**NA** (c)	**NA** (c)	**NA** (c)	**NA** (a)	**160** (14%) (90-220)	**NA** (a)	**150** (13%) (80-210)
	Short-Term Exposures	All ages	**NA** (c)	**NA** (c)	**NA** (c)	**NA** (c)	**NA** (c)	**NA** (c)	**50** (4%) (10-80)	**50** (4%) (20-60)	**50** (4%) (10-80)	**50** (4%) (30-60)
Ozone	Short-Term Exposures	All ages	**20** (2%) (5-30)		**20** (2%) (10-30)		**20** (2%) (10-40)		**NA** (c)		**NA** (c)	

(a) No suitable epidemiological study identified. (b) No Israel health data available. (c) No monitoring data available.
Estimated mean number of cases are presented in **bold**, followed by the percent of total cases of the health endpoint in parentheses.
The 95 percent confidence interval for each risk estimate is provided in the parenthesis underneath.

Table D1-B: Risk Estimates for Mortality: Man-Made Pollution

(Background Concentrations: PM_{10}= 20 ug/m^3; $PM_{2.5}$= 10 ug/m^3; O_3= 8 ppb; SO_2= 2 ppb; NO_2= 5 ppb)

Pollutant	Health Effects: Mortality	Population Evaluated	Annual Incidence 1995		Annual Incidence 1996		Annual Incidence 1997		Annual Incidence 1998		Annual Incidence 1999	
			PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
Greater Tel Aviv												
Particulate Matter	Annual Exposures	≥30	**NA** (a)	**670** (8%) (360-970)	**NA** (a)	**450** (6%) (240-650)	**NA** (a)	**680** (8%) (370-980)	**NA** (a)	**680** (8%) (370-980)	**NA** (a)	**630** (8%) (340-910)
	Short-Term Exposures	All ages	**180** (2%) (50-310)	**180** (2%) (90-230)	**210** (3%) (60-350)	**210** (3%) (110-260)	**220** (3%) (60-380)	**220** (3%) (110-280)	**260** (3%) (70-440)	**260** (3%) (140-320)	**230** (3%) (60-390)	**230** (3%) (120-290)
Ozone	Short-Term Exposures	All ages	**70** (1%) (20-120)		**70** (1%) (20-120)		**90** (1%) (30-150)		**NA**		**NA**	
Greater Ashdod												
Particulate Matter	Annual Exposures	≥30	**NA** (c)	**NA** (c)	**NA** (c)	**NA** (c)	**NA** (c)	**NA** (c)	**NA** (a)	**90** (9%) (50-130)	**NA** (a)	**90** (8%) (50-120)
	Short-Term Exposures	All ages	**NA** (c)	**NA** (c)	**NA** (c)	**NA** (c)	**NA** (c)	**NA** (c)	**30** (3%) (10-50)	**30** (3%) (20-40)	**30** (3%) (10-50)	**30** (3%) (20-40)
Ozone	Short-Term Exposures	All ages	**10** (1%) (3-10)		**10** (1%) (5-20)		**15** (1%) (5-25)		**NA** (c)		**NA** (c)	

(a) No suitable epidemiological study identified. (b) No Israel health data available. (c) No monitoring data available.
Estimated mean number of cases are presented in **bold**, followed by the percent of total cases of the health endpoint in parentheses.
The 95 percent confidence interval for each risk estimate is provided in the parenthesis underneath.

Table D2-A: Risk Estimates for Hospital Admissions: Natural and Man-Made Pollution

(Background concentration: 0 ug/m^3)

Pollutant	Health Effects	Population Evaluated	Annual Incidence 1995		Annual Incidence 1996		Annual Incidence 1997		Annual Incidence 1998		Annual Incidence 1999	
			PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
Greater Tel Aviv												
Particulate Matter	Respiratory - all causes	≥ 65 (PM_{10}) All ages ($PM_{2.5}$)	**1040** (8%) (600-1440)	**500** (3%) (70-820)	**1060** (8%) (630-1450)	**590** (4%) (80-970)	**1240** (9%) (720-1720)	**580** (3%) (80-960)	**NA** (c)	**680** (b,d) (90-1120)	**NA** (c)	**590** (b,d) (80-960)
	Cardiovascular - selected causes[i]	≥ 65	**1670** (3%) (910-2410)	**NA** (a)	**1810** (4%) (1000-2580)	**NA** (a)	**1890** (4%) (1030-2730)	**NA** (a)	**NA** (b)	**NA** (b)	**NA** (b)	**NA** (b)
Ozone	Respiratory - all causes	≥65	**1510** (11%) (450-2480)		**1490** (12%) (440-2430)		**1830** (13%) (550-2970)		**NA** (b)		**NA** (b)	
Sulfur dioxide	Respiratory - all causes	All ages	**1230** (7%) (440-1980)		**960** (6%) (340-1550)		**1150** (7%) (410-1840)		**NA** (b)		**NA** (b)	
Nitrogen dioxide	Respiratory infections[ii]	All ages	**1120** (6%) (450-1750)		**1070** (6%) (430-1680)		**760** (4%) (300-1200)		**NA** (b)		**NA** (b)	
Greater Ashdod												
Particulate Matter	Respiratory - all causes	≥ 65 (PM_{10}) All ages ($PM_{2.5}$)	**NA** (c)	**NA** (c)	**NA** (c)	**NA** (c)	**NA** (c)	**NA** (c)	**NA** (a)	**90** (b,d) (50-130)	**NA** (a)	**90** (b,d) (50-120)
	Cardiovascular - selected causes[i]	≥ 65	**NA** (c)	**NA** (c)	**NA** (c)	**NA** (c)	**NA** (c)	**NA** (c)	**NA** (a)	**120** (b,d) (10-190)	**NA** (a)	**120** (b,d) (20-210)
Ozone	Respiratory - all causes	≥65	**150** (12%) (40-250)		**230** (16%) (70-370)		**260** (17%) (80-410)		**NA** (b)		**NA** (b)	
Sulfur dioxide	Respiratory - all causes	All ages	**70** (4%) (30-120)		**80** (4%) (30-130)		**250** (7%) (90-410)		**NA** (b)		**NA** (b)	
Nitrogen dioxide	Respiratory infections[ii]	All ages	≤**30** (2%) (10-50)		≤**50** (3%) (20-80)		≤**90** (2%) (40-140)		**NA** (b)		**NA** (b)	

(a) No suitable epidemiological study identified. (b) No Israel health data available. (c) No monitoring data available.
(d) Hospital admission data not available; the C-R equation allows calculation of hospital admission based on total population.
Estimated mean number of cases are presented in bold, followed by the percent of total cases of the health endpoint in parentheses.
The 95 percent confidence interval for each risk estimate is provided in the parenthesis underneath.

[i] For both cities, this is likely an underestimate of risk. This estimate was limited to ICD Codes: 410, 411-414, 427. The original study included ICD Codes: 390-429.

[ii] For both cities, the baseline population was available only for ICD codes: 460-519 (all respiratory causes); number of respiratory infections (ICD codes: 464,466, 480-487, 494) was estimated at ~ 55 percent of total admissions based on Burnett et al., 1997, 1999. See references for complete citation.

Table D2-B: Risk Estimates for Hospital Admissions: Man-Made Pollution

(Background concentration: PM_{10}=20 ug/m^3; $PM_{2.5}$=10 ug/m^3; O_3=8 ppb; SO_2=2 ppb; NO_2=5 ppb)

Pollutant	Health Effects	Population Evaluated	Annual Incidence 1995		Annual Incidence 1996		Annual Incidence 1997		Annual Incidence 1998		Annual Incidence 1999	
			PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$	PM_{10}	$PM_{2.5}$
Greater Tel Aviv												
Particulate Matter	Respiratory - all causes	≥65 (PM_{10}) All ages ($PM_{2.5}$)	**600** (5%) (350-840)	**290** (2%) (40-470)	**660** (5%) (400-900)	**380** (2%) (50-630)	**800** (6%) (720-1110)	**370** (2%) (50-620)	**NA** (b)	**470** (b,d) (60-770)	**NA** (b)	**380** (b,d) (50-620)
	Cardiovascular - selected causes[i]	≥65	**970** (2%) (520-1400)	**NA** (a)	**1140** (2%) (640-1610)	**NA** (a)	**1220** (3%) (660-1760)	**NA** (a)	**NA** (b)	**NA** (b)	**NA** (b)	**NA** (b)
Ozone	Respiratory - all causes	≥65	**820** (6%) (240-1350)		**840** (7%) (240-1380)		**1120** (8%) (330-1850)		**NA** (b)		**NA** (b)	
Sulfur dioxide	Respiratory - all causes	All ages	**1080** (6%) (380-1740)		**820** (5%) (290-1330)		**1000** (6%) (350-1610)		**NA** (b)		**NA** (b)	
Nitrogen dioxide	Respiratory infections[ii]	All ages	**980** (6%) (390-1530)		**940** (6%) (380-1470)		**615** (4%) (250-970)		**NA** (b)		**NA** (b)	
Greater Ashdod												
Particulate Matter	Respiratory - all causes	≥65 (PM_{10}) All ages ($PM_{2.5}$)	**NA** (c)	**NA** (c)	**NA** (c)	**NA** (c)	**NA** (c)	**NA** (c)	**NA** (b)	**75** (b,d) (10-120)	**NA** (b)	**80** (b,d) (10-130)
	Cardiovascular - selected causes[i]	≥65	**NA** (c)	**NA** (c)	**NA** (c)	**NA** (c)	**NA** (c)	**NA** (c)	**NA** (b)	**NA** (b)	**NA** (b)	**NA** (b)
Ozone	Respiratory - all causes	≥65	**80** (7%) (20-140)		**160** (11%) (50-260)		**180** (12%) (50-290)		**NA** (b)		**NA** (b)	
Sulfur dioxide	Respiratory - all causes	All ages	**60** (3%) (20-90)		**70** (3%) (20-110)		**220** (6%) (80-360)		**NA** (b)		**NA** (b)	
Nitrogen dioxide	Respiratory infections[ii]	All ages	**10** (1%) (10-20)		**30** (2%) (10-50)		**60** (2%) (20-90)		**NA** (b)		**NA** (b)	

(a) No suitable epidemiological study identified. (b) No Israel health data available. (c) No monitoring data available.
(d) Hospital admission data not available; the C-R equation allows calculation of hospital admission based on total population.
Estimated mean number of cases are presented in **bold**, followed by the percent of total cases of the health endpoint in parentheses.
The 95 percent confidence interval for each risk estimate is provided in the parenthesis underneath.

[i] For both cities, this is likely an underestimate of risk. This estimate was limited to ICD Codes: 410, 411-414, 427. The original study included ICD Codes: 390-429.

[ii] For both cities, the baseline population was available only for ICD codes: 460-519 (all respiratory causes); number of respiratory infections (ICD codes: 464,466, 480-487, 494) was estimated at ~ 55 percent of total admissions based on Burnett et al., 1997, 1999. See references for complete citation.

Table D3-A: Risk Estimates for Other Health Effects: Natural and Man-Made Pollution

(Background concentration: 0 ug/m^3)

Pollutant	Health Effects	Population Evaluated	Annual Incidence 1995	Annual Incidence 1996	Annual Incidence 1997	Annual Incidence 1998	Annual Incidence 1999
Greater Tel Aviv							
Particulate Matter - PM_{10}	Respiratory Symptoms - Children	Ages 7-14	**36060** (25%) (18960-47650)	**36350** (26%) (19620-47590)	**39640** (28%) (21490-51310)	**40690** (28%) (22240-52450)	**39610** (28%) (21500-51270)
Particulate Matter - $PM_{2.5}$			**26010** (18%) (10850-37380)	**26620** (19%) (11530-37630)	**29180** (21%) (12490-41000)	**30060** (21%) (13080-42060)	**29170** (20%) (12510-40960)
Ozone	Impaired Lung Function with Symptoms	All ages	**NA** (c)	**NA** (c)	**NA** (c)	**NA** (c)	**NA** (c)
Sulfur dioxide	Asthmatic Response	All ages	(1%)	(1%)	(1%)	**NA** (b)	**NA** (b)
Greater Ashdod							
Particulate Matter - PM_{10}	Respiratory Symptoms - Children	Ages 7-14	**NA** (c)	**NA** (c)	**NA** (c)	**8150** (29%) (4400-10530)	**8310** (28%) (4520-10780)
Particulate Matter - $PM_{2.5}$			**NA** (c)	**NA** (c)	**NA** (c)	**5990** (21%) (2540-8430)	**6120** (20%) (2640-8600)
Ozone	Impaired Lung Function with Symptoms[i]	All ages	**< 710**	**< 4950**	**< 5120**	**NA** (b)	**NA** (b)
Sulfur dioxide	Asthmatic Response	All ages	(1%)	(1%)	(1%)	**NA** (b)	**NA** (b)

(a) No suitable epidemiological study identified. (b) No Israel health data available. (c) No monitoring data available. (d) For Particulate Matter PM10 and PM2.5 for Respiratory Symptoms - Children ages 7-14 the risk estimate percentage is for the total age group.
Estimated mean number of cases are presented in **bold**, followed by the percent of total cases of the health endpoint in parentheses.
The 95 percent confidence interval for each risk estimate is provided in the parenthesis underneath.

[i] The risk estimates for impaired lung function with symptoms from ozone are presented without adjusting for personal exposure. Indoor concentrations of ozone are typically much lower than outdoor levels because ozone is a highly reactive gas. According to Ozkaynak (1999) and Lee et al. (2002), typical indoor ozone levels in U.S. homes range around 10 percent to 30 percent of outdoor concentrations, depending on presence of air conditioners and other indoor source or ventilation characteristics. On the other hand, indoor/outdoor ozone ratios in residences with open windows could be much greater, or around 0.7 (Lee et al., 1997). Unfortunately, in the absence of Israel-specific data on ozone penetration factors into local homes (which are expected to be quite different than in the United States) and time spent outdoors by different individuals, it is impossible to develop at this time a reliable time-weighted personal exposure factor, based on ambient ozone measurements alone, that can be used to adjust impaired lung function risk estimates presented here using the ambient monitoring data collected in Israel. Moreover, the available C-R relationship for ozone and impaired lung functions with symptoms suggests a threshold below which the C-R relationship is zero, which further complicates this analysis. Thus, the calculations shown in this report, without employing a personal exposure adjustment factor, should be considered upper-bound risk estimates for the projected lung function impairments in Israel.

Table D3-B: Risk Estimates for Other Health Effects: Man-Made Pollution
(Background concentration: PM_{10}=20 ug/m^3; $PM_{2.5}$=10 ug/m^3; O_3=8 ppb; SO_2=2 ppb; NO_2=5 ppb)

Pollutant	Health Effects	Population Evaluated	Annual Incidence 1995	Annual Incidence 1996	Annual Incidence 1997	Annual Incidence 1998	Annual Incidence 1999
Greater Tel Aviv							
Particulate Matter - PM_{10}	Respiratory Symptoms - Children	Ages 7-14	**22440** (16%) (11290-30900)	**22750** (16%) (12010-30690)	**27140** (19%) (14140-36520)	**28620** (20%) (15010-38450)	**27150** (19%) (14160-36550)
Particulate Matter - $PM_{2.5}$			**15780** (11%) (6370-23350)	**16390** (11%) (7070-23620)	**19470** (14%) (8110-28170)	**20580** (14%) (8350-29700)	**19490** (14%) (8130-28180)
Ozone	Impaired Lung Function with Symptoms	All ages	**NA** (C)	**NA** (C)	**NA** (C)	**NA** (b)	**NA** (b)
Sulfur dioxide	Asthmatic Response	All ages	(1%)	(1%)	(1%)	**NA** (b)	**NA** (b)
Greater Ashdod							
Particulate Matter - PM_{10}	Respiratory Symptoms - Children	Ages 7-14	**NA** (C)	**NA** (C)	**NA** (C)	**5750** (20%) (2940-7720)	**5640** (19%) (2960-7610)
Particulate Matter - $PM_{2.5}$			**NA** (C)	**NA** (C)	**NA** (C)	**4800** (17%) (1670-5940)	**4060** (13%) (1710-5860)
Ozone	Impaired Lung Function with Symptoms[i]	All ages	**< 20**	**< 840**	**< 1490**	**NA** (b)	**NA** (b)
Sulfur dioxide	Asthmatic Response	All ages	(1%)	(1%)	(1%)	**NA** (b)	**NA** (b)

(a) No suitable epidemiological study identified. (b) No Israel health data available. (c) No monitoring data available. (d) For Particulate Matter PM10 and PM2.5 for Respiratory Symptoms - Children ages 7-14 the risk estimate percentage is for the total age group.
Estimated mean number of cases are presented in **bold**, followed by the percent of total cases of the health endpoint in parentheses.
The 95 percent confidence interval for each risk estimate is provided in the parenthesis underneath.

[i] The risk estimates for impaired lung function with symptoms from ozone are presented without adjusting for personal exposure. Indoor concentrations of ozone are typically much lower than outdoor levels because ozone is a highly reactive gas. According to Ozkaynak (1999) and Lee et al. (2002), typical indoor ozone levels in U.S. homes range around 10 percent to 30 percent of outdoor concentrations, depending on presence of air conditioners and other indoor source or ventilation characteristics. On the other hand, indoor/outdoor ozone ratios in residences with open windows could be much greater, or around 0.7 (Lee et al., 1997). Unfortunately, in the absence of Israel-specific data on ozone penetration factors into local homes (which are expected to be quite different than in the United States) and time spent outdoors by different individuals, it is impossible to develop at this time a reliable time-weighted personal exposure factor, based on ambient ozone measurements alone, that can be used to adjust impaired lung function risk estimates presented here using the ambient monitoring data collected in Israel. Moreover, the available C-R relationship for ozone and impaired lung functions with symptoms suggests a threshold below which the C-R relationship is zero, which further complicates this analysis. Thus, the calculations shown in this report, without employing a personal exposure adjustment factor, should be considered upper-bound risk estimates for the projected lung function impairments in Israel.

APPENDIX E:

SUMMARY OF AIR POLLUTION-RELATED EPIDEMIOLOGY IN ISRAEL[1]

Prepared by Eric Karsenty, MD, MPH

A number of studies have been conducted in Israel over the last two decades to determine the association between respiratory diseases and air pollution in urban surroundings. These studies were conducted all over the country in cities with different levels of air pollution (e.g., Tel Aviv, Haifa, Hadera, Ashdod, Ashkelon, and Yavne). Most of these studies took place in cities where a coal burning power plant was present (e.g., Tel-Aviv, Hadera, Ashdod) or with an air polluting industrial area (e.g., Ashdod, Haifa, Beit-Semesh). The studies that have been carried out in Israel can be placed into the following four categories, discussed in detail below:

- Pulmonary Function and Respiratory Symptoms
- Hospital Admission and Emergency Room Visits
- Mortality
- Cardiovascular

Pulmonary Function and Respiratory Symptoms

Ongoing research surveys have taken place in Israel since 1980 to assess the influence of the installation of coal-burning power plants on the health of the surrounding populations. These studies took place in the Hadera (Goren and Goldsmith, 1986; Goren, Hellman, and Goldsmith, 1988; Goren et al., 1991; Goren and Glaser, 1995; Goren and Hellman, 1995; Goren and Hellmann, 1997; Goren et al., 1999) and in the Ashkelon areas (Goren, Bibi, and Goldshmidt, 1991; Peled et al., 2001). Additional studies were conducted in Tel Aviv, where an old oil-burning power plant has been active for decades, and in towns with air of polluting industrial areas: Haifa (Goren et al., 1990; Epstein et al., 1991; Goren and Hellmann, 1993), Ashdod (Goren and Hellmann, 1988) and Bet-Shemesh (Goren et al., 1999).

A permit to build and operate the first 1400 megawatt coal-fired power plant in Israel was given on the condition that three monitoring systems--environmental, agricultural, and health--would be set up near the power plant. A series of cohort studies was carried out in the Hadera area, that included second-, fifth-, and eighth-grade school-children living in three communities with different expected levels of air pollution. Follow-up studies were conducted every 3 years (Goren and Goldsmith,1986; Goren, Hellman, and Goldsmith,1988; Goren et al, 1991; Goren and Glaser, 1995; Goren and Hellman, 1995; Goren and Hellmann, 1997; Goren et al., 1999). Children performed pulmonary function tests,[2] and their parents filled out American Thoracic Society-National Heart and Lung Institute health questionnaires. Statistical analysis of the data was carried out using SPSS[3] and BMDP[4] software. Differences in prevalence of reported respiratory symptoms and diseases according to area of residence were analyzed by means of the X^2 test. To evaluate the combined effect of all background variables in the pooled data set of the two areas of residence, a

[1] Although a working draft of this appendix was peer reviewed, it was subsequently entirely rewritten (and greatly improved upon) by Dr. Karsenty.

[2] Spirometry is the classic pulmonary function test, which measures the volume of air inspired or expired as a function of time. It can monitor quiet breathing and thereby measure tidal volume, and also trace deep inspirations and expirations to give information about vital capacity. Spirometry may also be used to measure forced expiration rates and volumes and to compute FEV1/FVC ratios. In a normal forced expiration curve, the volume that the subject can expire in one second (referred to as FEV1) is usually about 80 percent of the total forced vital capacity (FVC), or something like four liters out of five. Spirometry cannot, however, access information about absolute lung volumes, because it cannot measure the amount of air in the lung but only the amount entering or leaving (Interactive Respiratory Physiology, Johns Hopkins University, 1996).

[3] SPSS (Statistical Package for the Social Sciences) is a data management and analysis product. It can perform a variety of data analysis and presentation functions, including statistical analyses and graphical presentation of data. See: Nie NH, Hull, CH, Jenkins JG, Steinbrenner K, Bent DH (1975) SPSS: Statistical Package for the Social Sciences. Second edition. New York: McGraw-Hill. See also: Hull CH, Nie NH (1981) SPSS UPDATE 7-9: New Procedures and Facilities for Releases 7-9. New York: McGraw-Hill.

[4] BMDP is a set of programs for statistical analysis using a common control language for data input, transformations, and analyses. See: Dixon, WJ (ed.) (1983) BMDP Statistical Software (Vol. 1, 3rd ed.), Berkeley, CA: University of California Press.

logistical model was applied for the expected frequency of each respiratory condition. The variables used in the logistical regressions included crowding index, parents' education, and household heating. The odds ratio of suffering from a respiratory condition in the polluted area relative to the low-polluted area was calculated from the logistical regressions. It should be stressed, however, that the use of logistic models in Israel is problematic as there is a large degree of interdependence between variables such as ethnic origin, level of education, number of siblings, and socio-economic level. Therefore, the basic condition of strict independence of the variables for the model cannot generally be respected. Later studies tried to avoid this problem by using other statistical tests.

A significant ($p = 0.0024$) increase in the prevalence of asthma was observed among fifth-grade children in all three communities studied between 1980 and 1989. At the same time, a significant ($p = 0.0172$) rise in the self-reported prevalence of wheezing accompanied by breathing difficulty was observed. Pulmonary function tests of children reporting a history of asthma or of wheezing accompanied by breathing difficulty were generally lower than those of the other children, suggesting that the increase over time in the prevalence of asthma was a true increase in morbidity and not due to reporting bias. A similar trend could not be found for the prevalence of bronchitis and other respiratory conditions among the studied children. Changes in the prevalence of background variables over time could not explain the significant rise in the prevalence of asthma among the children. The increased prevalence of asthma could be observed in all the communities studied and did not seem to be connected with the operation of the power plant.

Among the cohort gathered in 1983 of second-graders living in the area expected to be most polluted, a significant increase in the reported prevalence of respiratory symptoms (cough and sputum, wheezing with and without cold, and wheezing accompanied by breathing difficulty) was evident in 1986. The prevalence of asthma among fifth-graders in this area was twice the prevalence ($p = 0.0273$) when they were second-graders. Among the children from the older cohort (fifth-graders in 1983) living in this community, a similar although milder trend could be observed, especially with regard to an increased prevalence of asthma in 1986 compared with 1983 (13.9 percent versus 8.1 percent). Annual increases in pulmonary function tests in the four groups of children (boys and girls from both cohorts) were found to be higher in the community expected to be polluted (especially in the younger cohort) compared with the two other communities. The discrepancy between the increased prevalence of respiratory symptoms and diseases and the higher annual increase in pulmonary function tests among children from what was expected to be a more polluted community may be partly attributable to differential annual increases in height and to a different distribution of background variables (such as socioeconomic status, passive smoking, heating, and respiratory diseases among parents) in the three communities.

The effects of socioeconomic background, smoking habits, and pulmonary diseases in the families on the distribution of respiratory symptoms, respiratory diseases, and pulmonary function tests of the children were also analyzed. Paradoxically, better pulmonary function tests and a lower prevalence of respiratory diseases were reported among children from crowded homes, though an excess in reported respiratory minor symptoms was noted. Mothers' smoking was found to be connected with significantly higher prevalence of bronchitis, but not with a higher prevalence of asthma, a significant lower FEV1/FVC,[5] or a trend of lower FEV1 and pulmonary function tests among their children. House heating did not affect the prevalence of respiratory symptoms and diseases of the children. A significantly higher prevalence of respiratory symptoms and diseases was found among children with a family history of pulmonary diseases, but no reduction of pulmonary function test parameters could be observed among them. The maternal contribution to respiratory symptoms and diseases of their children appeared to be more important than the paternal side. Most respiratory symptoms were more common among children with a history of pneumonia in childhood, especially those who had pneumonia recently.

Patients of eight clinics operated in the area by the General Sick Fund, served by 16 physicians, were followed up. The clinics were selected that were located as near as possible to the air pollution monitoring stations to be representative of different levels of exposure to pollution. A health recorder summarized each day's visits to each physician and tabulated the total visits for each day and the visits due to respiratory tract complaints. Multivariate stepwise regressions on total and on respiratory complaints were carried out. The independent

[5] During episodes of acute asthma, pulmonary function tests reveal an obstructive pattern. This includes a decrease in the rate of maximal expiratory air flow (a decrease in FEV1 and the FEV1/FVC ratio) due to the increased resistance, and a reduction in forced vital capacity (FVC) correlating with the level of hyperinflation of the lungs.

variables in the regressions were sulfur dioxide, meteorological parameters (such as temperature and humidity), and influenza epidemics. Only outdoor temperature was almost always significantly correlated with respiratory complaints, but less correlated with total visits among adults and children. Ambient air pollution levels did not exceed the Israeli air quality or the more stringent local air quality standards, the monthly and annual average sulfur dioxide and nitrogen oxides values were very low.

Goren and Hellman (1995) examined possible links between respiratory conditions among schoolchildren and exposure to environmental tobacco smoke and other home and community exposures. More than 8,000 second- and fifth-grade schoolchildren who lived in three towns along the Israeli coast were administered pulmonary function tests, and their parents completed standardized health questionnaires. The prevalences of the most reported respiratory symptoms were found to be higher, some of them significantly so, among children whose fathers or mothers were smokers, compared with children of non-smoking parents. These reported conditions were significantly more frequent among children growing up in medium- and highly-polluted communities than among children from low-polluted areas. House heating with kerosene or gas was seldom associated with higher prevalence of respiratory conditions among children. No consistent trend of reduced pulmonary function tests was associated with exposure to environmental tobacco smoke, with community pollution, or with house heating pollution. Exposure of schoolchildren to their parents' cigarette smoke and to community air pollution appeared to be associated with a higher prevalence of respiratory symptoms but not of impaired pulmonary functions.

A tentative effort to compare the health status of the population living in the Hadera region to the population living in the Ashdod area, a more heavily industrially polluted area, was also conducted by Goren, Hellmann, and Goldsmith (1988). The results of the cross-sectional survey of second- and fifth-grade schoolchildren living in two communities with different levels of air pollution did not enable meaningful conclusions due to the existence of a strong confounding bias. In this study, the parents of the children filled out ATS-NHLI health questionnaires.[6] The prevalence of reported respiratory symptoms and pulmonary diseases was found to be significantly higher among children growing up in the polluted community (Ashdod) than in the low-pollution area (Hadera). Relative risk values, calculated from the logistic models, were in the range of 1.47 for cough without cold to 2.66 for reported history of asthma among children from Ashdod, as compared with the Hadera children. The logistic models used in the analysis, however, included background variables that could be responsible for the observed differences.

A similar study to the one done in Hedera by Goren et al. (1991) was planned in the southern coastal area of Ashkelon. Peled et al. (2001) studied differences in lung functions of school-age children who lived near the electrical power plant in the Ashkelon. Lung-function tests were performed, and the American Thoracic Society questionnaire was administered in three study periods during the years 1990, 1994, and 1997. Measurements of the air pollutants (sulfur dioxide, nitric oxides, and ozone, but not particulate matter) were also made during the study periods. Statistical analyses included an estimation of a series of fixed-effects regression models. A total of 2,455, 1,613, and 4,346 observations were included in the analyses for study years 1990, 1994, and 1997, respectively. The authors controlled for age, sex, height, weight, parents' education and smoking status, and being born out of Israel. Substantial differences in lung function across the different communities and study periods were demonstrated in the study area; however, no significant association with air pollution was demonstrated.

The Bet Shemesh study was inconclusive. During the spring of 1995, Goren et al. (1999) studied schoolchildren age 7-13 in two nearby communities. In one community, the population was exposed to pollution from a cement factory and quarries; the population of the second community was not exposed to pollution from these sources. In the polluted area, total suspended particulate matter and levels of airborne particles less than 10 microns frequently violated the 24-hour Israeli standard of

[6] One of the most used questionnaires in asthma studies is the ATS-DLD-78, elaborated by the American Thoracic Society (ATS). It is composed of 46 questions about respiratory diseases, and has been initially released for administration in children over 13 years of age. Afterwards, ATS has adapted this questionnaires to parents and guardians of children under 13 years of age (ATS-DLD-78-C). See: Ferris BG (1978) Recomended Respiratory Disease Questionnaires for use with Adults and Children in Epidemiological Research. Am Ver Resp Dis; 188:1-79.

200 μg/m^3 and 150 μg/m^3, respectively. Lung functions of the children were measured and parents completed an American Thoracic Society-National Heart and Lung Institute health questionnaire, which included information about respiratory symptoms and diseases of the children and information about background variables. Minor respiratory symptoms (cough without cold, sputum without cold, and cough accompanied by sputum) appeared to be more prevalent among the 638 children who were growing up in the community that bordered the industrial zone, compared with 338 children from the unexposed community. Diagnosis of asthma by a family practitioner was also greater, but the difference was not statistically significant. No consistent trend of reduced pulmonary function tests was observed among these children, though the average peak expiratory flow was significantly lower than among the control group.

Epstein et al. (1991) studied the effects of exposure to SO_2 levels in a representative sample of over 3,500 households in the greater Haifa area. The sample was drawn from 16 high- and low-pollution neighborhoods that also represented different socioeconomic strata. A very significant relationship was found between levels of pollution in residential and work areas and self-reported respiratory symptoms and diseases. This finding was strongly supported by a statistically significant relationship between actual measured levels of SO_2 in the 2 weeks prior to the interview, the use of medical services by both adults and children, and by the number of days activity was restricted during that period.

Between 1984 and 1989, Goren et al. (1990, 1993) conducted a cohort study in the Haifa Bay area. While the first phase of the study was inconclusive, the second phase was hampered by a reporting bias. During spring 1984, 2,334 second- and 2,000 fifth-grade schoolchildren living in three areas in the Haifa Bay region were studied. The parents of these children filled out American Thoracic Society and the National Heart and Lung Institute health questionnaires, and the children performed pulmonary function tests. A trend of higher prevalence of most reported respiratory symptoms was found for schoolchildren growing up in the medium- and high-polluted areas compared with the low-pollution area. Analyses using logistic models found a trend in increased reporting of minor respiratory symptoms (relative risks ranging from 1.38 for sputum with cold to 1.81 for sputum without cold, among children in the polluted area). No consistent reduction in pulmonary lung functions was observed.

During spring 1989, seventh-graders (second-graders from the 1984 cohort) were reexamined and a new cohort of fifth-grade children was studied using the same techniques as in 1984. A very significant rise in the prevalence of most reported respiratory symptoms and diseases was observed among both fifth- and seventh-grade schoolchildren in 1989 compared to 1984, especially in the low- and medium-polluted areas, and less in the high-polluted area. Changes over time in the pulmonary lung functions of the older cohort were similar in the three areas. Results of the pulmonary lung functions of the fifth-graders from 1989 were similar to the ones found in 1984 among the first cohort fifth-graders. A closer examination of the logistic models used for the analysis show that the most significant factor in 1989 was the subjective attitude of their parents towards the deleterious effects of air pollution on their children's health, and the subjective estimate of their children's exposure to pollution rather than measured exposure. This strong reporting bias appeared to be the result of a huge campaign carried out during the survey against the main polluters in the Haifa Bay area.

Colin, Said, and Winter (1985) studied the prevalence of exercise-induced asthma in 313 Haifa schoolchildren: 160 living in an unpolluted urban area close to the sea and 153 living in a rural kibbutz (communal agricultural settlement) in the proximity of polluting industries. The overall prevalence of exercise-induced asthma was 8.6 percent; it was higher in the kibbutz group, but the difference was not significant. The prevalence tended to decrease with increasing age and did not change with sex.

Hospital Admission and Emergency Rooms Visits

Various studies have been conducted in Israel to examine the correlation between air pollution, weather conditions, airborne allergens, and the incidence of emergency room visits due to respiratory complaints (asthma, COPD, chronic bronchitis, etc.) in Tel-Aviv, Petah-Tiqva (Garty et al., 1998), Beer-Sheva (Gross et al., 1984; Porath et al., 1995) and Haifa (Cohen et al., in publication) areas.

Garti et al. (1998) examined the correlations between air pollutants, weather conditions, airborne allergens, and the incidence of emergency room visits of children with acute asthma attacks. The 1-year prospective study took place in the Schneider Pediatric Medical Center in Petach Tiqva, which serves the central area of Israel. Data on daily concentration of air pollutants,

weather conditions, and selected airborne allergens were collected and compared with the number of emergency room visits of asthmatic children. A study group of 1,076 asthmatic children (aged 1 to 18 years) was gathered between 1 January and 31 December 1993.

Correlations between fluctuations in emergency room visits of asthmatic children and various environmental parameters were more relevant for weekly than for daily values. Emergency room visits correlated positively with concentrations of NO_x, SO_2, and with high barometric pressure. A negative correlation was noted with O_3 concentration and minimal and maximal temperature. There were no significant correlations with concentrations of particulates, humidity, or airborne pollen and spores. An exceptionally high incidence of emergency room visits of asthmatic children was observed during September. This peak coincided with the beginning of the school year and the Jewish holydays, possibly associated with an increase in the number of viral infections and/or emotional stress. The correlations between emergency room visits and the environmental factors increased significantly when the September peak was excluded, revealing that 61 percent of the variance in emergency room visits was explained by NO_x, SO_2, and O_3 concentrations, 46 percent by weather parameters, 66 percent by NO_x, SO_2, and barometric pressure, and 69 percent by the combination of air pollutants and weather parameters.

In 1980, Gross et al. (1984) examined both total visits and visits for respiratory conditions to the Soroka Medical Center to test whether rain, heat, or pollutants led to increased requests for emergency room care. This hospital, located in Beer-Sheva, delivers medical services to the entire population of the southern part of Israel. Total visits for adults were 72,375, of which 1,727 (2.4 percent) were for selected respiratory conditions. Total visits for children were 19,232, and respiratory conditions were 3,980 (20.7 percent). There was a marked seasonal excess of respiratory visits for children in winter, and a lesser excess for adult respiratory conditions. Non-respiratory conditions and overall visits were higher in summer months. Fewer visits occurred on Saturday and on Friday, with the maximum number of visits on Sunday. No day-of-week trend could be noted for otitis and bronchitis among children. "High event" days for admissions were determined by fitting a Poisson distribution to the numbers of admissions by day for various respiratory complaints. The concordance between these high event days and days with rain, high pollution, or temperature was then tested. Adult respiratory conditions were more likely to occur on days with high levels of total suspended particulates (TSP).

In 1991, Porath et al. (1995) studied the influence of seasonality upon the characteristics of patients admitted to the general medical wards of Soroka hospital. The study took place during a 21-month period between January 1990 and September 1991. Cardiovascular and respiratory disease-related admissions accounted for 46.8 percent of the 18,774 admissions. Using a linear model, Porath et al. examined the relationship between seasonal and clinical characteristics of the patients admitted with these diagnoses. The number of admissions was higher during the winter (December-February) than the summer (June-August). The specific ratio of these hospitalizations, as compared to the other causes of admission, was 0.75 in the summer, 0.93 in the spring, 1.01 in the winter, and 0.82 in the autumn ($p < 0.0001$). Seasonality affected more patients with coronary heart disease, non-coronary cardiac disease, and chronic pulmonary disease than those with pneumonia or other pulmonary diseases, or cerebro-vascular events. Those admitted during the winter were older, had more than a single cardio-vascular or respiratory condition, and had longer hospital stays.

A study conducted by Cohen et al. (in press) during 1996-1999 found that total emergency room visits and admissions were associated during summer with an increase in the *Index100* index (the average of the median concentrations of the various air pollution indicators during the day).[7] A rise in its level of 10 $\mu g/m^3$ was associated with an increment of 4 percent (95 percent confidence interval of 1 percent to 7 percent) for total emergency room visits and admissions, and an increment of 7 percent (95 percent confidence interval of 3 percent to 13 percent) for admissions. Increasing concentrations of PM_{10} and SO_2 by 10 $\mu g/m^3$ during the day were associated with an increase of 2.5 percent and 26 percent, respectively, of the total emergency room visits.

[7] Index 100, was developed by Haifa District Municipal Association for the Environment according to U.S. EPA Pollutant Standard Index (PSI). The Index 100 was calculated as the difference between the PSI and 100. PSI is calculated relatively to the Israeli standards for ozone, particulate matter, sulfur dioxide and nitrogen oxides. A negative Index 100 indicates poor air quality. This index takes into account a "worst case" of air pollution.

A study was conducted in Tel Aviv (Goren et al., unpublished data) from September 1992 to August 1993 to assess the association between air pollution and the number of visits to emergency rooms of children with respiratory problems, age 0-18, living in the Tel Aviv-Jaffa urban area. The collected data included daily concentration levels of air pollutants together with meteorological and demographic variables for the more than 12,000 children who took part in this study. A multiple regression model was employed to analyze the results. A significant difference was found in the frequency of emergency room visits for respiratory diseases in the area of Tel Aviv. The number of visits was highest in the Bavli neighborhood. A negative correlation was found between the minimum temperature and the number emergency room visits. No significant correlation was observed with environmental parameters (e.g., air pollution, meteorology, etc.).

Mortality

Studies have been conducted in Israel to assess the extent of the association between air pollution and mortality. Ginsberg et al. (1998) developed a predictive model for assessing particulate matter-related mortality. They estimated that suspended particulate matter produced by tailpipe emissions in Tel Aviv-Jaffa alone accounted for 293 premature deaths each year, primarily among the elderly. An estimated ten additional annual deaths were related to refinery or power plant particulate emissions.

An evaluation done by Gabrieli (unpublished data) based upon the methodology used in the "Six Cities Study"[8] found a significant correlation between air pollution from suspended particulate matter and overall mortality. He calculated that a reduction in the concentration of suspended particulate of 20 $\mu g/m^3$ would reduce the mortality rate in Israel by 192 annual deaths per million, resulting in a total of 1,056 avoidable deaths per year.

A large multi-center study, known as the Air Pollution and Health European Approach (APHEA) project, has been initiated by the European Community. Fifteen cities from ten different European countries have initially taken part in this study, with all the research teams using the same methodology. After the first stage, cities in Eastern Europe, the United States, and Israel (Tel Aviv) were included in this study. The results of the first stage of the APHEA project show a positive association between the increase in air pollution levels and overall mortality rate, as well as with the number of hospital admissions related to respiratory and cardiovascular diseases.

Cardio-Vascular Morbidity

Studies in this field have been relatively few and limited by methodological problems. When assessing variations in cardio-vascular and respiratory mortality, there is a need to take into account the presence of strong confounders evidenced these last years in Israel. For example, ethnic origin in Israel has a critical influence on morbidity and mortality from this group of diseases (Bobak et al., 1999; Green et al., 1985). Changing epidemiological patterns have resulted from the integration of a huge wave of immigrants from the former Soviet Union since 1990, representing more than 10 percent of the present Israeli population (Rennert, 1994). Recent research has also demonstrated the significant relationship in Israel between variations of space proton flux[9] and cardio-vascular mortality and morbidity trends, and opened new previously unexplored fields for epidemiological research (Stoupel et al., 1995; Stoupel et al., 1997; Stoupel et al., 2000).

[8] Dockery DW, Pope CA 3rd, Xu X, Spengler JD, Ware JH, Fay ME, Ferris BG Jr., Speizer FE., An association between air pollution and mortality in six U.S. cities, N Engl J Med 1993 Dec 9;329(24):1753-9.

[9] The influence of solar activity (SA) and geomagnetic activity (GMA) on human homeostasis has long been investigated. Monthly levels of SA, GMA, and radiowave propagation (Fof2) are significantly and adversely correlated with monthly space proton flux. This offers a tool to appreciate the changes in the levels of exposure to natural cosmic radiation.

APPENDIX F:

REFERENCES

Allred EN, Bleecker ER, Chaitman BR, Dahms T.E, Gottlieb SO, Hackney JD, Pagano M, Selvester RH, Walden SM, Warren J (1989) Short-term effects of carbon monoxide exposure on the exercise performance of subjects with coronary artery disease. N Engl J Med, 321: 1426-1432.

Allred EN, Bleecker ER, Chaitman BR, Dahms TE, Gottlieb SM, Hackney JD, Pagano M, Selvester RH, Walden SM, Warren J (1991) Effects of carbon monoxide on myocardial ischemia. Environ. Health Perspect, 91: 89-132.

Argonne National Laboratory (1996) A Probabilistic Assessment of Health Risks Associated with Short-Term Exposures to Tropospheric Ozone (AIN/DIS-3). Prepared for U.S. EPA.

Bobak M, Hense HW, et al. (1999) An ecological study of determinants of coronary heart disease rates: a comparison of Czech, Bavarian and Israeli men. Int J Epidemiol, 28: 437-44.

Burnett RT, Cakmak S, Brook JR, Krewski D (1997) The role of particulate size and chemistry in the association between summertime ambient air pollution and hospitalization for cardiorespiratory diseases. Environ Health Perspect. 105(6): 614-20.

Burnett RT, Smith-Doiron M, Stieb D, Cakmak S, Brook JR (1999) Effects of particulate and gaseous air pollution on cardiorespiratory hospitalizations. Arch. Environ, Health 54(2): 130-9.

Cohen A, Doveh E, et al. (2002, in publication) Short term associations between air pollution and daily counts of hospital admissions and emergency room visits in the Haifa Metropolitan area (1996-1999).

Colin A, Said E, Winter ST (1985) Exercise induced asthma in schoolchildren. A pilot study in Haifa districts. Isr J Med Sci, 21: 40-43.

Dixon, WJ (ed.) (1983) BMDP Statistical Software (Vol. 1, 3rd ed.), Berkeley, CA: University of California Press.

Dockery DW, Pope CA III, Xu X, Spengler JD, Ware JH, Fay ME, Ferris BG Jr., Speizer FE (1993) An association between air pollution and mortality in six U.S. cities. N. Engl. Med. 329: 1753-1759.

Epstein L, Cohen A, et al. (1991) Air pollution and morbidity in the Haifa region. Harefuah, 120: 709-714 (in Hebrew).

Ferris BG (1978) Epidemiology standardization project. Am Rev Respir Dis, 118(6): 1-120.

Folinsbee LJ, McDonnell WF, Horstman DH (1988) Pulmonary function and symptom responses after 6.6-hour exposure to 0.12 ppm ozone with moderate exercise. JAPCA. Jan, 38(1): 28-35.

Gabbay S (1998) The Environment in Israel. Israel Ministry of the Environment, Jerusalem, Israel: 85-86.

Graber, M (Summer 1995) Transition to Clean Technologies Through Implementation of Emission Standards. Israel Environment Bulletin, Israel Ministry of the Environment, Vol. 18, No. 3: 21-24.

Garty BZ, Kosman E, et al. (1998) Emergency room visits of asthmatic children, relation to air pollution, weather, and airborne allergens. Ann Allergy Asthma Immunol. 81: 563-70.

Ginsberg G., Karsenty E, et al. (1998) Mortality from vehicular particulate emissions in Tel-Aviv-Jaffa. World Transport Policy and Practice, 4: 27-31.

Goren A, Bibi H, Goldshmidt JR (1991) Prospective lung health monitoring in relation to a new power plant. Public Health Rev, 19: 103-108.

Goren A, Hellman S, et al. (1999) Respiratory problems associated with exposure to airborne particles in the community. Arch Environ Health, 54: 165-171.

Goren AI, Brenner S, Hellmann S (1988) Cross-sectional health study in polluted and non-polluted agricultural settlements in Israel. Environ Res, Aug, 46(2): 107-19.

Goren AI, Goldsmith JR (1986) Epidemiology of childhood respiratory disease in Israel. Eur J Epidemiol, 2: 139-50.

Goren AI, Goldsmith JR, Hellmann S, Brenner S (1991) Follow-up of schoolchildren in the vicinity of a coal-fired power plant in Israel. Environ Health Perspect, Aug, 94: 101-5.

Goren AI, Hellman S (1995) Respiratory conditions among schoolchildren and their relationship to environmental tobacco smoke and other combustion products. Arch Environ Health, 50: 112-115.

Goren AI, Hellman S, Brenner S, Egoz N, Rishpon S (1990) Prevalence of respiratory conditions among schoolchildren exposed to different levels of air pollutants in the Haifa Bay area, Israel. Environ Health Perspect, Nov, 89: 225-31.

Goren AI, Hellman S, Glaser ED (1995) Use of outpatient clinics as a health indicator for communities around a coal fired power plant. Environ Health Perspect, 103: 1110-1115.

Goren AI, Hellmann S (1988) Prevalence of respiratory symptoms and diseases in schoolchildren living in a polluted and in a low polluted area in Israel. Environ Res, Feb, 45(1): 28-37.

Goren AI, Hellmann S (1993) Reporting bias related to an environmental hazard. J Expo Anal Environ Epidemiol, S1: 211-227.

Goren AI, Hellmann S (1997) Has the prevalence of asthma increased in children? Evidence from a long-term study in Israel. J Epidemiol Community Health, Jun, 51(3): 227-32.

Goren AI, Hellmann S, et al. (1990) Prevalence of respiratory conditions among schoolchildren exposed to different levels of air pollutants in the Haifa Bay area, Israel. Environ Health Perspect, 89: 225-31.

Goren AI, Hellmann S, et al. (1999) Respiratory problems associated with exposure to airborne particles in the community. Arch Environ Health , 54: 165-71.

Goren AI, Helman S, Goldsmith JR (1988) Longitudinal study of respiratory conditions among schoolchildren in Israel: interim report of an epidemiological monitoring program in the vicinity of a new coal-fired power plant. Arch Environ Health, Mar-Apr, 43(2): 190-4.

Green MS, Jucha E,Luz Y (1985) Ethnic differences in selected cardiovascular disease risk factors in Israeli workers. Isr J Med Sci, 21:808-16.

Gross J, Goldsmith Jr, et al. (1984) Monitoring of hospital emergency room visits as a method for detecting health effects of environmental exposures. Sci Total Environ, 27: 289-302.

Horstman DH, Folinsbee LJ, Ives PJ, Abdul-Salaam S, McDonnell WF (1990) Ozone concentration and pulmonary response relationships for 6.6-hour exposures with five hours of moderate exercise to 0.08, 0.10, and 0.12 ppm. Am Rev Respir Dis, 142(5): 1158-63.

Hull CH, Nie NH (1981) SPSS UPDATE 7-9: New Procedures and Facilities for Releases 7-9. New York: McGraw-Hill.

Israel Central Bureau of Statistics (2001) Statistical Abstracts of Israel 2000. http://www.cbs.gov.il/shnaton51.

Israel Central Bureau of Statistics (in print) 1996 Traveling Habits Survey.

Israel Ministry of Foreign Affairs (1994) Abatement of Nuisances Law, 1961 (English Translation). http://www.israel.org/mfa/go.asp?MFAH0aww0.

Israel Ministry of Foreign Affairs (1999) Israel's Air Resources Management Program. http://www.israel.org/mfa/go.asp?MFA00ib0.

Israel Ministry of the Environment (1998) Environmental Legislation and Enforcement. http://www.environment.gov.il/Eng-site/Legislation_and_Enforcement.html.

Israel Ministry of the Environment (2001) Bilateral Cooperation. http://www.environment.gov.il/Eng-site/Env_Org/org_frame.htm.

Israel Ministry of the Environment (Winter 1998) Covenant on Air Pollution Abatement. Israel Environment Bulletin, Vol. 21, No. 1: 8-12.

Israel Union for Environmental Defense (1993) Israel's Environmental Laws. Prepared for Israel Ministry of the Environment, Jerusalem, Israel: C6-C8.

Ito K, Thurston GD (1996) Daily PM10/mortality associations: an investigation of at-risk subpopulations. J Expo Anal Environ Epidemiol, 6(1): 79-95.

Johns Hopkins University (1996) Interactive Respiratory Physiology.

Kinney PL, Ito K, Thurston GD (1995) A sensitivity analysis of mortality/PM10 associations in Los Angeles. Inhalation Toxicol, 7: 59-69.

Lee K, Xue J, Geyh AS, Özkaynak H, Leaderer BP, Weschler CJ and Spengler JD. Nitrous Acid, Nitrogen Dioxide, and Ozone Concentrations in residential Environments. Environmental Health Perspectives, 110: 145-150, 2002.

Lee KY,Vallarino J, Dumyahn T, Özkaynak H, and Spengler JD (1999) Ozone Decay Rates in Residences. J. Air Waste Manag. Assoc. 49:1238-1244,.

Linn WS, Avol EL, Peng RC, Shamoo DA, Hackney JD (1987) Replicated dose-response study of sulfur dioxide effects in normal, atopic, and asthmatic volunteers. Am Rev Respir Dis, 136(5): 1127-34.

Linn WS, Avol EL, Shamoo DA, Peng RC, Spier CE, Smith MN, Hackney JD (1988) Effect of metaproterenol sulfate on mild asthmatics' response to sulfur dioxide exposure and exercise. Arch Environ Health, 43(6): 399-406.

Linn WS, Shamoo DA, Peng RC, Clark KW, Avol EL, Hackney JD (1990) Responses to sulfur dioxide and exercise by medication-dependent asthmatics:effect of varying medication levels. Arch Environ Health, 45(1): 24-30.

McDonnell WF, Kehrl HR, Abdul-Salaam S, Ives PJ, Folinsbee LJ, Devlin RB, O'Neil JJ, Horstman DH (1991) Respiratory response of humans exposed to low levels of ozone for 6.6 hours. Arch Environ Health, 46(3): 145-50.

National Research Council (1994) Science and Judgment in Risk. National Academy Press, Washington, DC.

Nie NH, Hull, CH, Jenkins JG, Steinbrenner K, Bent DH (1975) SPSS: Statistical Package for the Social Sciences. Second edition. New York: McGraw-Hill

Özkaynak H (1999) Exposure Assessment. In: Air Pollution and Health. Holgate, Koren, Samet and Maynard, Eds. Academic Press, London UK. 149-162.

Peled R, Bibi H, et al. (2001) Differences in lung function among school children in communities in Israel. Arch Environ Health, 6: 89-95.

Pope CA III, Dockery DW, Schwartz J (1995) Review of epidemiological evidence of health effects of particulate air pollution. Inhalation Toxicol. 7: 1-18.

Pope CA III, Schwartz J, Ransom MR (1992) Daily mortality and PM10 pollution in Utah Valley. Arch Environ. Health, 47(3): 211-7.

Porath A, Grinberg G, et al. (1995) Seasonal variation in hospital admissions for cardiorespiratory diseases in the Negev. Harefuah, 128:207-10 (in Hebrew).

Rennert G (1994) Implications of Russian immigration on mortality patterns in Israel. Int J Epidemiol, 23: 751-6.

Richmond H (July 2, 1997) Corrections to Risk Analysis Tables and Figures in the PM Staff Paper. Memorandum to the U.S. EPA PM docket.

Roger LJ, Kehrl HR, Hazucha M, Horstman DH (1985) Bronchoconstriction in asthmatics exposed to sulfur dioxide during repeated exercise. J Appl Physiol, 59(3): 784-91.

Samet JM, Zeger SL, Kelsall JE, Xu J, Kalkstein LS (1997) Particulate air pollution and daily mortality: analysis of the effects of weather and multiple air pollutants. The phase I.B report of the Particle Epidemiology Evaluation Project, Cambridge, MA. Health effects Institute, HEI special report.

Schwartz J (1993a) Air pollution and daily mortality in Birmingham, Alabama. Am J Epidemiol, 137: 1136-1147.

Schwartz J (1993b) Particulate air pollution and chronic respiratory disease. Environ Res, 62:7-13.

Schwartz J (1995) Short-term fluctuations in air pollution and hospital admissions of the elderly for respiratory disease. Thorax, 50: 531-538.

Schwartz J (1996) Air pollution and hospital admissions for respiratory disease. Epidemiology, 7: 20-28.

Schwartz J (1999) Air pollution and hospital admissions for heart disease in eight U.S. counties. Epidemiology, 10(1): 17-22.

Schwartz J, Dockery DW, Neas LM (1996a) Is daily mortality associated specifically with fine particles? J. Air Waste Manage. Assoc. 46(10): 927-39.

Schwartz J, Dockery DW, Neas LM, Wypij D, Ware JH, Spengler JD, Koutrakis P, Speizer FE, Ferris BG Jr (1994) Acute effects of summer air pollution on respiratory symptom reporting in children. Am J Respir. Crit Care Med, 50(5 Pt 1): 1234-42.

Schwartz J, Morris R (1995) Air pollution and hospital admissions for cardiovascular disease in Detroit, Michigan. Am J Epidemiol, 42(1): 23-35.

Schwartz J, Slater D, Larson TV, Pierson WE, Koenig JQ (1993) Particulate air pollution and hospital emergency room visits for asthma in Seattle. Am Rev Respir Dis, 147(4): 826-831.

Schwartz J, Spix C, Touloumi G, Bacharova L, Barumamdzadeh T, Le Tertre A, Piekarksi T, Ponce de Leon A, Ponka A, Rossi G, Saez M, Shouten JP (1996b) Methodological issues in studies of air pollution and daily counts of deaths or hospital admissions. J Epidemiol Commun Health, Apr, 50, Suppl 1: S3-11.

Stoupel E, Abramson E, et al. (1995) Relationship between suicide and myocardial infarction with regard to changing physical environmental conditions. Int J Biometeorol, 38: 199-203.

Stoupel E, Abramson J, et al. (1997) Space proton flux and the temporal distribution of cardiovascular deaths. Int J Biometeorol, 40: 113-6.

Stoupel E, Israelevich P, et al. (2000) Correlation of two levels of space proton flux with monthly distribution of deaths from cardiovascular disease and suicide. J Basic Clin Physiol Pharmacol. 11:63-71.

Taback HJ, Bazes J (2000) Current Status of Air Quality in Tel Aviv, Israel. Presented at the June 2000 Annual Meeting of A&WMA, Salt Lake City, Utah, USA.

The Lungs Clinic, Rabin Medical Center (In Hebrew). http://www.lung-rmc.co.il.

Thurston GD, Ito K, Hayes CG, Bates DV, Lippmann M (1994) Respiratory hospital admissions and summertime haze air pollution in Toronto, Ontario: consideration of the role of acid aerosols. Environ Res, 65(2): 271-90.

USEPA (1996a) Review of National Ambient Air Quality Standards for Ozone: Assessment of Scientific and Technical Information. OAQPS Staff Paper, Office of Air Quality Planning and Standards, Research Triangle Park, NC, EPA report no. EPA-452\R-96-007, June.

USEPA (1996b) Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information. OAQPS Staff Paper, Office of Air Quality Planning and Standards, Research Triangle Park, NC, EPA report no. EPA-452\R-96-013, July.

USEPA (1999) The Benefits and Costs of the Clean Air Act, 1970 to 1990: Report to Congress, Appendix D. U.S. Environmental Protection Agency, Washington, DC.

Weisel CP, Cody RP, Lioy PJ (1995) Relationship between summertime ambient ozone levels and emergency department visits for asthma in central New Jersey. Environ Health Perspect,103 Suppl 2: 97-102.

WHO (1999) Guidelines for Air Quality. World Health Organization, http://www.who.int/peh/air/Airqualitygd.htm.

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[illegible]:

1. [illegible] $PM_{2.5}$ [illegible] $PM_{2.5}$ [illegible].
2. [illegible] O_3 [illegible] O_3 (NO_2 [illegible]).
3. [illegible].
4. [illegible].
5. [illegible].
6. [illegible] $PM_{2.5}$ [illegible] O_3.

[illegible]:

7. [illegible] $PM_{2.5}$ [illegible].
8. [illegible].
9. [illegible].
10. [illegible].

[illegible] ,6% [illegible] 65 [illegible] PM_{10} [illegible]
[illegible] 65 [illegible] .1997 [illegible] 800
[illegible] 1,220 [illegible] ,3% [illegible] PM_{10} [illegible] (cardiovascular causes
.1997-1995 [illegible] PM [illegible] .1997 [illegible]

[illegible] 65 [illegible] ,1997 [illegible]
[illegible] 8% [illegible]
1997 [illegible] 1995 [illegible] 180 [illegible] 1,120 [illegible] 2%
[illegible]

[illegible] (SO_2) [illegible]
[illegible] 1000 [illegible] 6% [illegible]
.1997 [illegible] 200 [illegible]

[illegible] (NO_2) [illegible]
[illegible] .1997 [illegible] 60 [illegible] 615 [illegible] 2% [illegible] 4% [illegible]
[illegible] .1997 [illegible] 1995 [illegible]
[illegible] NO_2 [illegible] SO_2 [illegible]
[illegible] (PM) [illegible]

[illegible] 28,000 [illegible] ,20% [illegible]
[illegible] 1999-1997 [illegible]
1999-1997 [illegible] 20,000 [illegible] ,14% [illegible] .PM_{10}
-1998 [illegible] 6,000 [illegible] ,20% [illegible] .$PM_{2.5}$ [illegible]
[illegible] 4,400 [illegible] 15% [illegible] ,PM_{10} [illegible] ,1999
[illegible] 1% [illegible] .$PM_{2.5}$ [illegible]
.SO_2 [illegible]

[illegible] "best estimates" [illegible]
[illegible]
[illegible]
[illegible] 65 [illegible]

[illegible]

òÝØÛÝÝ-áØ

åáØîææ

1995 - 1999 åáèñä äØðñáÙ åááèÝðáê åáðÝÞØ áèñÙ ðáÝÝØ åÝÜáÞæ áòØÝÝñÜ åáèÝãáé ðïé

[illegible]

åáÛêáÝ Üðàæ

[illegible] (1) [illegible] (2) [illegible] .(NO$_2$) [illegible] (SO$_2$) [illegible] ,(O$_3$) [illegible] ,(PM - particulate matter) [illegible] 1.2 [illegible]

World) [illegible] 8 [illegible] ,(Health Organization, 1999 [illegible] .2020 [illegible]

[illegible] USEPA – US Environmental Protection) [illegible] .(Agency [illegible] .EPA [illegible]

[illegible]

òÝàáñ

[illegible] [1] [illegible]

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[1] [illegible] .("concentration response") [illegible]

סקר סיכונים השוואתי מזיהום אוויר
באזורי תל-אביב ואשדוד
לשנים 1995 - 1999

ינואר 2003

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