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POLICY RESEARCH WORKING PAPER 1301

Estimating the Health Effects of Air Pollutants

A Method with an Application to Jakarta

Bart Ostro

How does one assess the health benefits of air pollution control? Does response functions applied to data on Jakarta reveal that air quality improvements will reduce illness, premature death, and learning disabilities in children. Lead and respirable particles are the most important problems.

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Summary findings

To develop efficient strategies for pollution control, it is essential to assess both the costs of control and the benefits that may result. These benefits will often include improvements in public health, including reductions in both morbidity and premature mortality.

Until recently, there has been little guidance about how to calculate the benefits of air pollution controls and how to use those estimates to assign priorities to different air pollution control strategies. Ostro describes a method for quantifying the benefits of reduced ambient concentrations of pollutants (such as ozone and particulate matter) typically found in urban areas worldwide. He then applies the method to data on Jakarta, Indonesia, an area characterized by little wind, high population density (8 million people), congested roads, and ambient air pollution.

The magnitude of the benefits of pollution control depend on the level of air pollution, the expected effects on health of the pollutants (dose-response), the size of the population affected, and the economic value of these effects.

The results for Jakarta suggest that significant benefits result from reducing exposure to both outdoor and indoor air pollutants. For example, if annual concentrations of particulate matter were reduced to the midpoint of the World Health Organization guideline (and former U.S. ambient standard), the estimates indicate a reduction per year of 1,400 premature deaths (with a range of 900 to 1,900), 49,000 emergency room visits, 600,000 asthma attacks, 7.6 million restricted-activity days (including work loss), 124,000 cases of bronchitis in children, and 37 million minor respiratory symptoms.

In the case of Jakarta, the methodology suggests that reducing exposure to lead and nitrogen dioxide should also be a high priority.

An important consequence of ambient lead pollution is a reduction in learning abilities for children, measured as I.Q. loss. Apart from that, reducing the proportion of respirable particles can reduce the amount of illness and premature mortality.

Clearly, air pollution represents a significant public health hazard to residents of Jakarta and other cities consistently exposed to high levels of air pollution, such as Bangkok, Mexico City, and Santiago, Chile.

This paper—a product of the Public Economics Division, Policy Research Department—is part of a larger effort in the department to analyze the economics of pollution control in developing countries. The study was being funded by the Bank's Research Support Budget under the research project "Pollution and the Choice of Economic Policy Instruments in Developing Countries" (RPO 676-48). Copies of the paper are available free from the World Bank, 1818 H Street NW, Washington, DC 20433. Please contact Carlina Jones, room N10-063, extension 37699 (63 pages). May 1994.

Estimating the Health Effects of Air Pollutants
A Method with an Application to Jakarta

by

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I. INTRODUCTION

Until recently, there has been little guidance about the calculation of the social costs of air pollution and about using these costs to evaluate alternative air pollution control strategies. With limited resources, rational decision-making requires some quantification of the potential benefits of controlling air pollution. These benefits are dependent on the expected health effects of the pollutant, the magnitude of the effect in response to air pollution (dose-response), the economic valuation of the adverse effect, and the existence of subpopulations particularly sensitive to air pollution. Information about health and economic effects of air pollution needs to be categorized for pollutants commonly discharged by mobile and stationary sources. This report describes a method for determining quantitative estimates of the benefits of reducing ambient concentrations of five pollutants: particulate matter, sulfur dioxide, nitrogen dioxide, ozone and lead. This methodology is then applied to Jakarta, Indonesia. A brief review of the effects of carbon monoxide and carbon dioxide is also provided. Once the benefits (both quantified and unquantified) of control are calculated, they can be incorporated in decisions about prioritizing control strategies. For cost-benefit analysis of air pollution control, a common denomination for various health effects would be used. It could be based on willingness to pay, medical treatment costs and the value of lost productive days and years. Such valuation is beyond the scope of this paper, however.

It should be acknowledged, however, that large uncertainties about the existence and magnitude of the health effects of air pollution continue to exist. Therefore, the analysis provided below should be viewed as an attempt to present, in the judgement of this author, the most likely and well-documented health impacts for which quantitative information exists. This assessment will probably change over time as new clinical, epidemiologic and economic research is completed.

In the past, the U.S. Environmental Protection Agency has estimated the health and welfare effects of air pollution in its Regulatory Impact Analysis for national ambient air quality standards, as required by the Presidential Executive Order #12291 issued in 1981.[1] Additional information and methodological improvements were incorporated in the subsequent analysis of economic benefits of air

quality programs in selected U.S. locations.[2,3] Recently, broad estimates of the health benefits of controlling ozone and particulate matter were provided for both the U.S. and for the ambitious control plans being considered in Southern California. [4,6]

The analysis reported here uses a similar approach to estimate health effects of criteria pollutants in Jakarta, with two improvements: the most recent set of research findings are utilized and a full range of health endpoints are included. Dose-response functions that relate various health outcomes to air pollution are taken from the available peer reviewed literature. Estimates of selected health effects of air pollution are generated by applying these functions to ambient levels either observed from monitoring stations located throughout the city or estimated from available dispersion models. Using results from both time-series and cross-sectional epidemiologic analyses from the United States, Canada, and Britain, effects are estimated for such health outcomes as premature mortality, hospital visits and admissions, emergency room visits, restrictions in activity, acute respiratory symptoms, acute bronchitis in children, asthma attacks, IQ loss, and blood pressure changes. At this time, however, because of uncertainties about the coverage and representativeness of the existing monitors in the city, and about the applicability of health studies undertaken in the U.S. to the developing world, the results should be viewed as providing only general estimates of the impacts of air pollution.

Following this introduction, there are four sections in this report. Section II describes the methodology for estimating the health effects associated with changes in air pollution. The section also details the data and baseline assumptions that are necessary for such estimations. Section III provides a brief review of the literature that quantitatively links changes in air pollutants with adverse health outcomes. From this review, dose-response functions, along with associated confidence intervals, are developed. Also, suspected health effects for which quantitative estimates cannot be provided, are

indicated. Section IV applies the methodology and provides estimates of health effects of air pollution for Jakarta. Section V discusses the results and indicates the research and data on developing countries needed to enhance the accuracy of these estimates.

II. METHODOLOGY AND BASELINE ASSUMPTIONS FOR ESTIMATING HEALTH EFFECTS

A. Methodology

The estimation of the health and economic effects of air pollution involves the use of methodology similar to that used by the U.S. Environmental Protection Agency (EPA) in their Regulatory Impact Analysis for a new national air quality standard for particulate matter.[1] Estimation techniques are also derived from the analyses of economic benefits of air quality control programs in selected U.S. locations.[2-4] To estimate the economic value associated with changes in air pollution, four factors must be determined: the dose-response relationships, the susceptible populations, the relevant change in air pollution, and an economic valuation of the health endpoints. In this paper, health effects for a range of health outcomes are provided, while valuation of these is not performed.

The first step is to develop estimates of the effects of air pollution on various health outcomes. Dose-response functions that relate health impacts to ambient levels of air pollution are taken from the published epidemiologic literature. This step involves calculating the partial derivative (or slope, b) of the dose-response function, to provide an estimate of the change in the prevalence of a given health effect associated with a change in outdoor air quality (A). Sufficient information is provided in this report to understand the sources of the selected dose-response functions, but a more complete review of the literature can be obtained in the EPA scientific review of the health effects of criteria pollutants.[7]

The next step involves multiplying this slope by the relevant population that is believed to be exposed and susceptible to the air pollutant effect under consideration (POP). For certain pollution-related health effects this may include the entire exposed population; for other effects there may be particularly sensitive subgroups such as children or asthmatics.

A third step in the calculation of health effects of air pollution involves the change in air quality (dA) under consideration. The actual change is dependent on both the policy issue under consideration and the available data. For example, it may be relevant to consider the change from current air pollution levels to some ambient air quality standard, either a local one, the EPA standard, or the WHO air quality guideline. A second change that might be relevant for consideration is a given percent reduction, such as 10 percent. A third method of determining the relevant change in air pollution is to assume that air quality changes in some simple proportion to the change in emissions, as in a simple linear rollback model. In that case, a 10 reduction in the total tonnage of particulate emissions, for example, is assumed to reduce ambient particulate air pollution and health effects by 10 percent. Finally, the ambient changes associated with a given change in a stationary or area-wide pollution source can be calculated through use of computer models, if the necessary data are available.

In this report, we examine a change from existing levels to several alternative ambient standards, including: (1) proposed Indonesian standards, (2) EPA ambient air quality standards, (3) WHO guidelines, and (4) California state ambient air quality standards. For the case of lead, we also calculate the benefits associated with a 90 percent reduction of ambient lead, assumed to be accomplished through a ban on leaded gasoline. The relevant standards, expressed in terms of the annual average concentration in micrograms per cubic meter ($\mu g/m^3$), are as follows:

Ambient Air Quality Standards for Annual Averages (micrograms/m^3)

Pollutant	Proposed Indonesian	EPA	WHO	California
Total Suspended Particles (TSP)	90	75*	60-90	55#
Lead	0.5	N/A	0.5-1.0	N/A
Nitrogen Dioxide	100	100	N/A	N/A
Sulfur Dioxide	60	80	50	N/A
Ozone	200	240	150	180

*In 1986, the EPA standard was replaced by a PM10 standard. # The California standard has been converted from the current PM10 standard to a TSP equivalent for the purpose of this analysis. N/A signifies standards with averaging time other than annual average.

With this information, the estimated health impact can be represented as follows:

$$dH_i = b * POP_i * dA$$

where: dH_i = change in population risk of health effect i

b_i = slope from dose-response curve

POP_i = population at risk of health effect i

dA = change in air pollution under consideration

To complete benefit estimation for health effects, one would calculate the economic valuation of this effect (V_i), as well. The valuation could be developed from estimates of the willingness to pay (WTP) for reducing risk, in order to attach values to the expected changes in premature mortality, or a modified cost of illness (COI) approach, to value changes in morbidity. Thus, the change in total social

value (dT) of the health effects due to the change in air pollution under consideration is the summation of all effects and can be represented by:

$$dT = \Sigma V_i dH_i$$

Unfortunately, there is still a great deal of uncertainty and controversy about much of the research on which these estimates are based. Recognizing this uncertainty, upper and lower bound estimates are provided to indicate the ranges within which the actual health effects are likely to fall. In addition, the categories presented in this paper are not all-inclusive, since quantitative evidence is not available for every health effect suspected of being associated with air pollution. Also, air pollution has been associated with non-health effects, including materials damage, soiling, vegetation losses and visibility degradation.

These types of omissions suggest that the results of this analysis are likely to underestimate the health effects, and will certainly underestimate the total effects, resulting from air pollution.

B. Baseline Assumptions

An important question in all of the health effects estimates is whether a threshold level exists, below which health effects no longer occur, or whether the slope of the dose-response function diminishes significantly at lower concentrations. There is a presumption by some that a threshold exists at the EPA air quality standard, or at the WHO ambient guidelines for criteria pollutants. Most of the studies reported here have estimated linear or log-linear functions suggesting a continuum of effects down to the lowest levels, and have not specifically identified a threshold level. When efforts have been made to identify a threshold, little conclusive evidence has been found that one exists. In fact, many recent epidemiologic studies report an association between air pollution and health at ambient concentrations at or below the current federal standard. The former Administrator of the EPA has stated, "in a heterogeneous population it is unlikely that, for any pollutant, there will be a single scientifically defensible threshold applicable to all people. Instead, there will be a series of thresholds for different

sensitive populations and a threshold of zero for some people".[8] Therefore, for this report, we calculate the effects of bringing pollution down to alternative standards, without taking a position on what would happen at even lower pollution levels.

A basic assumption of the model is that the association between air pollution and health estimated in the cited studies can be applied to estimate the health impact in Jakarta. These studies show that when the readings at fixed site monitors change, there is a change in the observed incidence of many health effects. Although the monitors do not measure actual exposures, they do provide a general measure of air quality which is obviously related to ultimate exposure. The use of these results implicitly assumes a similar distribution of baseline factors - health status (e.g., incidence of chronic disease), chemical composition of pollutants, occupational exposures, seasonality, time spent out of doors, general activity - and that results from other studies can be applied to the study area. As described in greater detail in Section V, since the baseline health status in developing countries tends to be poorer than that experienced in the western, developed world, this assumption will likely lead to an underestimate of the more severe health outcomes. Another source of underestimation will be present since the population is assigned a pollution concentration based on their residential location. Effects of air pollution will be higher if the person commutes to the central business district and the subsequent higher exposures are incorporated into the analysis. Therefore, the quantitative assessment of health effects presented below are likely to be underestimates.

To the extent that the original studies were primarily time-series studies relating daily changes in air pollution to the daily incidence of a health effect, the likelihood of confounding from other factors is minimized. For example, if a study was conducted over a 3-month period, and daily emergency room visits were associated with PM10, it is extremely unlikely that a change in smoking habits, occupational exposure, diet, exercise and activity patterns, indoor exposure, etc. would change on a daily basis and be correlated with daily particulate matter enough to drive the observed association.

Our use of epidemiologic studies also assumes that the spatial relationship between pollution monitors and population that exists in the original studies is generally similar in Jakarta. Thus, with these assumptions, the relationship between the levels of air pollution and subsequent health effects in the cited studies can be extrapolated to estimate the health impact in Jakarta.

III. REVIEW OF HEALTH EFFECTS AND PROVISION OF DOSE-RESPONSE FUNCTIONS

A. Selection Criteria

For this report, dose-response functions have been identified and adapted from published epidemiologic and economics literature. These functions allow the estimation of the change in health effects that would be expected to occur with changes in ambient pollution levels. For each health effect, a range is presented within which the estimated effect is likely to fall. The central estimate is typically selected from the middle of the range reported in a given study, or is based on the most recent study using the most reliable estimation methods available. When several different studies are available for a given health effect, the range reflects the variation in results observed across the studies. When only one study is available, the range is based on the statistical confidence that can be placed on the reported results.

The reported epidemiologic investigations involve two principal study designs: statistical inference based on time-series and cross-sectional data sets. Time-series analysis examines changes in a health outcome within a specific area as air pollution levels fluctuate over time. A cross-sectional analysis compares the rate or prevalence of given health outcomes across several locations for a given point in time. The time-series studies have the distinct advantage of reducing or eliminating the problems associated with confounding or omitted variables, a common concern in the cross-sectional studies. Since the population characteristics are basically constant over the study period, the only factors that may vary with daily mortality are environmental and meteorologic conditions. In general, researchers are able to

more easily elicit the effects of air pollution and weather on health in the time-series studies. Therefore, this review focuses primarily on time-series studies. The use and extrapolation of results from time-series analysis, however, is predicated on its applicability to other areas and for other time periods.

Several specific criteria had to be met for a paper to be included in this review. First, a proper study design and methodology were required. Therefore, there was a focus on time-series regression analyses relating daily health effects to air pollution in a single city or metropolitan area. Second, studies that minimized confounding and omitted variables were included. For example, research that compared two cities or regions and characterized them as "high" and "low" pollution area were not included because of potential confounding by other factors in the respective areas. Third, concern for the effects of seasonality and weather had to be demonstrated. This could be accomplished by either stratifying and analyzing the data by season, by pre-filtering to reduce patterns in the data, by examining the independent effects of temperature and humidity, and/or by correcting the model for possible autocorrelation. A fourth criterion for inclusion was that the study had to include a reasonably complete analysis of the data. Such analysis would include an careful exploration of the primary hypothesis as well as an examination of the robustness and sensitivity of the results to alternative functional forms, specifications, and influential data points. When studies reported the results of these alternative analyses, the findings judged as most representative of the overall findings were those that were summarized in this paper. Fifth, for inclusion in this review, the study had to provide an air pollution measure that could be converted into a common metric. For example, studies that used weekly or monthly average concentrations or that involved measurements in poorly characterized metropolitan areas (e.g., one monitor representing a large region) were not included in this review. In addition, studies that used measures of particulate matter that could not be converted into total suspended particulates (TSP or particles of all sizes) or particulate matter below 10 microns in diameter (PM10) were not used. Sixth, the study had to involve relevant levels of

air pollution. The air pollution levels in Jakarta are well within the range of those observed in the epidemiologic studies used in this report.

B. Development of Dose-Response Estimates

1. Particulate Matter

Epidemiologic studies provide dose-response relationships between concentrations of ambient particulate matter and several adverse health outcomes including: mortality, respiratory hospital admissions, emergency room visits, restricted activity days for adults, lower respiratory illness for children, asthma attacks, and chronic disease. Among these studies, statistically significant relationships have been found using several alternative measures of particulate matter, including TSP, fine particles (particles less than 2.5 microns in diameter), British smoke, coefficient of haze (COH) and sulfates. Few have involved measurement of PM10, the metric used by the EPA in the national ambient air quality standard. The studies have been conducted in several different cities and seasons, thereby incorporating a wide range of climates, chemical compositions of particulate matter, and populations.

For comparison of results and the calculation of final dose-response functions, alternative measures of particulate matter were converted into PM10. Ideally, this would be accomplished by comparing co-located monitors at each study site. Unfortunately, for many of the measures, these data are not available and we are forced to use broad estimates of the relationships between alternative measures of particulate matter. The results of our analysis of consistency, however, indicates that the findings are generally robust to these assumptions. To convert from TSP to PM10, we relied on the EPA estimate [7] that PM10 is between 0.5 and 0.6 of TSP, and use the mean of 0.55. Using the reported averages from 100 cities in 1980, we assumed that sulfates constitute approximately 0.14 of TSP.[9] Therefore, the ratio of sulfates to PM10 is 0.25. The "British Smoke" (BS) measurement is based on the amount of light reflected through a filter paper stained by ambient air flowing through the paper. Since monitors for BS do not admit particles greater than 4.5 microns in diameter, they are indicators of

concentrations of fairly small particles, but they do not measure particle mass like TSP and PM10. However, available data for co-located BS and TSP monitors in London indicate an average ratio of BS/TSP of 0.55, the same as the ratio of PM10 to TSP.[10] Based on this and additional analysis by the California Air Resources Board,[11] it is assumed that PM10 is roughly equivalent to British smoke.

Premature Mortality. Since the effects of particulate air pollution on mortality is the source of such large potential benefits, the evidence for an effect and its potential magnitude will be reviewed in detail. The most relevant studies are reviewed below.

London. Among the earliest empirical estimate of mortality outcomes associated with particulate matter is the analysis of data from London for the winter of 1958-59, where a statistically significant relationship was found between daily deaths and daily levels (24-hour average) of British smoke. London data for 14 winters, 1958-59 through 1971-72 have been analyzed by Mazumdar et al.,[13] Ostro [14] and Schwartz and Marcus,[15-16] and reviewed by the U.S. EPA.[17] In the earlier winters, the levels of British smoke were extremely high; the mean for the first seven winters was 270 $\mu g/m^3$. However, the mean for the last seven winters was 80 $\mu g/m^3$, and in three of the last four years the mean concentrations were below 70 $\mu g/m^3$. The concentrations of British smoke in London in the last years are more comparable to those commonly found in the U.S.

Although these analyses involve several different statistical methods, the following general conclusions can be drawn: (1) there is a strong relationship between particulate concentrations and daily mortality in London, which holds both for the entire data set and for individual years (the later years exhibited almost an order of magnitude decrease in air pollution concentrations); (2) there is no indication of a "no effects level" (i.e., a threshold) at the lower concentrations of air pollution experienced in London; (3) the association between air pollution and mortality cannot be "explained away" by meteorologic factors or by serial correlation in the data; and (4) regardless of the model specified, the quantitative implications of the studies are very similar. For this review, quantitative estimates of the

London data are taken from Schwartz and Marcus[17] which involves the most complete examination of the effects of temperature and humidity, autocorrelation, and functional form. Their results over the 14 years suggest the following for all-cause mortality:

Daily mortality = 2.31 * (daily average BS)$^{0.5}$ in London

The standard error of the estimated regression coefficient was 0.150. During the period of study, there was an average of 280 deaths per day and a mean concentration of BS of 174 $\mu g/m^3$ in London. After taking the derivative of the above and substituting in the mean of daily deaths, the effects of PM10 can be expressed as:

% change in mortality = (0.4125 PM10^{-5}) * change in PM10

Thus, at the mean level of pollution, a 10 $\mu g/m^3$ increase in PM10 is associated with a 0.31 percent increase in mortality. Using plus or minus one standard error from the estimate, a confidence interval of 0.29 to 0.33 percent is obtained. This change is similar to that predicted from the linear models described above.

Ontario, Canada. Plagiannakos and Parker[18] used pooled cross-sectional and time-series data for nine counties in Southern Ontario, Canada, for the period 1976 to 1982. Their model attempted to explain mortality as a function of several socioeconomic factors (education, population over age 65, alcohol consumption), time, meteorology, and air pollution including TSP, sulfates, and sulfur dioxide. There was no correction for autocorrelation. Since mean ambient concentrations were not provided by the authors, graphical displays were used to estimate pollution levels. The mean for TSP appeared to be approximately 70 $\mu g/m^3$ while the mean for sulfate was approximately 12 $\mu g/m^3$.

A statistically significant association was found between all-cause mortality and sulfates, and between respiratory-related mortality and both TSP and sulfates. For all-cause mortality, the model with the highest association between air pollution and mortality, is represented by the following quantitative relationship:

log (annual mortality in Ontario) = 0.047 log (sulfate)

The standard error of the estimated coefficient is 0.0235. To estimate the change in mortality per μg/m3 of PM10, we take the total derivative of (3) and obtain:

% change in mortality = 0.047 (change in sulfate/mean sulfate)

Substituting the mean concentration of sulfates and the ratio of sulfate to PM10:

% change in mortality = 0.098 * change in PM10

This indicates that a change in PM10 of 10 $\mu g/m^3$ corresponds to a 0.98 percent change in all-cause mortality. Applying plus and minus one standard error, a 10 $\mu g/m^3$ change in PM10 generates an effect ranging from 0.49 to 1.47 percent.

Steubenville and Philadelphia. Two recent time-series studies[19,20] used similar methods to examine the association between daily mortality and TSP. Both studies controlled for the effects of weather and seasonality, and in both a statistically significant relationship was found between TSP and mortality. After converting to PM10 equivalence, the Steubenville study implies that a 10 $\mu g/m^3$ change in PM10 corresponds to a 0.64 percent increase in mortality over baseline. The confidence interval, based on plus or minus one standard error, is 0.44 to 0.94 percent. The Philadelphia study implies that a 10 $\mu g/m^3$ in PM10 is associated with a 1.20 percent increase in mortality, with a one standard error confidence interval of 0.96 to 1.44 percent.

Santa Clara County. A recent time-series analysis examined the relationship between coefficient of haze (COH) and mortality for the metropolitan area surrounding San Jose, California.[21] Daily mortality and suspended particles measured as COH were compared between 1980 and 1986. A statistically significant association was found between COH and both all-cause mortality and respiratory-related mortality, after controlling for temperature and humidity. The models were also tested for the influence of year, season, day, and weather, with little change in the overall results.

The general model for all-cause mortality, chosen by the author as most representative, is indicated the following:

Daily mortality in Santa Clara = 0.0084 COH

The standard error of the estimated coefficient was 0.0029.

To obtain the effect of a 10 $\mu g/m^3$ change in PM10 on the percent change in mortality, several adjustments must be made. First, as discussed by the author, only readings from the central city monitor were used in the study. This monitor averaged about one-third higher concentrations than the metropolitan-wide average, a metric typically used in the other studies. The author also found that the COH to TSP ratio was at least one. Therefore, we assume that PM10 is approximately .50*COH. Finally, the study population averaged about 20 deaths per day. Therefore, we adjust the coefficient by (4/3)(1/.5)(1/20) to obtain:

% change in mortality = 0.112 * change in PM10

This implies that a 10 $\mu g/m^3$ change in PM10 results in a 1.12% change in mortality. Applying the standard error, we obtain a range of effect of 0.73 to 1.51 percent associated with a 10 $\mu g/m^3$ change in PM10.

Ozkaynak and Thurston. Additional evidence is provided by a study using cross-sectional data. Ozkaynak and Thurston[9] examined roughly 100 metropolitan areas in the United States using the 1980 vital statistics. This study controlled for socioeconomic characteristics and conducted additional sensitivity analysis to determine the impact of certain cities and alternative model specifications. The authors found statistically significant relationships between mortality rates and alternative measures of ambient particulate matter including sulfates and fine particulates. Specifically, the study reports that existing sulfate concentrations (mean of 11.1 $\mu g/m^3$) correspond to a 4 to 9 percent increase in mortality. Assuming a ratio of sulfates to PM10 of 0.25 as above, this suggests that a 10 $\mu g/m3$ change in PM10 corresponds to a 0.92 to 2.06 percent change in all-cause mortality, with a mean of 1.49 percent.

Summary of mortality effect. This review suggests that the recent studies linking particulate matter to mortality generate remarkably consistent results[22,23] (see Table 1). The mean effect of a 10 $\mu g/m^3$ change in PM10 implied by these studies varies between 0.31 and 1.49 percent, with a mean of 0.96 percent. Several more recent studies support this magnitude of effect[24,25] and indicate that a 10 $\mu g/m^3$ change in PM10 relates to a 1 percent change in mortality. In our range of studies, the upper confidence level varies between 0.33 and 2.06 percent with a mean of 1.30 percent, with a mean lower bound of 0.62. Although similar studies have not been undertaken in Indonesia, there is one set of data available to test for the existence of an effect. Annual mortality and TSP in Bandung have been reported for 1983 through 1989.[26] Regressions run on these data suggest that a 10 $\mu g/m^3$ change in TSP is associated with a 0.695 percent change in mortality, if a crude mortality rate of 0.01 is assumed. This corresponds to 1.26 percent change in mortality associated with a 10 $\mu g/m^3$ in PM10. If the crude mortality rate is 0.007, a 10 $\mu g/m^3$ change in PM10 is associated with a 0.99 percent change in mortality. Regardless of the assumed crude mortality rate, the estimated air pollution effects are fairly similar to those derived from the studies summarized above. Therefore, we assume the following association:

Central percent change in mortality = 0.096 * change in PM10

Table 1: **Summary of Mortality Studies Indicating Percent Change in All-Cause Mortality Association with 10 $\mu g/m^3$ Change in PM10**

	Central Estimate	High Estimate*
London, UK	.31	.33
Ontario, Canada	.98	1.47
Steubenville, Ohio	.64	.94
Philadelphia, PA	1.2	1.44
Santa Clara County, CA	1.12	1.51
US Metropolitan Area	1.49	2.06

* A high (low) estimate is obtained by increasing (decreasing) the coefficient by one estimated standard deviation. The crude mortality rate in the U.S. is approximately 0.007.

The upper and lower percent changes in mortality have coefficients of 0.130 and 0.062, respectively. The central estimate for the number of cases of premature mortality, can be expressed as:

Change in mortality = 0.096 * change in PM10 * (1/100) * crude mortality rate * exposed population

The crude mortality rate in the U.S. is approximately 0.007, while in Indonesia the rate is 0.0095. However, since Jakarta is expected to have a lower mortality rate than the rest of Indonesia, we assume a rate of 0.007. Therefore, the range of changes in mortality (per person) is:

Upper change in mortality = $9.10 * 10^{-6}$ * change in PM10

Central change in mortality = $6.72 * 10^{-6}$ * change in PM10

Lower change in mortality = $4.47 * 10^{-6}$ * change in PM10

Respiratory Hospital Admissions. As described above, Plagiannakos and Parker[18] used pooled cross-sectional and time-series data for nine counties in their study for the period 1976 to 1982 Southern Ontario, Canada. A statistically significant relationship was found between the incidence of hospital admissions due to respiratory diseases (RHA) and ambient sulfate levels. Additional evidence for an effect of particulates on hospital admissions is provided by a study by Pope.[27] In this study a statistically significant association was found between monthly RHA, including admissions for pneumonia, asthma and bronchitis, and monthly average PM10 in two valleys in Utah studied between 1985 and 1989. Ozone concentrations were close to baseline during the winter seasons when both PM10 and RHA were elevated so the effect appears to be mostly related to particles. After analyzing, the results suggest the following functions:

Upper change in RHA per 100,000= 1.56 * change in PM10

Central change in RHA per 100,000 = 1.20 * change in PM10

Lower change in RHA per 100,000 = 0.657 * change in PM10

Emergency Room Visits. Samet et al.[28] analyzed the relationship between emergency room visits (ERV) and air pollution levels in Steubenville, Ohio, an industrial town in the midwestern United States. Daily ERV (mean 94.3) at the primary hospital in the area were matched with daily levels of total suspended particulates (TSP), sulfur dioxide levels, and nitrogen dioxide levels for March, April, October, and November of 1974 through 1977. Daily ERV were regressed on maximum temperature and each of the pollutants in separate runs. The particulates and sulfur dioxide coefficients were statistically significant in separate regression, but these measures were highly correlated.

We have selected the estimated regression coefficient for TSP as the best estimate and have used plus or minus one standard deviation from the coefficient to generate high and low estimates. The results obtained by Samet et al. indicate the following relationship:

Change in daily ERV = .011 * change in daily TSP in Steubenville

Since the approximate population in Steubenville during this period was 31,000, and PM10 is 0.55 of TSP, we annualize this equation and obtain an estimate of:

Central change in ERV per 100,000 = 23.54 * annual change in PM10

The upper and lower coefficients are 34.25 and 12.83, respectively.

Restricted Activity Days. Restricted activity days (RAD) include days spent in bed, days missed from work, and other days when activities are significantly restricted due to illness. Ostro[29] examined the relationship between adult RAD in a two week period and fine particles (FP, diameter less than 2.5 microns) in the same two week period for 49 metropolitan areas in the United States. The RAD data were from the Health Interview Survey conducted annually by the National Center for Health Statistics. The FP data were estimated from visual range data available for airports in each area. Since fine particles

have a more significant impact on visual range than do large suspended particles, a direct relationship can be estimated between visual range and FP.

Separate regression estimates were obtained for 6 years, 1976-1981.[30] A statistically significant relationship between FP and RAD was found in each year and supported earlier findings relating RAD to TSP. We selected the approximate average of the six coefficients in calculating a central estimate, and derived the upper and lower estimates from the range in the coefficients over the six years. The form of the estimated relationship was such that the coefficient for FP gives the percentage change in RAD associated with a unit change in FP. Specifically, the results indicate:

Change in RAD per adult per year = b * annual RAD * change in FP

where the high, central, and low estimate of b are 0.0076, 0.0048, and 0.0034 respectively.

To convert this function for our use, we use the following information from the original study: the annual average RAD per adult was about 19 days and sulfates were 40 percent of FP. Therefore, we analyze the results and convert from FP to sulfates to PM10, to obtain the following relationship:

Upper change in RAD per person per year = 0.0903 * change in PM10

Central change in RAD per person per year = 0.0575 * change in PM10

Lower change in RAD per person per year = 0.0404 * change in PM10

These estimates are applied to all adults. Subsequent work by Ostro[31,32] focused on currently working males and obtained generally similar results.

Lower Respiratory Illness in Children. Estimates of lower respiratory illness in children are based on an analysis by Dockery et al.[33] of children in six cities in the United States. The study related TSP, PM15, PM2.5 and sulfate levels to the presence of chronic cough, bronchitis, and a composite

index of respiratory illness (prevalence of cough, bronchitis, or respiratory illness) as measured during health examinations of samples of children in each city. A logistic regression analysis was used to estimate the relationship between the probability of an illness being present and the average of the 24-hour mean concentrations during the year preceding the health examination. Due to the likely overlap of the health endpoint measures, only the results for bronchitis is used, noting that this could include chronic cough or a more general respiratory illness. The results are applied to the population age 17 and below (17.07 percent of the total population). The basic findings that are used suggest the following:

Log odds of bronchitis = log (B/(1-B)) = 0.02368 * PM15

where B = the baseline probability of bronchitis

The change in the probability of bronchitis due to a change in PM10 can be calculated since, after taking the partial derivative of the above, the following holds:

$dB = b\ p_o\ (1-p_o)$ * change in PM15

where:

dB = change in probability of bronchitis, b = estimated regression coefficient, and p_0 = baseline probability of bronchitis.

To determine the effect of a change in PM10, we assume that it is 0.9 of PM15 and use the baseline probability of bronchitis of 6.47 percent. The central estimate uses the estimated regression coefficient reported by Dockery et al. (0.02368) and the upper and lower ranges are plus or minus one standard error from this coefficient (0.03543 and 0.01197).

Therefore, the central estimate for the effect of a unit change in PM10 equals (0.02368)(1/0.9)(0.0647)(0.9353) = 0.00169. Incorporating the above data, the following relationship for changes in the annual risk of bronchitis in children are determined as:

Upper change in bronchitis = 0.00238 * change in PM10

Central change in bronchitis = 0.00169 * change in PM10

Lower change in bronchitis = 0.0008 * change in PM10

Asthma Attacks. Several studies have related air pollution to increases in exacerbation of asthma. For example, in a study of asthmatics in Los Angeles, Whittemore and Korn[34] reported a relationship between exacerbations of asthma and daily concentrations of TSP and ozone using logistic regression analysis. Also, Ostro et al.[35] recently reported an association between several different air pollutants, including sulfates, and increases in asthma attacks among adults residing in Denver. Additional evidence for an effect of particulate matter on asthmatic children is provided by Pope et al.[36] This study examines the effects of air pollution on a clinic-based sample and from a school-based (and relatively untreated) sample. Associations were reported between particulate matter, measured as PM10, and both respiratory symptoms and the use of medication.

The Ostro et al. study took place during the winter months when asthma attacks were influenced by respiratory infections. Thus, this study is used to help derive the upper estimate. Specifically, the central estimate of this study is averaged with the upper estimate of Whittemore and Korn study to generate the upper bound. The central and lower bound estimates are derived from Whittemore and Korn's central regression estimate and minus one standard error. For our calculations, their reported asthma attack incidence of 0.26 was halved to better represent the general population of asthmatics, since many asthmatics with low attack prevalence were dropped from the analysis. The regression model includes both TSP and ozone so the total effects on asthma are apportioned to these two pollutants in accordance with their findings.

As described in greater detail in Section IV, based on available Indonesian data, we have assumed that 8.25 percent of the population of Jakarta are asthmatic versus approximately 5% in the U.S.[37]. The

estimates for increases in the annual probability of an asthma attack (per asthmatic), based on annual changes in particles, are:

Upper change in asthma attacks = 0.273 * change in PM10

Central change in asthma attacks = 0.0326 * change in PM10

Lower change in asthma attacks = 0.0163 * change in PM10

Respiratory Symptoms. Respiratory symptoms are an additional measure of acute effects of air pollution. Results of Krupnick et al.[38] can be used to determine the effects of particulate matter. This study examined the daily occurrence of upper and lower respiratory symptom among a panel of adults in Southern California. A Markov process model was developed to determine the effects of air pollution on health which incorporated the probability of illness on the prior day and controlled for autocorrelation. Among the pollutants examined independently, coefficient of haze (COH) was found to be statistically associated with the probability of reporting a symptom (b=0.0126, s.e. = .0032). Data from the study suggest a ratio of COH(units/100 ft) and TSP of 0.116. The marginal effect of COH was calculated by incorporating the stationary probabilities as described in the paper. Therefore, using the results of regressions when COH was the sole pollutant included as an explanatory variable, the following ranges were determined:

Upper change in symptom days per year per person = 0.274 * change annual PM10

Central change in symptom days per year per person = 0.183 * change annual PM10

Lower change in symptom days per year per person = 0.091 * change annual PM10

Chronic Bronchitis. Recent epidemiologic studies have related long-term exposure of air pollution to a higher prevalence of chronic respiratory disease or significant decrements in lung function. For example, Detels et al.[39] found that residents living in the Los Angeles air basin who were exposed over a long period of time to relatively high levels of particulates and oxides of sulfur and nitrogen had significantly lower lung function than a cohort less exposed. Abbey et al.[40] conducted a study on 6,600 Seventh Day Adventists, nonsmokers who had lived for at least 11 years in California. In this study, participants above age 25 (n=3914) were matched with 10 years of exposure to ambient pollutants based on their monthly residential location. New cases of chronic bronchitis between 1977 and 1987 were recorded. A logistic model was estimated that included adjustment for sex, past and passive smoking and occupational exposure. A statistically significant association was reported between long-term exposure to TSP and chronic bronchitis. Using the mean incidence rate of bronchitis during the 10-year period, the functions were linearized and converted to PM10 equivalence, and annual number of cases of bronchitis can be estimated. The functions are as follows:

Upper change in chronic bronchitis $= 9.18 \times 10^{-5}$ * change in annual PM10

Central change in chronic bronchitis $= 6.12 \times 10^{-5}$* change in annual PM10

Lower change in chronic bronchitis $= 3.06 \times 10^{-5}$ * change in annual PM10

Other (Non-quantified) Health Effects. There is limited evidence linking long term exposure to TSP to increases in cancer in women.[41] Since TSP includes several materials known or believed to be carcinogenic, reductions in particulate matter will likely reduce cancer cases, as well. There also may be other acute and chronic effects for which no empirical information is currently available. For example, the mortality effects calculated in this study only relate to acute exposures; longer term exposures to particles may increase the likelihood of premature mortality. Finally, no estimates are

provided for changes in lung function that are likely to occur after exposure to certain forms and levels of particulate matter.

Table 2 summarizes the morbidity outcomes associated with particulates in this review.

Table 2: Morbidity Effects of 10 $\mu g/m^3$ Change in PM10

Morbidity	Central	High#
RHA/100,000	12.0	15.6
ERV/100,000	235.4	342.5
RAD/person	0.575	0.903
LRI/child/per asthmatic	0.0169	0.0238
Asthma attacks/per asthmatic*	0.326	2.73
Respiratory Symptoms/person	1.83	2.74
Chronic Bronchitis/100,000	61.2	91.8

*Applies to the 8.25 percent of the Indonesian population that is assumed asthmatic.
#A high (low) estimate is obtained by increasing (decreasing) the coefficient by one estimated standard derivation.

2. **Sulfur Dioxide**

Effects of sulfur dioxide (SO_2) on the respiratory system have been observed after either short-term (less than one hour average) or longer term (24-hour average or longer) exposures. Several recent epidemiologic studies indicate that changes in 24-hour average exposure to SO_2 may affect lung function, the incidence of respiratory symptoms and diseases, and risks of mortality. These studies have been conducted in different geographic locations and climates, and with different populations and covarying pollutants. Although many of these investigations also indicate that particulate matter or ozone was associated with these adverse health outcomes, several studies appear to show an effect of SO_2 alone. Furthermore, in some of the publications reporting an effect of both SO_2 and particulates, they are highly correlated, but in others, the correlation of the daily levels is only weak to moderate. Thus, it is possible

to infer an effect of SO_2 or a sulfur species highly related to SO_2. Below, a brief review of several relevant studies and available dose-response relationships are provided.

Premature Mortality. Epidemiologic studies undertaken in several locations indicate that SO_2, acting alone or as a surrogate for other sulfur-related species, is associated with increased risk of mortality. These includes studies in France,[42] England,[43], Poland,[44] and Athens.[45] Unfortunately, most of the available studies do not provide dose-response functions. Our estimate is derived from Hatzakis et al.[45] A brief summary of the studies indicating an effect of SO_2 on mortality follows. It is also important to note that after oxidation in the atmosphere, some of the SO_2 will turn into sulfate. As reported in the earlier section of particulate matter, changes in sulfates are associated with both mortality and morbidity. Therefore, some of the benefits of reducing SO_2 relate to the reductions in particulate matter described above, and one should be aware of potential double counting of health benefits from reduced pollution.

Derriennic et al.[42] analyzed daily mortality for individuals over 65 years of age in Marseilles and Lyons, France between 1974 and 1976. Daily averages of SO_2 and suspended particulates were .03 ppm and 106 $\mu g/m^3$, respectively, but monthly SO_2 averages were above .07 ppm during certain times of the year. These two pollutants were moderately correlated ($r = .46$). Seasonal influences were prefiltered from the data, which were also corrected for autocorrelation. The authors noted that temperature, which was inversely correlated with SO_2 was correlated with respiratory-related mortality in Lyons and with cardiovascular-related mortality in Marseilles. The results, based on multiple regression and controlling for temperature, indicated a statistically significant association between SO_2 and respiratory deaths in both cities, and between SO_2 and circulatory deaths in Marseilles. The authors argue that the SO_2 effects are independent of the impact of temperature, since the regression coefficient relating SO_2 to respiratory mortality is similar for the two cities, but in only one is temperature correlated with mortality. Similarly, the association of SO_2 with circulatory deaths in Marseilles (but not Lyons) may be explained by the high

correlation in that city between SO_2 and temperature, and temperature and mortality. No association between particulates and mortality was detected in either city.

Chinn et al.[43] investigated the association between mortality of people aged 45 to 74 and air pollution in London, England and Wales. Mean SO_2 and smoke levels for 1971 are not provided explicitly, but visual inspection of the relevant graph in the text suggests means of .06 ppm and 80 $\mu g/m^3$, respectively. Two age groups for both men and women were analyzed: those aged 45 to 64 and 65 to 74. In addition to total mortality, several specific causes of death were considered, including hypertensive disease, influenza, and chronic bronchitis. There was little correlation between either SO_2 or smoke and mortality, prompting the authors to suggest this was a negative study. However, one particularly significant finding was a correlation between SO_2 and mortality from chronic bronchitis among men above 65 ($r = .22$) and women between 45 and 65 ($r = .26$).

Krzyzanowski and Wojtyniak[44] examined the association between individual-specific daily mortality and air pollution over a ten-year period for residents living in Cracow, Poland versus those living just outside the city. The results indicated a significant statistical relationship between air pollution, measured as particulate matter and SO_2, and all-cause mortality for men.

Hatzakis et al.[45] explored the relationship between daily mortality and air pollution in Athens, Greece during 1975-1982. Mean daily levels of SO_2 and British smoke were 85 $\mu g/m^3$ and 63 $\mu g/m^3$, respectively, with an average of 28.48 deaths per day. The pollutants were fairly strongly correlated ($r = .55$). Mortality was adjusted for seasonal patterns over time by calculating an observed minus predicted measure. Regression analysis was used to control for temperature, humidity, holidays, and annual, seasonal, monthly and weekly trends. SO_2 and excess all-cause mortality were correlated when all other independent variables were taken into account. Particulates, however, were not related to mortality.

The high estimate of the mortality effect is taken from the crude (i.e. no covariates included) regression results of this study. The linear coefficient relating SO_2 to daily deaths was 0.0346. Thus a

10 $\mu g/m^3$ (or 0.004 ppm) change in SO_2 is associated with a daily increase of 0.346 deaths or a 0.346/28.48 = 1.21 percent increase. For the lower range of the effect, we use the regression equations that includes adjustment for seasonality, year, interactions between year and season, day of study, several meteorologic factors, and dummy variables for months. These adjustments result in a lowering of the coefficient to 0.0058 (standard error = 0.0029, p = 0.046), suggesting a 10 $\mu g/m^3$ change in SO_2 is associated with a 0.2 percent change in mortality. The central estimate uses the model that adjusts for seasonality and year and implies that a 10 $\mu g/m^3$ change is associated with a 0.48 percent change in mortality. The ranges are as follows:

Upper percent change in mortality = 0.121 * change in SO_2

Central percent change in mortality = 0.048 * change in SO_2

Lower percent change in mortality = 0.020 * change in SO_2

Respiratory Symptoms. Recent studies that provide evidence of an effect of SO_2 on symptoms include Charpin et al.,[46] Bates and Sizto,[47-48], Ponka,[49] Dodge et al.,[50] and Schwartz et al.[51,52] Dose-response information can be generated from the latter two studies.

Schwartz et al.[51] relate daily levels of SO_2 to respiratory symptoms among a sample of approximately 280 children in Watertown, Massachusetts who were part of the Harvard Six-Cities Study. A daily diary completed by parents recorded several acute symptoms of their children including upper respiratory illness and cough. The correlation among pollutants was not reported. A logistic regression was used to examine the relationship of pollution to these symptoms. Sulfur dioxide had a statistically significant association with cough. The impacts of other pollutants were unclear from this primarily methodological article. Nevertheless, the results suggest the following:

logit (cough) = 0.0130 * SO_2 (ppm)

The standard error of the estimated regression coefficient was 0.0059 and the mean incidence rate was one percent. Taking the derivative, converting into $\mu g/m^3$ (1 ppm = 2,860 $\mu g/m^3$), annualizing and substituting, we obtain the following functions for children:

Upper change in the probability of cough per 1,000 kids per year = 0.0262 * change in SO_2

Central change in the probability of cough per 1,000 kids per year = 0.0181 * change in SO_2

Lower change in the probability of cough per 1,000 kids per year = 0.010 * change in SO_2

Schwartz et al.[52] examined the effects of air pollution among a population beginning nursing school in Los Angeles in the early 1970s. Daily diaries were completed and provided information on incidence of symptoms including cough, phlegm, and chest discomfort. Pollutants under investigation included oxidants, sulfur dioxide, nitrogen dioxide and carbon monoxide. In models corrected for autocorrelation, a significant association was found between SO_2 and chest discomfort. Daily concentrations of SO_2 averaged approximately 0.09 ppm. Specifically, the results indicated:

logit (chest discomfort) = 1.88 * SO_2 (ppm)

The standard error of the estimated regression coefficient was 0.094. Taking the derivative, annualizing, converting into $\mu g/m^3$ and substituting the mean rate of chest discomfort of 0.04, the following functions are obtained:

Upper change in probability of chest discomfort per year = 0.015 * change in SO_2

Central change in the probability of chest discomfort per year = 0.010 * change in SO_2

Lower change in the probability of chest discomfort per year = 0.005 * change in SO_2

Table 3 summarizes the health effects that have been quantified for SO_2.

Table 3: Effects of 10 $\mu g/m^3$ Change in SO_2 Concentrations

Sulphur Dioxide	Central Estimate	High# Estimate
Mortality (percent change)	0.48	1.21
Respiratory Symptoms/ 1,000 child/year	0.18	.26
Chest Discomfort/adult/year	0.10	.15

#A high (low) estimate is obtained by increasing (decreasing) the coefficient by one estimated standard derivation.

3. **Ozone**

Ozone is the primary component of photochemical smog. As such, it has been associated with several adverse respiratory outcomes including increased upper and lower respiratory symptoms, eye irritation (oxidants), restrictions in activity, and exacerbation of asthma. Most of the evidence of the effects of ozone is derived from clinical studies in which subjects are exposed to a known amount of ozone in a controlled setting. For example, healthy individuals may be exposed to moderate levels of ozone in a chamber while engaging in moderate exercise. Unfortunately, these studies usually focus on changes in lung function and less so on increases in symptoms. Also, it is difficult to develop dose-response functions from some of these studies or extrapolate from their findings to the free-living population. However, several epidemiologic studies are available and provide a basis for dose-response estimates.

Respiratory Hospital Admissions. Current evidence indicates that ozone may be associated with hospital admissions related to respiratory disease (RHA).[53,54] This possibility is supported by findings from panel studies of asthmatics indicating that exacerbations occur in response to ozone.[34,55] Clearly, some of these exacerbations may result in either emergency room visits or hospital admissions. Unfortunately, because of the high covariation between ozone and other pollutants in the summer when most of the studies have been undertaken, it is difficult to determine the effect on RHA attributable to ozone alone. However, by using information from several studies, it is possible to begin to apportion the effects of the different pollutants. Thurston et al.[53] found a significant association between RHA and both ozone and sulfates in New York City (the Bronx) and Buffalo in the summer 1988. In this analysis, corrections for autocorrelation and day-of-week effects were made. At the mean, the effect of ozone was approximately twice the effect observed for sulfates. Burnett et al.[54] also reported a statistically significant association between hospital admissions and both ozone and sulfates in Ontario, Canada for the years 1983 through 1988. Their findings suggest that the ozone effect was approximately 3 times that of sulfates, based on a regression equation that included both pollutants. Therefore, it is reasonable to apportion the effects of RHA based on the relative coefficients in Thurston et al. The average of the coefficients for the two cities in that paper is 21.3 RHA per day/ million/ppm ozone, which becomes the central estimate, with a standard error of 10.9. Thus, after annualizing, the functions for RHA become:

Upper change in RHA per person = 0.012 * change in daily 1-hour max ozone (ppm)

Central change in RHA per person = 0.0077 * change in daily 1-hour max ozone (ppm)

Lower change in RHA per person = 0.0038 * change in daily 1-hour max ozone (ppm)

Restrictions in Activity. Portney and Mullahy[56] used the 1979 Health Interview Survey conducted by the National Center for Health Statistics, to examine the relationship between ozone and

the occurrence of minor restrictions in activity (MRAD). These involve restrictions in activity that do not result in either work loss or bed disability. Individual-level health data for 14,000 adults living throughout the United States were combined with data on air pollution and meteorology. The health outcome was based on a two-week recall period. The general regression model included socioeconomic and demographic factors, chronic health status, and city-wide variables. A statistically significant association was found between the number of MRAD in a two-week period and ozone concentrations during a similar period.

For the most general results, using a poisson model, the coefficient on ozone represents the percent change in MRAD per person per two weeks for a one part per million (ppm) change in ozone. The estimated beta coefficient was 6.883, with a standard error of 3.4 ($p < .05$). Therefore, the central estimate becomes:

$$\% \text{ change in MRAD per two week period} = 6.883 * \text{change in two week average of 1-hour daily maximum ozone}$$

Since the mean MRAD per two week period is roughly 0.19, the above equation becomes:

$$\% \text{ change in MRAD per year} = (6.883)(.19)(26) * \text{change in annual average of 1-hour maximum ozone (ppm)}$$

This implies that reducing the average daily maximum ozone concentration by 0.01 ppm for one year would reduce the number of MRAD per person by 0.34. The high and low ranges are developed by using plus and minus one standard error of the estimate. The annual increase in the number of cases per person for a change in the annual average of the 1-hour daily maximum of ozone (ppm) is:

Upper MRAD per person per year = 51.0 * change in 1-hr max ozone (ppm)

Central MRAD per person per year = 34.0 * change in 1-hr max ozone (ppm)

Lower MRAD per person per year = 17.0 * change in 1-hr max ozone (ppm)

Respiratory Symptoms. Results of Krupnick et al.[38] can be used to estimate the effects of ozone on respiratory symptoms. As noted above, this study examined the daily occurrence of upper and lower respiratory symptoms among a panel of adults in Southern California. A Markov process model, which incorporated the probability of illness on the prior day and controlled for autocorrelation, was developed to determine the effects of air pollution on health.

Many regression models included both ozone and particulate matter. Therefore, to prevent double counting, the effects of these pollutants were apportioned according to the regression results when the pollutants were examined together. The marginal effect of ozone was calculated by incorporating the stationary probabilities as described in the paper. The high estimate is obtained from the specification that includes all other pollutants, the central estimate includes only some of the pollutants and the low estimate is one standard error below the central estimate. Therefore, the following ranges were determined for the number of symptom days related to a change in the annual average of 1-hour daily maximum ozone:

Upper change in symptom days per year per person = 96.6 * change in 1-hour max ozone (ppm)

Central change in symptom days per year per person = 54.75 * change in 1-hour max ozone (ppm)

Lower change in symptom days per year per person = 28.11 * change in 1-hour max ozone (ppm)

Eye Irritation. Schwartz et al.[57] provide empirical estimates relating oxidants to eye irritation using the data from a population beginning nursing school in Los Angeles, as described above. Using

logistic models corrected for autocorrelation, a significant association was found between oxidants and daily incidence of eye irritation. Specifically, the results indicated:

logit (eye irritation) = 0.0202 * ozone (pphm)

The standard error of the estimated regression coefficient was 0.0018. Taking the derivative, analyzing, and substituting the mean incidence rate of eye discomfort of 3.75 percent, the following functions are obtained for adults, in terms of cases per year per annual change in average 1-hour maximum ozone (ppm):

Upper change in eye irritations = 29.9 * change in 1-hour max ozone (ppm)

Central change eye irritations = 26.6 * change in 1-hour max ozone (ppm)

Lower change in eye irritations = 23.4 * change in 1-hour max ozone (ppm)

Asthma Exacerbation. Whittemore and Korn[34] studied the acute effect of oxidants (including ozone) on the increased probability of a daily asthma attack. The data were taken from 16 panels of adult asthmatics located in six Los Angeles communities. The median one-hour maximum oxidant level ranged from 0.03 to 0.15 ppm across the communities studied, with single day peaks of 0.40 ppm. The study used a statistically powerful approach to estimate both individual-level and group effects. Oxidants were found to be statistically related to exacerbation of asthma, after controlling for asthma status on the previous day, temperature, humidity, and day of study. Using a logistic model, they obtained a regression coefficient of 1.66 and a standard error of 0.72. We use a baseline daily probability of asthma attack of 0.13 as discussed above.

In a similar study using daily data on asthmatics in Houston, Stock et al.[55] report an association between ozone and the likelihood of an asthma attack. The regression specification included several pollutants, pollen, humidity, temperature and whether an effect occurred on the previous day. The results from the regression model that included particulate measurements was used to generate the upper bound.

The results were not used for the central estimate since the sample size was so small (n=41). Therefore, the functions for the effect of ozone in terms of ppm 1-hour maximum are:

Upper change in asthma attacks per year = 189.8 * change in ozone (ppm)

Central change in asthma attacks per year = 68.44 * change in ozone (ppm)

Lower change in asthma attacks per year = 38.69 * change in ozone (ppm)

Table 4 summarizes the outcomes that have been quantified for ozone.

Table 4: Effects of 1 ppm Change in Ozone

	Central Estimate*	High Estimate
Hospital Admissions/persons	0.0077	0.012
Minor Restrictions in Activity/person	34.0	51.0
Respiratory Symptoms/person	54.75	96.6
Eye Irritation/adult	26.6	29.9
Asthma Exacerbation/asthmatic	68.44	189.8

*The coefficients apply to the annual average of the daily 1-hour maximum ozone.

4. Lead

Exposure to ambient lead occurs primarily from leaded fuel in automobiles and from stationary sources including primary and secondary smelters and battery recycling plants. Once absorbed, lead is distributed throughout the body and is only slowly removed. Lead has been reported to cause many different health effects. Based on current knowledge, clinical effects that may occur at the lowest blood lead concentrations include neurodevelopmental effects in children, and hypertension and related

cardiovascular conditions in adults. These two effects provide the basis for our estimates of the impact of lead. A thorough review of health outcomes associated with lead exposure is provided by U.S. EPA[58, 59] and ATSD.[60]

Effects of Lead on Blood Pressure in Adults. The association between lead and increased blood pressure was first observed in animals. This effect has been shown across a range of doses and in several species,[57] and has been examined in occupational and population-based epidemiologic studies. The population-based studies will be briefly reviewed here.

Several investigators[62-67] have used NHANES II data, published by the National Center for Health Statistics, to investigate the relationship between blood lead level and blood pressure. NHANES II is a large, individual-level database that includes information on a variety of potentially confounding factors. Therefore, these studies avoided common study design problems (e.g., healthy worker effect, workplace exposures to other toxic agents, selection bias, and problems of control group selection). Using these data, Harlan et al.[62] demonstrated statistically significant linear associations ($p < 0.001$) between blood lead concentrations and both systolic and diastolic blood pressure among males aged 12 to 74 years. Further analyses reported by Pirkle et al.[63] focused on white males, aged 40 to 59 years. This age group was used to reduce any potential confounding effects of age on blood pressure. In the subgroup studied, significant associations were found between blood lead and blood pressure even after controlling for most risk factors known to be correlated with blood pressure. Furthermore, no threshold for the effect was observed across a blood lead range of 7 to 34 micrograms per deciliter of blood (μg/dl). Schwartz reanalyzed the data of Pirkle et al. and showed that the association decreased but remained significant for both systolic and diastolic blood pressure when adjusted for site.[64-67] In addition, an association between blood lead and the likelihood of hypertension (diastolic blood pressure greater than 90 mm Hg) was reported.

Additional support for the effect of blood lead on diastolic blood pressure is provided by another major population-based study conducted in Britain by Pocock et al.,[68,69] data from the Canada Health Survey,[70] and a study of San Francisco bus drivers by Sharp.[71]

Taken together, these studies indicate a significant effect of lead on blood pressure and hypertension. In addition, investigators have estimated the subsequent impact of a increase in blood pressure (due to increases in blood lead) on other significant cardiovascular events. For example, large-scale epidemiologic studies including the Pooling Project Research Group[72] and the Framingham study[73] have shown that elevated blood pressure increases the risk of cardiovascular disease. For example, the Pooling Project reported that smoking, serum cholesterol, and diastolic blood pressure were major risk factors in the incidence of coronary heart disease (CHD). The Framingham study[73] was one of the studies included in the Pooling Project. Besides estimating the incidence of CHD, this study of white middle-aged men considered the incidence of deaths from all causes. Diastolic blood pressure was again identified as a significant predictor of all-cause mortality. These studies were used by Pirkle et al.[63] to estimate the quantitative effects of blood lead on diastolic blood pressure and subsequent CHD and mortality.

The methodology used here to estimate the effects of lead on blood pressure is similar to that used by the U.S. EPA in its analyses of the effects of reducing lead in gasoline,[74] and the effects of reducing lead in drinking water.[75] Dose-response functions are provided to estimate the effect of a change in air lead on the likelihood of hypertension, and the effects of more serious health outcomes including myocardial infarction (heart attacks) and mortality.

Hypertension. To estimate the change in hypertension related to air lead we use dose-response information provided by Schwartz et al.[67] Additional documentation for this estimation process is found in Pirkle et al.[63] and Brennan et al.[76] The estimates are based on a logistic regression of the probability of hypertension versus blood lead. The original estimates were conducted for the subset of the population of adult males age 40 to 59. However, sensitivity analysis conducted by Schwartz et al.[64] and analysis

by Landis and Flegal[77] indicate that it is not unreasonable to apply the blood lead-blood pressure relationship to all males, age 20 to 70.

The probability of hypertension as a function of log of blood lead is expressed by:

$$\text{Change in H} = (1 + \exp(2.744 - 0.793 (\ln PbB_1)))^{-1} - (1 + \exp(2.744 - 0.793 (\ln PbB_2)))^{-1}$$

where:

The standard error of the estimate of 0.793 is 0.25.

H = the probability of hypertension

PbB_1 = initial blood lead level (μg/dl)

PbB_2 = new blood lead level (μg/dl)

Since the risk of all-cause mortality and of heart attacks was provided as a function of diastolic blood pressure, the association between blood lead and diastolic blood pressure is needed. Based on Pirkle et al.[63] and U.S. EPA,[78] we use the following relationship:

$$\text{change in DBP} = 2.74 (\ln PbB_1 - \ln PbB_2)$$

where DBP = diastolic blood pressure

Using the sample means of the independent variables in the Pooling Project, we can express the relationship between the change in blood pressure and the change in the probability of a CHD event (non-fatal myocardial infaction) in the following ten years as:

$$\text{Change in Pr(CHD)} = (1 + \exp(5.0 - 0.030 (DBP_1)))^{-1} - (1 + \exp(5.0 - 0.030 (DBP_2)))^{-1}$$

where PR(CHD) = the 10-year probability of a coronary heart disease event; DBP_i initial and new diastolic blood pressure levels.

Premature Mortality. The Framingham study[75] can be used to estimate the change in mortality due to the change in diastolic blood pressure. Controlling for serum cholesterol levels and smoking, the association can be estimated by:

$$\text{change in Pr(MORT)} = (1 + \exp(5.3 - 0.035(DBP_1)))^{-1} - (1 + \exp(5.3 - 0.035(DBP_2)))^{-1}$$

where PR(MORT) = the 12 year probability of death from all-causes and the standard error of the coefficient of 0.035 is .007.

For this analysis, it may be necessary to establish a relationship between changes in lead emitted into the air and subsequent concentrations of lead in the blood of adults. To determine this relationship, we relied on many of the studies that relate air concentrations to the subsequent change in blood lead through many different media and exposure routes. Results are obtained from both observational studies in the field and studies in experimental exposure chambers. The latter are particularly useful in providing estimates of the blood lead/air lead slope since they may involve longitudinal examination of individuals exposed to relatively small changes in air lead under controlled conditions. A review of many of these studies is supplied by EPA.[75] Considering both the experimental and observational information, we assume a blood lead/air lead slope of 2.0 for adults.

Since quantitative relationships have been established between air lead and blood lead, and between blood lead and health effects, we can use the above information to directly estimate health effects related to changes in ambient lead.

For adults, the effects of ambient lead on blood pressure can be expressed as:

$$\text{change in H} = (1 + \exp(2.744 - 0.793\ (\ln 2PbA_1)))^{-1} - (1 + \exp(2.744 - 0.793\ (\ln 2PbA_2)))^{-1}$$

where:

H = the probability of hypertension

PbA_1 = initial air lead level (μg/dl)

PbA_2 = new air lead level (μg/dl)

The initial air lead level is the current average concentration for the population, while the new air lead level is the presumed ambient standard. The calculated change in risk of hypertension is then multiplied by the adult male population to obtain the total number of cases. We can also estimate the effect of a change in air lead on diastolic blood pressure and subsequent circulatory disease. Based on Schwartz[67] the change in diastolic blood pressure can be expressed as:

$$\text{change in DBP} = 2.74 (\ln PbA_1 - \ln PbA_2)$$

As a baseline level of DBP, the U.S. average of 76 mm Hg is appropriate. The change in diastolic blood pressure (due to a change in air lead) can be calculated and substituted into the following equations to estimate the subsequent changes in mortality and a coronary heart disease.

$$\text{change in Pr(CHD)} = (1 + \exp (5.0 - 0.030(DBP_1)))^{-1} - (1 + \exp (5.0 - 0.030 (DBP_2)))^{-1}$$

$$\text{change in Pr(MORT)} = (1 + \exp (5.32 - 0.035(DBP_1)))^{-1} - (1 + \exp (5.32 - 0.035(DBP_2)))^{-1}$$

where PR(CHD) = the 10-year probability of a coronary heart disease event; DP_i = initial and new diastolic blood pressure levels; and PR(MORT) = the 12 year probability of death from all causes.

Effects of Blood Lead on Neurodevelopment in Children. Research indicates that lead's neurodevelopmental effects, at low to moderate exposure levels appear to include: (1) decreased intelligence, (2) short-term memory loss, (3) reading and spelling underachievement, (4) impairment of visual motor functioning, (5) poor perception integration, (6) disruptive classroom behavior, and (7)

impaired reaction time.[80] Prospective studies in Boston,[81] Cincinnati,[82] Cleveland,[83] and Port Pirie (Australia)[84] have examined the relationship between alternative measures of intelligence and concentrations of lead in blood in children. Taken together, these studies indicate an association between general measures of intelligence and either pre- or post-natal blood lead concentrations. No threshold level has been identified.

Needleman and Gatsonis[85] conducted a meta-analysis using several recent studies that relate lead exposure with neurodevelopmental effects in children. From their review of 24 studies, six can be used to provide regression coefficients that relate a 1 μg/dl change in blood lead to subsequent IQ decrements.[86-91] Reanalysis of the studies was conducted by the Centers for Disease Control (CDC)[92] which computed the estimated change in IQ for every unit change in blood lead. Each regression coefficient was weighted by the inverse of the variance around each coefficient. Thus, they obtained a result that indicated each 1 μg/dl change in blood lead results in a 0.25 point change in IQ. Winneke et al.[93] combined results from eight countries using a common study protocol to examine the effects of blood lead on IQ. The strongest effects were observed between blood lead and visual-motor integration. Intelligence scores were also related to lead levels, but the effects were less consistent across the groups. However, the aggregate impact using all 1700 children suggested that mean IQ dropped by 0.12 for every μg/dl increase in blood lead. This estimate supports the CDC estimate for the U.S., which is incorporated into our estimates. Therefore. the association between blood lead and IQ is assumed to be:

IQ decrement = 0.25 IQ points * change in blood lead (μg/dl)

Again, it is useful to relate the health effect to ambient concentrations; this requires the establishment of a relation between air lead and blood lead. These studies typically compare ambient concentrations and blood lead levels for individuals residing near some lead source with a "control group". The studies attempt to control for age, sex, and other potential confounders and effect modifiers. Although these studies linking blood lead in children to air lead suffer from problems with measurement

and methodology, taken together they describe a fairly consistent range for the blood lead/air lead aggregate slope of between 2.5 and 5.3.[78] If an association is needed to apply this methodology, a slope of 3.9 is recommended.

Therefore, for children, the relationship between air lead and loss in IQ becomes:

IQ decrement = 0.25 IQ points/(μg/dl of blood lead) * 3.9 μg/dl in blood lead per μg/m^3 change in air lead,

IQ decrement = 0.975 IQ points * change in air lead (μg/m^3)

Table 5: **Effects of 1 μg/m^3 Change in Ambient Lead***

	Low Estimate	Central Estimate[#]	High Estimate
IQ loss, points per child **	-	.975	-
Cases of Hypertension (one million males aged 20-70)	44,800	72,600	97,800
Non-fatal Heart Attacks (one million males aged 40-59)	180	340	500
Deaths (one million males aged 40-59)	200	350	650

[#]Assumes change from 0.5 to 1.5 μg/m^3 and a diastolic blood pressure of 76mm Hg.

*For lead, the effect of exposure time is not well known.

**It is uncertain whether effect occurs at certain stage (say age 3) or through childhood. A reasonable interpretation is that a child on average will avoid an IQ loss of .975 if it grows up with ambient lead concentrations of .5 rather than 1.5 μg/m^3.

5. Nitrogen Dioxide

The epidemiologic evidence for an effect of nitrogen dioxide (NO_2) on respiratory symptoms is more uncertain than the effects of the other criteria pollutants described above. Many studies have

demonstrated an effect on children from indoor exposure to gas stoves, the primary source of indoor NO_2. However, effects from outdoor NO_2 on either children or adults have rarely been found. This may be due to (1) large errors related to measuring outdoor NO_2 (since animal experiments indicate that very short peaks may be responsible for the adverse health outcomes); (2) the occurrence of effects only at the high levels of NO_2 associated with gas stoves; or (3) the possibility that chronic, not acute, effects of NO_2 are important. However, at least one recent epidemiologic study has found health effects related to ambient NO_2.

In a reanalysis of the Los Angeles student nurses data described earlier, Schwartz and Zeger[57] found an association between NO_2 and the increased likelihood of phlegm production. Daily concentrations of NO_2 averaged approximately 0.13 ppm. Specifically, the results indicated:

logit (respiratory symptoms) = 0.843 * NO_2 (ppm)

The standard error of the estimated regression coefficient was 0.343. Taking the derivatives, substituting the mean incidence rate of phlegm of 0.0345 and analyzing, the following functions are obtained for adults, based on a average annual change in the 1-hour daily maximum:

Upper change in respiratory symptoms per year = 14.42 * change in NO_2 (ppm)

Central change in respiratory symptoms per year = 10.22 * change in NO_2 (ppm)

Lower change in respiratory symptoms per year = 6.02 * change in NO_2 (ppm)

6. Carbon Monoxide

Carbon monoxide (CO) affects health by interfering with the transport of oxygen to the heart and other muscles and to brain tissues. When CO enters the respiratory system, it forms carboxyhemoglobin (COHb) and reduces the oxygen carrying capacity of the blood. Individuals with ischemic heart disease or coronary artery disease, which is the leading cause of disability and death in

industrialized nations, are particularly at risk from exposure to CO, since they already have a restricted flow of oxygen in the blood. Increases in oxygen demand from increased activity or reductions in oxygen delivery can result in decreased time to the onset of angina pectoris in this group. At higher concentrations of CO, people with anemia and other blood disorders, chronic lung disease, pregnant women, fetuses, and newborns may be at risk. At much higher levels, healthy individuals are also at risk of much less severe effects, such as headache and fatigue.

Unfortunately, there is little quantitative dose-response information linking CO exposure to a meaningful health endpoint. Part of the difficulty in estimating dose-response is due to the nature of CO itself. CO dissipates rapidly in the environment and while it may exist at high concentrations near a source, such as a highway, much lower concentrations may exist only a short distance away . Therefore, the use of fixed-site monitors to indicate population exposure is often inappropriate. Because of these shortcomings, no quantitative estimates of the effects of CO are provided in this review.

7. Carbon Dioxide

In recent years, there has been increasing concern about man's potential to alter the earth's climate through the emissions of gases that may result in a "greenhouse effect". Specifically there is evidence that excess emissions of gases, including carbon dioxide (CO_2), methane, nitrous oxide, tropospheric ozone, and chlorofluorocarbons may be associated with a rise in mean global temperature. However, at this time, major uncertainties about this phenomenon still exist and preclude the development of quantitative estimates associated with health effects. First, the precise association between the production of CO_2 and global warming is unknown. Second, the relationship between global warming and subsequent health outcomes are unclear. Global warming will likely result in a rise in sea levels and changes in climate. However, there is little information about the impact these changes will have on health outcomes. Therefore, no quantitative effects related to CO_2 are presented in this report.

IV. APPLICATION TO JAKARTA, INDONESIA

A. Background Information

Jakarta, the capital of Indonesia, is located in the tropics, just south of the equator. The city is located on a level plain on the northwest coast of the island of Java and occupies around 650 km2, at 7 to 10 meters above sea level. The population is believed to be around 8.2 to 9 million, but some estimates have put it as high as 12 million. The city is very densely populated with densities ranging from more than 30,000 per km2 in central Jakarta to less than 10,000 per km in most other parts of the city. Average annual temperature is 27.5°C, with a humidity of around 80 percent, and low wind speed. The low wind, high population density, and high rates of car ownership (currently 1.7 million vehicles in Jakarta) indicate the potential for significant air pollution concentrations.

Data from the Ministry of Population and Environment provide population information for subdistricts of Jakarta. A report from the Bandung Institute of Technology[26] further disaggregates the population data into 5 x 5 km grids. This report also is the source of information on current ambient pollution levels. For this analysis, the total population for Jakarta is assumed to 8.2 million. Indonesian census data indicates that 35.7 percent of the population is below age 18.

Since existing monitors provide incomplete coverage of the region, particulate matter concentrations for metropolitan Jakarta were based on dispersion models developed by Doedomo et al.[26] Emissions data are provided for the four major activities in Jakarta: fuel use from transportation, industrial sources, fuel use for household cooking, and the burning of solid waste. Transportation emissions are based on density rate of traffic for each subdistrict of Jakarta. Fuel-specific estimates are provided for automobiles, motorcycles, buses and trucks. Industrial emissions are based on land use information from the city government that details the location and kind of plant operation. Household fuel use and solid waste are assumed proportional to population density in each grid. Open burning is the

primary means of solid waste disposal in Jakarta. After total emissions were calculated for each grid cell, a Eulerian multi-box dispersion model, incorporating local meteorologic and topographic features, was developed and used.

For this report, only benefits related to reductions in TSP, lead and nitrogen dioxide are estimated. Estimates are not provided for pollutants such as carbon monoxide, ozone, and sulfur dioxide because of a lack of monitoring (ozone), a lack of dose-response information (carbon monoxide), and concentrations that are below the air quality standards (sulfur dioxide). Of the emissions of TSP, an estimated 30 percent come from transportation sources (including gasoline and diesel-powered vehicles), 35 percent from fuel combustion (including residential cooking), 8 percent from solid waste disposal (including municipal incinerators and open burning), 15 percent from industrial process, and 12 percent from other sources including construction and fugitive dust. Figure 1 displays the resultant isopleths for annual averages of TSP, in 1989. Ambient concentrations were determined for each grid and combined with the population data for that grid to calculate the population-micrograms of exposure. As such, this method is a significant improvement over previous studies that have calculated city-wide averages for the entire metropolitan area based on readings from one or more fixed site monitors. The results, supported by recent monitoring efforts, suggest that ambient concentrations of annual average TSP are 3 to 4 times the levels typically observed in the United States.

For lead concentrations in Jakarta, vehicles using leaded gasoline are the primary source, although smelters and battery recyclers also contribute. For nitrogen dioxide, 73 percent of the emissions are from the transportation sector, with industry contributing around 16 percent. Figures 2 and 3 display the isopleths for lead and nitrogen dioxide for 1989. Again, the figures indicate that the levels are substantially higher than levels observed in the United States, and higher than most existing ambient standards by a factor of 3 or 4.

Baseline health status in Indonesia is much poorer than that observed in the U.S. For example, the infant mortality rate in Indonesia is 78 per 1000 live births versus 10.1 in the U.S. The difference is due to a multitude of factors associated with poverty and lower income such as high population density and lower levels of nutrition, medical care and access, and to factors related to behavior and infrastructure such as occupational exposures, high exposure to passive smoke and kerosene cookers and insufficient ventilation in the homes. For example, Achmadi[96] examined a cohort of children under five and found that the episodes of acute respiratory tract infection are related to the degree of indoor air quality, population density, and socioeconomic status . Research in the U.S. and Britain has indicated that frequent episodes of lower respiratory disease may be associated with chronic obstructive pulmonary disease in adulthood. Particular problems may be expected in rural areas where an estimated 88% of the households cook with wood or charcoal, frequently indoors, in stoves without flues and in poorly ventilated kitchens, most of the year. In urban areas like Jakarta, unvented kerosene cookers may be a significant source of respiratory disease. The 1990 Census reports that in Jakarta, 84 percent of all households cook with kerosene, followed by gas (11.6 percent), wood or charcoal (2.3 percent), and electric (2.1 percent). In some cases, therefore, extrapolating dose-response estimates from western developed countries may underestimate the more serious health outcomes associated with both acute and chronic exposures.

Because of all of these factors, Indonesians experience high rates of morbidity from acute respiratory infections, and a high prevalence of chronic respiratory disease. Although respiratory conditions in Indonesia have not been conclusively linked to indoor and outdoor air pollution, there is ample descriptive evidence of the magnitude of the problem. The 1990 Indonesian Census indicates that among children under age five, acute respiratory infections account for 14.4 percent of all mortality. It is the second leading cause of death after diarrheal disease, which is responsible for 15.6 percent of all

mortality for this age group. It is likely that the rates of respiratory disease are higher among infants in the more densely populated cities.

For Indonesia as a whole (all ages), inflammation of the respiratory tract was the sixth leading cause of death (after accidents, diarrhea, cardiovascular disease, tuberculoses, and measles), accounting for 6.2 percent of all mortality.[95] In the U.S., diseases related to respiratory tract information account for about 4.4 percent of all mortality. However, in Jakarta, this outcome accounts for 12.6 percent of all mortality,[96] more than double the rate for all of Indonesia. It is reasonable to hypothesize that exposure to high indoor and outdoor air pollution, along with high population density and limited health care access, may be responsible for these higher rates in the city. For the entire population, upper respiratory tract conditions are the leading cause of morbidity, responsible for 44.8 percent of all reported cases. In Indonesia in 1986, acute respiratory tract infections had the highest reported prevalence rate at 21.3 per 1000. Following this outcome in prevalence were skin infections (7.6 per 1000), gastrointestinal tract conditions (6.9), other infections (6.5) and bronchitis and asthma (6.4) (Ministry of Health, 1989). Data from the 1990 Indonesian census indicates that among children under age five living in Jakarta, 10.7 percent experienced cough or shortness of breath during the two weeks prior to the census survey period. Of these, 65 percent were taken to a health care facility. Finally, as the indicator of respiratory disease status, a study of asthma in children reported a prevalence rate of 16.5 percent[96] versus approximately 10 percent among children in the United States, 10 percent in Bogor, 6 percent in Jogjakarta, and 3.7 percent in Bali. The health data are revealing particularly when relative rates of respiratory disease in Jakarta are compared not only with these United States, but even with other cities in Indonesia, or with the country as a hole, which may be a more appropriate baseline for comparison. Using this information, we assumed a baseline rate of asthma for the population of Jakarta of 8.25 percent or (16.5/10) times the U.S. asthma prevalence rate of 5 percent.

B. Estimated Health Effects

The estimates of the reduction in health effects achieved from reaching alternative standards of particulate matter are provided in Tables 6 through 8. Significant reductions in health effects could be expected. For example, if annual TSP were reduced to the midpoint of the WHO guideline (also the former U.S. standard) of 75 $\mu g/m^3$ (Table 7), this central estimates suggest an expected reduction per year of 1,400 deaths (with a range of 900 to 1,900), 2,500 hospital admissions for respiratory disease, 49,000 emergency room visits, 600,000 asthma attacks, 7.6 million restricted activity days including work loss, 125,000 cases of bronchitis in children, 37 million minor respiratory symptoms, and 12,000 cases of chronic bronchitis. Meeting the Indonesian standard (Table 6) might save an estimated 1,200 lives per year (range 750 to 1,600) and reduce restrictions in activity by 6.3 million days. Attaining the lower California standard would save almost 1,800 lives per year (range 1,100 to 2,400) and 47 million days of restricted activity. Clearly, particulate matter air pollution represents a significant public health hazard and economic burden to residents of Jakarta and to residents of other cities consistently exposed to such high levels of particulate matter such as Mexico City, Bangkok, and Santiago, Chile.

Table 9 displays the health effects associated with current levels of lead in Jakarta. For example, consider the effects of reducing lead from current levels to the low end of the range of the WHO guidelines (annual average of 0.5 $\mu g/m^3$). For this case, the model estimates a annual decrease of 136,800 cases of hypertension, 190 cases of coronary heart disease, 158 cases of mortality related to cardiovascular disease in adult men, and a total loss of 2,070,000 IQ points in children.

Table 10 relates similar calculations for reducing nitrogen dioxide concentrations to the Indonesian standard of 0.05 ppm (100 $\mu g/m^3$).

V. IMPROVING THE ESTIMATES OF AIR POLLUTION DAMAGES: UNCERTAINTIES AND FUTURE NEEDS

As indicated in the introduction, the current estimates of the benefits of reducing ambient levels of particulate matter are based on broad averages and fairly simple assumptions. Nevertheless, they do provide information about the health effects that can be quantified. These effects can, in turn, be valued in order to provide a range for the economic value of controlling these pollutants. These estimates can then form the basis for prioritization when choosing among several air pollution control strategies as well as for assessing the value of additional control efforts. Additional uncertainties arise when applying these numbers to other countries, particularly those that are less developed. This section provides a brief review of the major areas of uncertainty in the estimates, and describes tasks that could be undertaken to reduce this uncertainty. The greatest uncertainties are generated by the need to extrapolate the epidemiologic health effects from the U.S. and Western industrialized nations to other countries.

Most of the dose-response functions provided in the literature are based on research conducted primarily in the U.S. Extrapolating these results to countries in very different stages of development adds additional uncertainty to any estimate of benefits associated with reduced air pollution. These uncertainties arise since we must assume that the population characteristics that relate to the sensitivity to air pollution in the U.S. will be similarly distributed in other countries. This may include factors such as: age and sex distribution, smoking rates, general health status, exercise and diet, medical access and use, competing risks, averting behavior, and activity patterns.

It may be instructive to consider a few cases that indicate the importance of these differences when extrapolating from the U.S. studies. For example, consider the effects of activity patterns on the effective dose. Ideally, the dose would reflect the ambient air quality and the amount of time spent outdoors. Studies of populations in the U.S. suggest that people spend about 90 percent of their time indoors. Many of these indoor environments are well sealed and dramatically reduce the penetration of

outdoor pollutants into the indoors. (The tight homes also may keep indoor pollutants indoors, however). Therefore, the estimated dose-response relationships between ambient air pollution and health, by necessity, incorporate the large proportion of time spent indoors by most people. This aspect will lead to a significant underassessment of dose when applying the existing dose-response functions to warm climates such as those in South and Southeast Asia, Latin America, and parts of South America. An underestimate is likely because residents in these climates will likely spend a greater portion of their time outdoors, both on a daily and an annual basis. In addition, indoor exposures in the homes in many of these countries appears to be quite high. If this is true, it is likely that a given level of air pollution, everything else constant, would generate much greater health effects in poorer, warmer countries.

Other disparities between countries may result in different quantitative impacts from exposure to ambient air. For example, some developing countries may have a greater proportion of individuals with chronic respiratory disease, and will not have the public warnings on high air pollution days that occur in the U.S. This could significantly increase the health impacts of a given level of air pollution. Similarly, deficiencies in vitamin C and E, likely to be more common in the developing world, reduce the host defense mechanism and may increase the effects of air pollution.

Only after epidemiologic studies are completed in some of the developing countries, can the uncertainty about the impact of these factors be reduced. In the mean time, the suggested methodology is to assume either that the demographic and health status profiles in the developing world are similar to those where the original studies were undertaken, or make some arbitrary adjustments to the dose-response relationships. Of particular importance would be studies relating mortality, hospital visits, asthma and respiratory symptoms to both indoor and outdoor air pollution in Jakarta. A study design that involves the collection of daily counts of mortality or emergency room visits in state-owned facilities for the city as whole, would be appropriate for comparing air pollution effects with those developed from the U.S. This effort would require conscientious data collection and management, and quality-assured

daily air pollution monitoring. Studies of symptomatology among the general population and of asthma exacerbation could be accomplished through recruitment of cohorts living within Jakarta. As data sets on health and air pollution are presently becoming available, including those from less developed countries, knowledge will likely improve dramatically over the next few years. Surely if such research can contribute to a better understanding of how to maximize the benefits of control programs, they can be well worth the cost.

Summary Table of Dose-Respose Functions: **Estimated Increment in Annual Health Effects Associated with Unit Change in Pollutants**

Outcome	Pollutants (Units)				
	PM10 (10 $\mu g/m^3$)	SO_2 (10 $\mu g/m^3$)	Ozone (pphm)	Lead (1.0 $\mu g/m^3$)	NO_2 (pphm)
Premature Mortality (% change)	0.96	0.48			
Premature Mortality/100,000	6.72				
RHA/100,000	12.0		7.70		
ERV/100,000	235.4				
RAD/person	0.575				
LRI/child	0.016				
Asthma symptoms/asthmatic	0.326		0.68		
Respiratory symptoms/person	1.83		0.55		
Chronic bronchitis/100,000	61.2				
MRAD/person			0.34		
Respiratory symptoms/1,000 children		0.18			
Respiratory symptoms/adults		0.10			0.10
Eye irritations/person			0.266		
Hypertension/100,000 adult males				7,260	
Coronary disease/100,000 adult males				34.0	
Premature Mortality/100,000 adult males				35.0	
IQ decrement (100,000) children)				97,500	

Note: RHA = Respiratory hospital admissions; ERV = Emergency room visits;
RAD = Restricted activity days; LRI = lower respiratory illness;
MRAD = Minor restricted activity days.
PPHM = Parts per hundred million

Table 6: Health Benefits of Reducing Particulate Matter to Indonesian Standard

Health Effect	Low	Medium	High
Premature Mortality	750	1,200	1,600
Hospital Admissions	1,100	2,000	2,700
Emergency Room Visits	22,100	40,600	59,100
Restricted Activity Days	4,460,000	6,330,000	9,905,000
Lower Respiratory Illness (children)	49,300	104,000	146,600
Asthma Attacks	232,000	464,000	3,885,000
Respiratory Symptoms	15,705,000	31,000,000	47,100,000
Chronic Bronchitis	4,800	9,600	14,300

Table 7: Health Benefits of Reducing Particulate Matter to WHO Standard

Health Effect	Low	Medium	High
Premature Mortality	900	1,400	1,900
Hospital Admissions	1,400	2,500	3,200
Emergency Room Visits	26,600	48,800	71,000
Restricted Activity Days	5,360,000	7,595,000	11,876,000
Lower Respiratory Illness (children)	59,200	125,100	176,200
Asthma Attacks	279,000	558,000	4,668,000
Respiratory Symptoms	18,873,000	37,331,000	56,619,000
Chronic Bronchitis	6,100	12,300	18,400

Table 8: Health Benefits of Reducing Particulate Matter to California Standard

Health Effect	Low	Medium	High
Premature Mortality	1,100	1,700	2,400
Hospital Admissions	1,700	3,100	4,100
Emergency Room Visits	33,600	61,700	89,800
Restricted Activity Days	6,762,000	9,571,000	14,949,000
Lower Respiratory Illness (children)	74,800	158,000	222,600
Asthma Attacks	353,000	705,000	5,900,000
Respiratory Symptoms	23,844,000	47,165,000	71,533,000
Chronic Bronchitis	8,000	16,000	24,000

Table 9: Health Benefits of Reducing Ambient Lead

	Standard		
Effect	Indonesian	WHO	90 Percent Reduction
Hypertension	62,350	135,660	241,000
Non-fatal Heart Attacks	68	190	559
Premature Mortality	57	158	461
IQ Loss (points)	1,091,860	2,073,205	3,130,918

Table 10: Health Benefits of Reducing Ambient Nitrogen Dioxide to Indonesian Standard

	Low	Medium	High
Respiratory Symptoms (millions)	1.04	1.77	2.50

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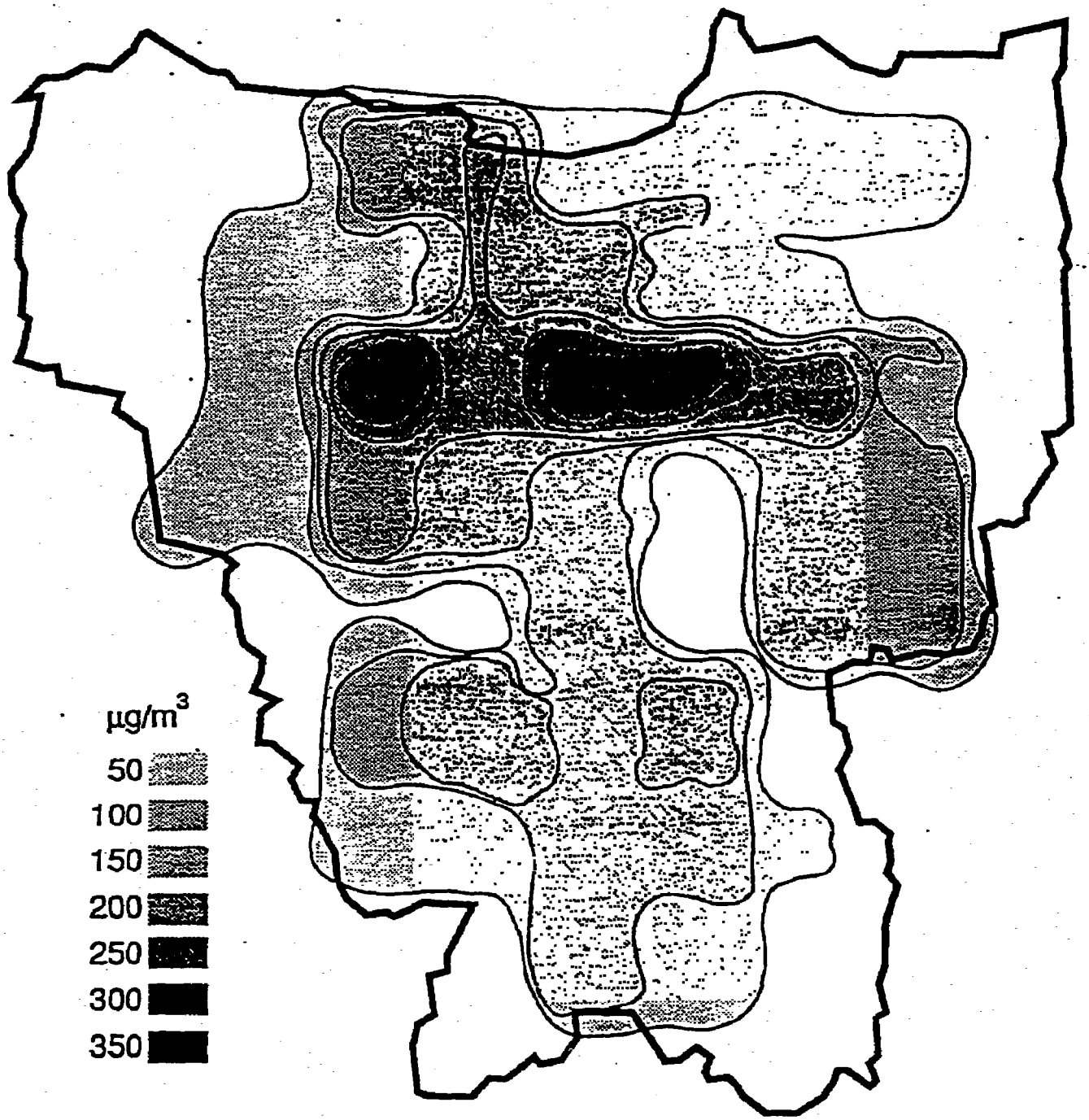
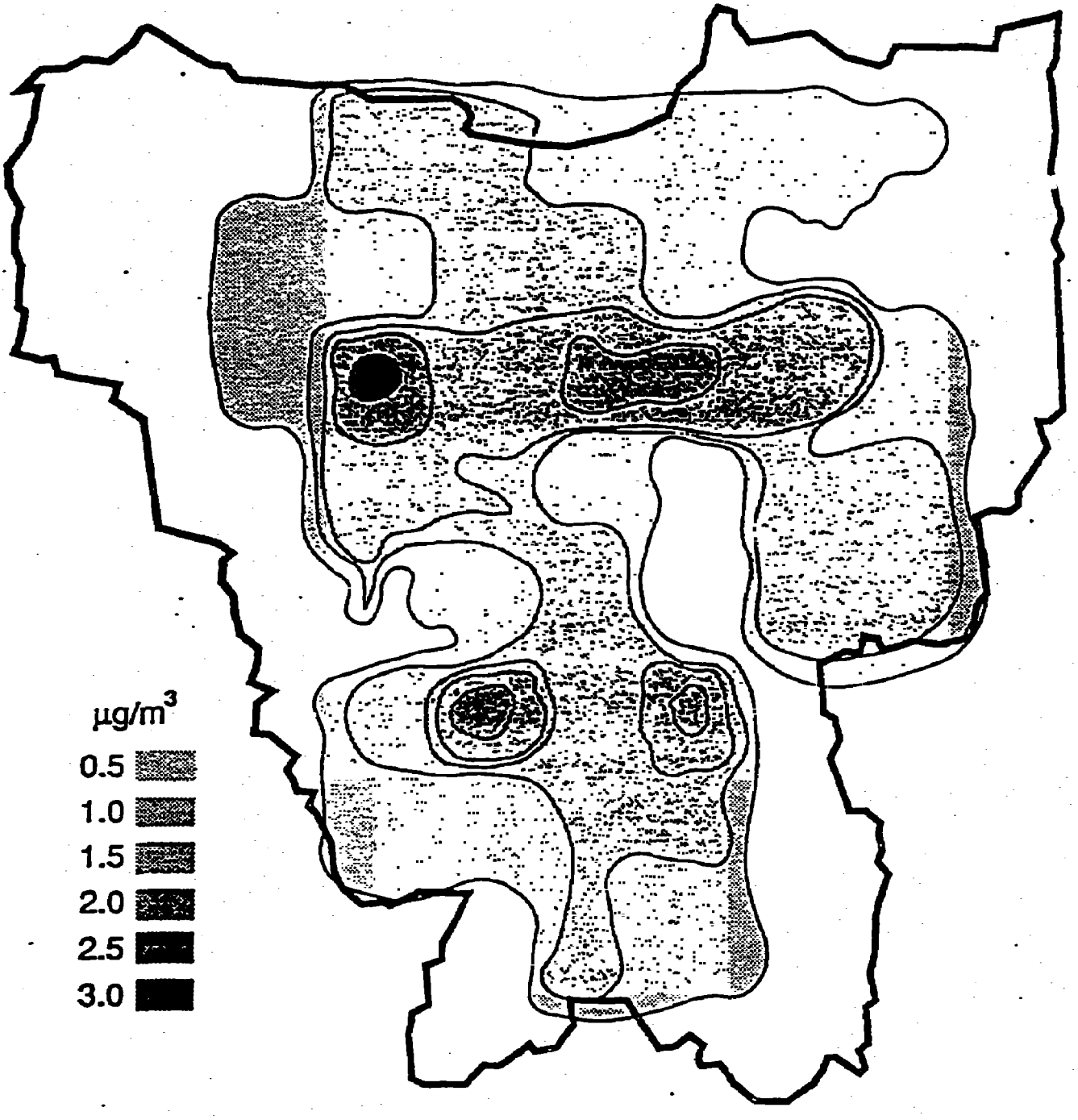


Isopleths of Annual Average NOx in Jakarta, 1989



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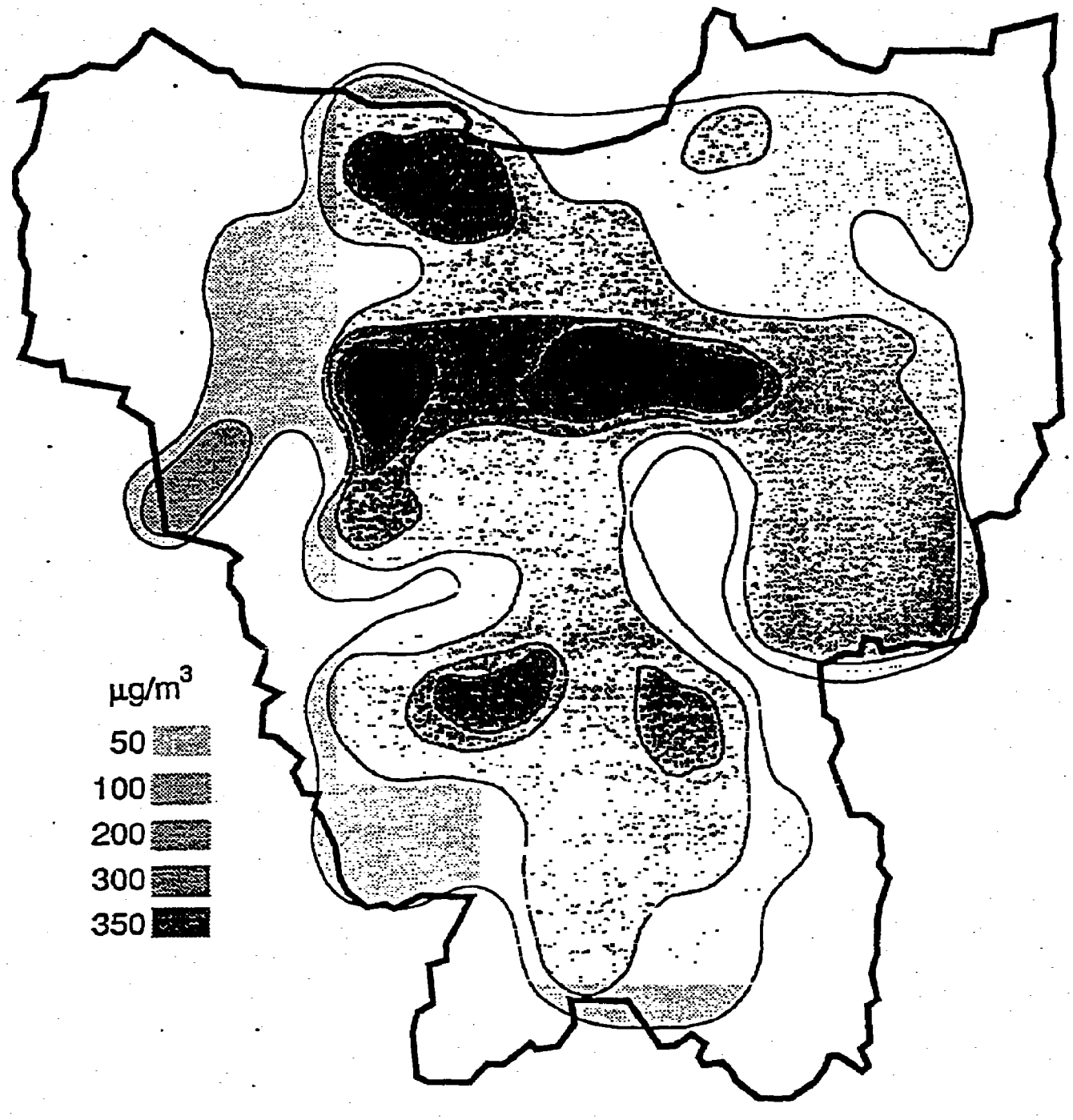

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