HEALTH AND ENVIRONMENT ANALYSIS FOR DECISION-MAKING

A METHODOLOGY FOR ESTIMATING AIR POLLUTION HEALTH EFFECTS

Office of Global and Integrated Environmental Health
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A Methodology for Estimating Air Pollution Health Effects

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ABSTRACT

While significant air pollution concentrations have been reported in developing countries in Latin America, Eastern Europe, and South and Southeast Asia, consistent information about the health implications of these environmental exposures is limited. Governmental health and environmental agencies in these regions have had little basis for prioritizing among alternative pollution control strategies. It is also difficult for these agencies to compare the benefits obtained from controlling air pollution with other social investments aimed at protecting or improving public health. This report presents a methodology for quantifying a broad range of health effects expected to be associated with exposure to a common air pollutant, particulate matter. Preliminary estimates are presented for Santiago, Chile, which experiences high levels of particulates primarily due to its unique topography and climate and from emissions from cars, taxis and diesel-powered buses. The results suggest that reductions in particles would result in significant health benefits. For example, for this population of 4.4 million, lowering annual PM10 (particulate matter less than 10 microns in diameter) to 30 μg/m³ would prevent 5,200 premature deaths and 13 million days of restricted activity due to respiratory illness each year.
I. INTRODUCTION

While significant air pollution concentrations have been reported in developing countries in Latin America, Eastern Europe, and South and Southeast Asia, consistent information about the health implications of these environmental exposures is limited. Governmental health and environmental agencies in these regions have had little basis for prioritizing among alternative pollution control strategies. It is also difficult for these agencies to compare the benefits obtained from controlling air pollution with other social investments aimed at protecting or improving public health. With limited resources, rational decision-making requires some quantification of the potential benefits of controlling air pollution. This report presents a methodology and preliminary results for quantifying the health effects of exposure to a common air pollutant, particulate matter, in Santiago, Chile. Although uncertainties exist about several of the components of this methodology, the results provide reasonable estimates of the range of effects that may be expected from current exposures to this air pollutant.

Previous efforts at estimating the health and economic benefits of reducing air pollution relating to alternative ambient air pollution concentrations have been undertaken by the United States Environmental Protection Agency (EPA) (EPA, 1984). Additional information and methodological improvements were incorporated in the subsequent analysis of economic benefits of air quality programs in selected U.S. locations (Chesnut et al., 1987). Recently, broad estimates of the health benefits of controlling particulate matter or ozone were provided for both the U.S. as a whole and for the ambitious pollution control plans under consideration in Southern California (American Lung Association, 1995; Hall et al., 1992; Krupnick and Portney, 1991). A generally similar methodology was employed to estimate the benefits of reducing lead in gasoline (Schwartz et al., 1985). Epidemiologic research over the last few years has provided additional evidence on the health effects of air pollution and provides a rich basis for predicting several adverse outcomes that are associated with exposure to air pollution. Using these more recent studies, Ostro (1994) provided a methodology for estimating the health effects of particulate matter, ozone, and lead. This methodology was then applied to determine the impacts of exposure to the current ambient air pollution concentrations experienced in Jakarta, Indonesia.

Following this introduction, there are four additional sections in this report. Section II provides background information on air pollution in Santiago, Chile to which the quantitative methodology is applied. Section III describes the methodology, assumptions and data used for estimating the health effects associated with air pollution. Section IV briefly reviews the literature that quantitatively links changes in air pollutants with subsequent 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 V applies the methodology and provides estimates of health effects of PM10 for Santiago. An Appendix is provided that details the calculations used to derive the dose-response estimates.

II. BACKGROUND

This report provides a methodology for and estimates of the health effects of particulate matter in Santiago, Chile using detailed air quality information and considering a broad range of potential health outcomes. Particulate-matter air pollution, sometimes referred to as "dust and soot," is a pollutant to which all of the general population is regularly exposed. It is typically monitored in the ambient air as either total suspended particles (TSP), which include particles of all sizes, or as particles 10 microns or less in diameter (PM10). The smaller particles, such as PM10 (or fine particles which are below 2.5 microns in diameter) are of greater health concern than particles of all sizes since they are more likely to penetrate into the deep lung. PM10 is a heterogeneous mixture of chemicals and particle sizes. It includes particles directly emitted into the air such as diesel soot, road or agricultural dust, or particles resulting from wood burning or manufacturing processes. It is also produced through photochemical reactions among pollution gases such as sulfur or nitrogen oxides produced from fuel
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combustion. In the mid-1980s, the United States converted from a TSP to a PM10 standard.

Chile has experienced extremely rapid economic growth since the early 1980s, with accompanying increases in traffic from automobiles and buses. In addition, the capital city of Santiago is now the home of 4.7 million people, almost a third of all Chileans. The high levels of PM10 experienced in Santiago are primarily a result of its unique topography and climate and from emissions from motor vehicles including private cars, taxis and diesel-powered buses. Fossil fuel use for energy production and industrial processes, and blowing and resuspended dust, also contribute to the problem. The city is situated in a basin surrounded by mountains, with fairly stable atmospheric conditions including low velocity, turbulence and frequency of winds. An inversion layer intensifies in the autumn and winter, preventing natural dispersion of pollutants and trapping most particles within 400 meters above the city. Thus, between the months of July and August, during the Chilean winter, particulate concentrations in Santiago are among the highest observed in any urban area in the world.

SECTION III. METHODOLOGY, BASELINE ASSUMPTIONS AND DATA

1. Methodology

To estimate the health impacts associated with air pollution, three factors must be resolved: the dose-response relationships, the susceptible populations impacted, and the change in air pollution under consideration. The product of these three factors generates the total health impact. The first factor involves the selection of estimates that relate exposure to air pollution to various health outcomes.

1. A. DEVELOPMENT OF DOSE-RESPONSE FUNCTIONS

Dose-response functions that correlate mortality and morbidity outcomes with ambient air pollution concentrations are taken from published epidemiologic studies, with support from the human clinical and animal toxicology literature. Epidemiologic studies provide "real world" evidence of associations between air pollution and health and are based on normal living conditions and exposures. The use of these studies eliminates the need for low-dose or cross-species extrapolation that would be required in animal toxicologic and most human clinical studies. However, it is difficult to isolate the effects of a specific air pollutant in these studies, since pollutants tend to covary in the natural environment. Finding a statistically significant association between a health effect and a specific air pollutant does not prove causality. The inference of causation is strengthened, however, if epidemiologic results are duplicated across several studies and if a range of effects is found for a given pollutant.

The development of dose-response estimates first involves the selection of scientific studies that are likely to provide the best estimates of an effect. From this study or studies, a marginal effect (or slope) is determined which provides an estimate of the change in the probability of a given health effect associated with a change in PM10, holding other factors constant. A basic review of the studies used for the dose-response estimates is provided in this report, and more complete reviews of the literature can be obtained in the EPA or World Health Organization (WHO) scientific reviews of the health effects of criteria pollutants (for example, EPA, 1995; WHO, 1987). Current efforts by WHO to assess the effects of air pollution are ongoing. While there is a vast amount of epidemiologic evidence linking PM10 with adverse health, considerable uncertainty about the precise quantitative relationships continues to exist. Recognizing the uncertainty in these estimates, high and low estimates are provided to indicate the likely ranges within which the actual damages are likely to fall. Within a given study there is statistically measurable uncertainty indicated by the reported confidence interval around any given estimate. Such studies, however, typically provide many empirical results, and researchers are, in fact, encouraged to do so in order to demonstrate the sensitivity of the results to the basic model assumptions. In addition, for certain health outcomes; multiple studies may provide empirical results. Therefore, for this report, the central estimate is typically selected from the middle of the range reported in the study or group of studies, or is the "best estimate" based on the original or this author's assessment of the performance of the models examined. For the mortality effects, a meta-analysis of the most relevant studies has been conducted to provide a central estimate and confidence interval. For morbidity outcomes, high
and low values based on a single study are based on plus or minus one standard error of the estimated regression coefficient. When several acceptable studies are available for a given health effect, the selected range affects the variation in results across the studies. Alternatives to this approach could include: (1) use of plus or minus two standard errors around the central estimate, (2) the use of the absolute highest and lowest estimates obtained, or (3) the development of formal meta-analytic approaches to provide central estimates and ranges. The first two approaches would increase, and the third would decrease the range around the central estimate. Therefore, the approach used in this report reflects a reasonable range of uncertainty.

To be included as a basis for the dose-response functions, an epidemiologic study had to meet several specific criteria.

- First, a proper study design and methodology were required. Studies were expected to have data based on continuous monitoring of the relevant pollutants, careful characterization and selection of exposure measures, and minimal bias in study sample selection and reporting. In addition, the studies had to provide dose-response relationships over a continuum of relevant exposures.

- Second, studies that recognized and attempted to minimize confounding and omitted variables were included. For example, studies that compared two cities or regions and characterized them as "high" and "low" pollution areas were not used for quantitative purposes because of potential confounding by other factors in the respective areas and vague definition of exposure.

- Third, controls for the effects of seasonality and weather had to be included. This could be accomplished by stratifying and analyzing the data by season, by examining the independent effects of temperature and humidity, or by other statistical methods such as smoothing techniques or pre-filtering.

- A fourth criterion for study inclusion was a reasonably complete analysis of the data. Such analysis would include a careful exploration of the primary hypothesis and preferably 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 quantitative estimates that were judged as most representative of the overall findings were those selected for use in this assessment.

- Fifth, the study had to provide an airborne particulate measure that could be reasonably converted into PM10. Therefore, studies that failed to quantitatively characterize air pollution or for which exposure assessment was poorly characterized were not included.

- Sixth, the study had to involve relevant levels of air pollution. Thus, studies that examined only high level pollution "episodes" were not relied on for quantitative information.

- Finally, only studies that addressed clinical outcomes or identifiable changes in behavior were included. Therefore, estimates for endpoints such as changes in lung function that may be difficult to link to clinically significant symptoms were not included.

Air pollution epidemiology studies typically involve estimation of a statistical relationship between the frequency of a specific health outcome observed in a given study population in its normal environment and air pollution concentrations measured at fixed-site monitors in the study area. The reported epidemiologic investigations involve two principal study designs: time-series and cross-sectional. Time-series analysis examines changes in a health outcome within a specific area as air pollution levels fluctuate. An example of this study design would be daily observations of emergency room visits and air pollution in a community over several years. A cross-sectional analysis compares differences in health outcomes across several cities at a selected point or period of time. This would include, for example, studies that compare chronic bronchitis rates in several locations at a single period of time. The time-series studies have the distinct advantage of reducing the problems associated with confounding or omitted variables, a common concern in the cross-sectional studies. Since the population characteristics (e.g., age, smoking habits, occupational exposure, health habits) are basically unchanged over the study period, the only factors that likely vary with daily mortality and morbidity are environmental and meteorologic conditions. In general, researchers are able to elicit more easily the effects of air pollution and weather on mortality using daily time-series analysis of acute effects. Therefore, dose-response estimates for this report are
based primarily on these types of studies. The use and extrapolation of results from time-series analysis, however, is predicated on its applicability to other areas and time periods.

It also needs to be stressed that the health endpoints quantified in this report are not all-inclusive, since quantitative evidence is not available for every health effect suspected of being associated with PM10. In addition, for certain effects for which statistical evidence of an effect appears convincing, appropriate information for the development of quantitative estimates may not be available. Examples of such cases will be provided below.

The estimates provided in the epidemiologic studies can be biased in either direction owing to statistical uncertainties. For example, other things being equal, random mismeasurement of exposure will tend to bias the estimated effect downward. In addition, a downward bias may result from individuals altering their normal behavior to avert the effects of air pollution. While the health effect may be averted, a social cost relating to this change in the preferred behavior is exacted. On the other hand, omitting confounding variables that may be related to both air pollution and the health effect will likely result in an upward bias. In general, however, the overall direction of bias in this methodology is more likely to be downward.

1.8. Determining the Exposed Population

The next step in quantification involves determining the relevant population to be included. Ideally, exposures for an entire metropolitan area can be developed, using either fixed-site monitors or dispersion models. For certain pollution-related health effects the relevant population may include the entire exposed population; for other effects there may be particularly sensitive subgroups, such as children, asthmatics or the elderly. Local data should be used to determine the appropriate proportion of the population within each of these subgroups. These populations are then assigned pollution exposures based on residence, and sometimes work location.

1.9. Relevant Change in Air Pollution

The third factor in the quantification process involves the determination of the change in air quality under consideration. Therefore, both the current ambient concentration and some appropriate “target” concentration such as an ambient air quality standard (or a threshold level) must be depicted. If there is an adequate monitor configuration within a metropolitan area so as to ensure reasonable representativeness, then readings from fixed-site monitors can be used to approximate current concentrations. Several options for the use of these monitors are available. For example, local populations can be assigned an exposure value equal to the closest local monitor. As an alternative, residents of the metropolitan area can be assigned an exposure based on some weighted average of all of the monitors in the region. The exposure assignment either can use equal weights for each site, weight central city monitors more heavily to incorporate the likelihood of exposure to and from work and during the workday, or use more sophisticated techniques. The latter may involve determining exposures for each individual by weighting each monitor (or a subset of monitors) by the inverse of its distance to the individual. Previous analyses have indicated, however, that the overall results are relatively insensitive to these alternatives.

Unfortunately, many major cities in developing countries do not have a comprehensive set of monitors. Therefore, as an alternative to monitors, dispersion models can be developed and used if major emitters can be well characterized. For example, in Jakarta, population and emission data for certain pollutants are disaggregated down to a grid of 5 x 5 km cells (Ostro, 1994). Dispersion models have been used, after incorporating local meteorologic and topographic features, to determine ambient concentrations throughout the region. Individuals were then assigned exposures based on their current residence. Again, alternatives to this procedure are available such as assigning a weighted exposure based on both residence and potential workplace (e.g., the central business district).

There is also latitude concerning the choice of a "target" concentration. Currently, the existence of threshold level PM10, below which no effects related to exposure are expected to occur, has not been identified. There is also little evidence that the slope of the dose-response function diminishes significantly at lower concentrations. Most of the epidemiologic studies reviewed have estimated fairly linear or near-linear functions that suggest a continuum of effects down to the lowest particulate matter levels observed in the study sample, and have not identified a threshold level. For example, for mortality, most studies report a linear association
between the relative risk in a population (percent increase in mortality) and the concentration of PM10. When efforts have been made to identify a threshold, little conclusive evidence has been found that one exists (Ostro, 1984). Burnett et al. (1995) conducted a threshold analysis in a study of hospital admissions to Canadian hospitals and particulate matter concentrations. Their preliminary results show statistically significant effects on days with PM10 equivalent as low as 25 μg/m³. Many recent epidemiologic studies show a consistent association between particulate matter and health effects across the entire range of measured particulate levels, including levels well below the current U.S. standards for particulate matter. For example, Schwartz and Dockery (1992a, 1992b) have found that the observed relationship between mortality and particulate matter in two eastern U.S. cities is similar across all four quartiles of daily particulate matter.

The lowest 5 to 10 percentile levels of particulate matter in these studies were in the range of 30 to 40 μg/m³ TSP (24-hour measurements). Likewise, studies involving PM10 in Santiago, Chile (Ostro et al., 1996) and Utah Valley (USA) (Pope et al., 1992) observed continuous effects down to 20 or 30 μg/m³.

On the basis of the evidence of health effects at relatively low particulate matter concentrations, the assumption recommended and used for this analysis is that effects are likely to occur down to 30 μg/m³, the annual average standard for PM10 in California. (The current U.S. annual average standard is 50 μg/m³.) However, some sensitivity analyses using other ambient concentrations is reasonable. Table 1 summarizes some current alternative standards. The World Health Organization (WHO) guidelines are included for comparative purposes.

<table>
<thead>
<tr>
<th>Standards or Guidelines</th>
<th>24 Hour Average</th>
<th>Annual Average</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brazil</td>
<td>150</td>
<td>50</td>
</tr>
<tr>
<td>California</td>
<td>50</td>
<td>30</td>
</tr>
<tr>
<td>China (urban areas)</td>
<td>165*</td>
<td></td>
</tr>
<tr>
<td>India (residential areas)</td>
<td>77</td>
<td></td>
</tr>
<tr>
<td>Indonesia</td>
<td>143*</td>
<td></td>
</tr>
<tr>
<td>Japan</td>
<td>100</td>
<td></td>
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<tr>
<td>Mexico</td>
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<tr>
<td>Russia</td>
<td>82.5*</td>
<td></td>
</tr>
<tr>
<td>United States</td>
<td>150</td>
<td>50</td>
</tr>
<tr>
<td>WHO</td>
<td>83 - 127*</td>
<td>33 - 50*</td>
</tr>
</tbody>
</table>

* TSP standard converted to PM10 assuming PM10 = 0.55 x TSP.

In sum, the estimated health impact can be represented by:

\[
\Delta H_{ij} = b_i (\Delta A_j) (POP_{ij})
\]

where:

\[\Delta H_{ij} = \text{change in population risk of health effect } i \text{ in region } j\]

\[b_i = \text{slope from dose-response curve for health effect } i \text{ indicating the expected health effects per unit of PM10}\]

\[\Delta A_j = \text{reduction in PM10 in region } j\]

\[POP_{ij} = \text{population at risk of health effect } i \text{ in region } j.\]
2. Baseline Assumptions

A basic assumption of this methodology is that the association between ambient concentrations, measured at fixed site monitors, and subsequent health effects can be extrapolated from the original study areas to the applied areas. These studies show that when the concentrations 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. To the extent that the original studies are 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 were to be conducted over a three-month period, and daily emergency room visits were associated with PM10, it is extremely unlikely that 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. The extrapolation of results from these epidemiologic studies assumes a fairly similar spatial relationship between pollution monitors and population. Thus, a 10 µg/m³ change in PM10 is expected to result in the same increase in risk in the applied area as in the original study area. 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 potential health impact of other regions. Obviously it would be beneficial to use as much data and epidemiologic results from the study area as possible.

An additional assumption in the methodology is that the adverse health outcomes and resultant behaviors observed in the epidemiologic studies undertaken in the United States and Europe (e.g., emergency room visits, restrictions in activity) provide reasonable indicators of the health burden on the population even though the actual utilization of health services in other countries may vary. For example, if a given number of emergency room visits are predicted from the dose-response functions, the methodology assumes that this is an indication of the degree of pain and suffering that will exist in the population under study. However, differences in the countries that are being analyzed, including cultural norms and availability and use of health services, will likely result in a difference in the observed number of emergency room visits. Thus, for this report, the analysis uses the behavioral health response in the United States as the “norm”. Once additional epidemiologic studies are conducted in other countries, this assumption can be revised.

Epidemiologic studies provide dose-response relationships between concentrations of PM10 and several adverse health outcomes including: mortality, respiratory hospital admissions, emergency room visits, restricted activity days for adults, lower respiratory tract illness for children, asthma attacks, respiratory symptoms 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. British Smoke and COH are measures of visibility impairing particles in the air. Only a few recent studies have actually used PM10 as the exposure metric. Therefore, for the determination of dose-response, alternative measures of particulate matter were converted into PM10, based on the best available information on their relationships. Thus, it was assumed that PM10 constitutes about 0.55 of TSP (EPA, 1982) and using the reported averages from 100 cities in the U.S. in 1980, that sulfates constitute approximately 0.14 of TSP (Ozkaynak and Thurston, 1987).

Since the data used for the application to Santiago are in terms of annual averages, this methodology ultimately relates changes in health status to annual average PM10 measured in micrograms per cubic meter (µg/m³). This requires that the regression coefficients from analyses using daily data be converted into annual equivalents and that a given change in the annual average will consist of equal changes on a daily level. If daily data on PM10 are available in the study area, then daily dose-response functions could be used and directly applied.

Uncertainty about the specific biologic mechanisms that underlie the associations observed in the epidemiologic studies remains. In addition, the specific constituent of particulate matter responsible for many of the adverse health outcomes is unknown. However, studies finding an effect of particulate matter have been conducted in many different cities and seasons, incorporating a wide range of climates, chemical compositions of particulates, and populations. For example, a similar magnitude of effect for particles has been reported in both high- and low-ozone areas, high- and low-sulfur dioxide
areas, where particles peak during the winter and where they peak during the summer. In addition, the range of studies tends to meet the criteria for causality often suggested for epidemiologic studies (Ostro, 1992). Therefore, it is assumed that reductions in PM10 will also reduce the causative agent of particulate matter associated with health.

3. Data

Santiago, the capital of Chile, is located in the center of a closed basin, about 33 degrees south latitude on the western edge of South America. The population of the metropolitan Santiago area is estimated to be 4.7 million, roughly one-third of the entire population of Chile of 13.2 million (Chile, Ministerio de Economia, 1990). In 1992, males are estimated to constitute 47.6 percent of the population of metropolitan Santiago, with 10.5 percent of the total consisting of children under age five, and 36.3 percent below age 18. The proportion of the population above age 25 is 46.9 (Chile, Instituto Nacional de Estadísticas, 1989).

Emissions data for particulate matter are provided for five main sources in Santiago (Sandoval and Martínez, 1990; Universidad de Chile, 1985). The emission sources for total suspended particulates are natural dust including from unpaved roads (72%), industrial (17%), residential (5%), diesel vehicles (4%) and gasoline-powered vehicles (2%). For PM10, the emissions sources are natural dust (49%), industrial (20%), residential (7%), diesel vehicles (19%) and gasoline-powered vehicles (5%). Clearly, diesel-powered vehicles contribute more to the fine mode particles than to all size particles, and are of particular concern. When considering the ambient concentrations based on monitors in downtown Santiago, these diesel sources constitute 74 percent of the ambient PM10, with gasoline-powered vehicles, industrial, and residential sources responsible for 6 percent, 6 percent and 2 percent of the concentrations, respectively (Chile, Comisión Especial de Descontaminación de la Región Metropolitana, 1990).

The existing monitor network provides incomplete spatial coverage for metropolitan Santiago; 4 of the 5 monitors recording daily PM10 readings are located in or near the central business district. Therefore, air pollution concentrations for 1993 were based on dispersion models developed by Ulriksen et al. (1994). These models divided Santiago into a grid of 289 cells and used local data on population and emissions, and metropolitan area data on topography and meteorology. The output of these models are isopleths that depict the annual average concentrations that will be expected in different parts of metropolitan Santiago. A similar set of isopleths have been determined for Jakarta (Ostro, 1994). For this example, the population residing in each cell was assigned the annual average reported in that cell.

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Section IV. Dose-Response Estimates Utilized

1. Mortality

1.1. Acute Exposure

Among the earliest empirical estimates 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 concentrations of particles measured as British Smoke (Martin and Bradley, 1960). Several researchers have analyzed the London data for 14 winters, 1958-59 through 1971-72 (Mazumdar et al., 1982; Ostro, 1984; Schwartz and Marcus, 1990). These efforts indicated that mortality was associated with air pollution over the entire range of ambient concentrations, not only during the high episodes associated with the earlier London winters.

Since these efforts, other research using regression analysis has demonstrated on a daily basis associations between concentrations of particles (TSP, PM10 or COH) and mortality in several cities in the United States including: Philadelphia, Pennsylvania (Schwartz and Dockery, 1992a), Steubenville, Ohio (Schwartz and Dockery, 1992b), Santa Clara County, California (Fairly, 1990), Birmingham, Alabama (Schwartz, 1991), and Utah Valley, Utah (Pope et al., 1992). These studies used daily counts of all-cause mortality as their outcome measure. They typically examined
the sensitivity of the regression results to alternative model specifications and carefully controlled for potential confounders such as weather, seasonality, day of the week, and other pollutants.

Recent studies conducted in Latin America support these findings. For example, Ostro et al. (1996) examined the association of daily PM10 and mortality in Santiago, Chile between 1989 and 1991. Multiple regression analysis was used to explain the variation in mortality, with particular attention to the influence of temperature and season. The results suggest a strong and consistent association between PM10 and total mortality, respiratory-mortality, and mortality for those above age 65. In general, the relative risks observed in this study were consistent with those reported in the U.S. studies reported above. Saldiva et al. (1995) examined the association between daily concentrations of particles and mortality among elderly people in São Paulo, Brazil. A statistically significant and robust association was reported between total mortality and PM10. In addition, a preliminary analysis of Mexico City (Borja-Aburto et al., 1995) for 1990 to 1992 also reports an association between particulate matter, measured as TSP, and daily mortality. The analysis controlled for weather, other pollutants and seasonality. Taken together, the studies on the United States and Latin America suggest a fairly linear association between the PM10 and the percent increase in mortality. This association appears to exist over the range of data from 30 μg/m³ to over 400 μg/m³.

Some of the mortality studies have also tested for associations between PM10 and disease-specific mortality. Specifically, an association between PM10 and either respiratory or cardiovascular mortality has been reported in analyses of Philadelphia (Schwartz and Dockery, 1992a), Santa Clara (Fairly, 1990), Birmingham (Schwartz, 1993), Utah Valley (Pope et al., 1992), and Santiago, Chile (Ostro et al., 1996). Estimates from these disease-specific findings can be extrapolated to generate quantitative estimates of mortality.

Other studies have found associations in England and Wales (Chinn et al., 1981), Lyon and Marseilles, France (Derrienic et al., 1989), Athens, Greece (Katsouyanni et al., 1993), Erfurt, Germany (Spix et al., 1993), Santiago (Salinas and Vega, 1995) and Beijing (Xu et al., 1994). These latter studies, however, are difficult to use for quantitative purposes.

Several reviews of these studies suggest that, after converting the alternative measures of particulate matter used in the original studies to PM10, the effects on mortality are very consistent (Ostro, 1993; Pope and Dockery, 1994; Schwartz, 1994). Specifically, the mean effect of a 10 μg/m³ change in PM10 implied by these studies is approximately 1.0 percent, with a range of effects of 0.5 percent to 1.6 percent. Among the Latin American studies, the results from Chile (Ostro et al., 1996) indicate that a 10 μg/m³ change in daily PM10 is associated with a 1.1 percent change in mortality, with a 95 percent confidence interval of 0.7 to 1.4 percent. The results from Mexico City (Borja-Aburto et al., 1993) indicate that a 10 μg/m³ change in PM10 (assumed to be 0.55 of TSP) is associated with a 1.0 percent change in mortality with a 95 percent confidence interval of 0.6 to 2.2 percent. The ranges reported in the Latin American studies are consistent with those found in the studies conducted for U.S. cities.

For the quantitative estimates of mortality, the dose-response functions are derived from a meta-analysis of the epidemiologic studies that used PM10 as the actual measure of particle concentration. Limiting the dose-response estimate to these studies reduces the uncertainty introduced through converting from TSP, sulfates or coefficient of haze. Each study estimate is weighted by the inverse of its variance to obtain a group estimate. Table 2 summarizes the studies used in the meta-analysis. Based on these results, the recommended dose-response coefficients for estimation of the cases of total mortality from acute exposure per 10 μg/m³ is 1.23 percent as a central estimate, with a range (based on a 95% confidence interval) of 0.46 to 1.93 percent.
Table 2  Results of Acute Mortality Studies Using PM10

<table>
<thead>
<tr>
<th>Location</th>
<th>Reference</th>
<th>% Change in daily mortality for each 10 µg/m³ change in PM10 (95% confidence interval)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Birmingham, AL</td>
<td>Schwartz (1993)</td>
<td>1.0 (0.2, 1.9)</td>
</tr>
<tr>
<td>St. Louis, MO</td>
<td>Dockery et al. (1992)</td>
<td>1.5 (0.1, 2.9)</td>
</tr>
<tr>
<td>Kingston, TN</td>
<td>Dockery et al. (1992)</td>
<td>1.6 (-1.3, 4.6)</td>
</tr>
<tr>
<td>Utah Valley, UT</td>
<td>Pope et al. (1992)</td>
<td>1.5 (0.9, 2.1)</td>
</tr>
<tr>
<td>Santiago, Chile</td>
<td>Ostro et al. (1995)</td>
<td>1.0 (0.4, 1.5)</td>
</tr>
<tr>
<td>COMBINED</td>
<td></td>
<td>1.23 (0.46, 1.93)</td>
</tr>
</tbody>
</table>

As an example of an application of the methodology, the calculation of the mortality effects of a hypothetical change in PM10 in Santiago are detailed below. The central estimate is assumed to be 0.123 percent change in mortality per µg/m³, using a crude mortality rate in Santiago of 5 per 1,000, a population of 4.7 million, and a hypothetical annual change in PM10 of 50 µg/m³. The total number of expected cases of premature mortality resulting from acute exposure is calculated as:

\[
\text{Expected Cases} = \text{percent effect per } \mu g/m^3 \times \frac{1}{100} \times \text{baseline mortality rate} \times \text{change in PM10} \times \text{exposed population}
\]

\[
\text{Expected Cases} = 0.123 \times \frac{1}{100} \times 0.005 \times 50 \times 4.7 \text{ million} = 1,445
\]

Quantitative estimates of air pollution-mortality effects can be generated using at least two methods. The first, described above, uses results of estimates of the impact of PM10 on the percent change in total (all-cause) mortality. The second method uses results of estimates of the impact of PM10 on disease-specific (i.e., respiratory and cardiovascular) mortality. Each method has certain advantages. Using total mortality ensures that, based on the original studies, all mortality cases affected by air pollution are included in the dose-response function. If only cardiovascular- and respiratory-specific mortality are used in the dose-response function, the mortality effect may be underestimated if death certificates used in the original studies were miscoded. However, use of disease-specific mortality may provide more accurate estimates of the effect of air pollution if the disease patterns in the region where this methodology is to be applied are very different from those in the original studies. For example, if some developing country has much higher infant and childhood mortality not related to air pollution and/or higher rates of accidental deaths, the application of the dose-response functions for total mortality may not be appropriate. If the composition of mortality is vastly different in the developing country, bias may result by applying an air pollution-related percent increase in mortality over the baseline crude mortality rate. Thus, using dose-response estimates for respiratory and cardiovascular-mortality and the associated local disease-specific mortality rates may provide better estimates of the air pollution impact on mortality since it better incorporates the local population at risk. Note that if total mortality functions are used, differences in population characteristics, per se, such as age structure, nutritional and overall health status, and smoking rates, and in local geography and climate, may not necessarily result in bias since these factors will be reflected in the crude mortality rate. For example, as reviewed earlier, the percent increase in mortality per µg/m³ of PM10 in Chile, with a much lower crude mortality rate, is similar to that found in many cities in the United States.

As indicated above, several studies provide a basis for developing disease-specific dose-response functions. The meta-analysis of Dockery and Pope (1993) suggests that a 10 µg/m³ change in PM10 is associated with a 3.4 percent change in respiratory mortality with a range (95% confidence interval) of
0.74 to 6.25 percent. For cardiovascular mortality, the analysis suggests a 10 μg/m³ in PM10 is associated with a central estimate of 1.4 percent, with a range of 0.6 percent to 2.6 percent. Therefore, these coefficients could be used with disease-specific mortality rates in the country or city under study to generate alternative estimates of mortality related to PM10.

If the expected change in annual average PM10 is large (e.g., greater than 100 μg/m³), greater accuracy will be obtained by using the formula for attributable risk. Specifically, the change in mortality could be calculated as:

\[
\text{Expected Cases} = r(1 + r) \times \text{CM}
\]

where \( r \) is the additional risk associated with the current level of particles (P) relative to the target or standard (Po); and CM is the current mortality per year at the concentration level P. CM and r are calculated by:

\[
CM = \text{mortality rate} \times \text{exposed population}
\]

\[
r = \frac{\text{estimated percent effect of PM10 per μg/m}^3 \times (1/100)}{\text{change in PM10}}
\]

Using some of the figures from the above example, assume a mortality rate of 5 per 1,000, a population of 4.7 million, and a hypothetical annual change (P - Po) in PM10 of 110 μg/m³. Under these circumstances, \( r = (0.123 \times (1/100)) \times 110 = 0.135 \), and CM = \((5/1000) \times (4.7 \text{ million}) = 23,500\). Therefore, the expected number of cases of mortality due to the change in PM10 = \(0.135/(1.135)\times23,500 = 2795\).

The mortality calculations related to acute exposure are likely to represent both cases where a subgroup is already very ill and close to death, and those cases where individuals may be under particularly compromising circumstances which render them temporarily more vulnerable to the added insult of air pollution. However, uncertainty about both the sensitive subgroup and the biologic mechanism continues to exist. A few researchers have attempted to address this question by examining the data to determine if there is a measurable decline in mortality several days after higher pollution days have taken their potential toll. This is a challenging statistical question and the evidence on this remains inconclusive at this time. If the only effect of particulate matter exposure were to accelerate oncoming death by a few days, average mortality rates would not differ between higher and lower pollution locations. However, cross-sectional studies do find differences in average mortality rates. Therefore, it is likely that the time-series results represent, in some cases, a significant shortening of life.

I.8. CHRONIC EXPOSURE

Since it is possible that cumulative long-term exposure to particles may also contribute to premature mortality, it is likely that the acute exposure studies described above underestimate the effects of air pollution. Two types of long-term exposure studies have found statistically significant associations between mortality rates and particulate matter levels in the United States. The first type uses an ecologic cross-sectional study design in which mortality rates for various locations at a single point in time are analyzed to determine if there is a statistical correlation with average air pollutant levels in each location. Such studies have consistently found measurably higher mortality rates in cities with higher average levels of particulate matter (Ozkaynak and Thurston, 1987; Evans et al., 1984; Lipfert, 1994). These studies each conducted a thorough examination of data for 100 or more U.S. cities, including average TSP or sulfate concentrations for each city, with special emphasis on the effects of including or excluding potential confounding factors such as occupations or migration. However, concern persists about whether these studies have adequately controlled for potential omitted and confounding factors.

A second type of long-term exposure study involves a prospective cohort design in which a sample is selected and followed over time in each location. Dockery et al. (1993) published results for a 15-year prospective study based on samples of individuals in six cities. Pope et al. (1995) published results of a seven-year prospective study based on samples of individuals in 151 cities in the United States. These studies are similar in some respects to the ecologic cross-sectional studies because the variation in pollution exposure is measured across locations rather than over time. These studies rely on the same type of pollutant exposure data as that used in the ecologic studies, which is based on average pollutant levels measured at stationary outdoor monitors in a given location. However, these studies use individual-level data so that other health risk factors can be better characterized. Specifically, the authors of the prospective studies were able to control for
mortality risks associated with differences in body mass, occupational exposures, smoking (present and past), alcohol use, age, and gender. Both of these studies report a robust and statistically significant association between exposure to particulate matter (measured as PM10 or fine particles) and mortality.

To quantify the effects of chronic exposure, the Pope et al. (1995) study used since it has a larger sample size. The empirical results for fine particulates were converted to PM10 using a ratio of 0.65. The study results imply that a 10 µg/m³ change in long-term exposure to PM10 is associated with a 4.2 percent change in annual all-cause mortality, with a 95 percent confidence interval of 2.3 to 6.3 percent.

Results from both the acute and chronic exposure studies are used to derive a range for the dose-response relationship to mortality. The low estimate for mortality, a 1.2 percent change per 10 µg/m³, is derived from the acute studies. The high estimate for mortality, a 4.2 percent change per µg/m³, is based on the chronic studies, which likely include both acute and chronic effects. The central estimate of 2.7 percent is based on the arithmetic mean of the low and high estimates.

2. Morbidity

Epidemiologic studies also report consistent associations between PM10 and several morbidity outcomes. A review of the relevant studies and the development of the dose-response functions are provided below. Greater detail on the development of the quantitative functions are provided in the Appendix. Once these functions are determined, quantitative estimates are generated by multiplying the pollution regression coefficient of the dose-response function (the expected change in health per unit of PM10) by the relevant population and change in air pollution, as described earlier.

2.1. Respiratory Hospital Admissions

Evidence for an effect of particulates on hospital admissions is provided by a study by Pope (1991). In this study a statistically significant association was found between monthly admissions for pneumonia, asthma and bronchitis, and monthly average PM10 in two valleys in Utah studied in the winters of 1985 through 1989. An association between hospital admissions and particles was also reported by Thurston et al. (1992) and Burnett et al. (1994, 1995). The Thurston et al. (1992) study examined the relationship between daily admissions and air pollution in Buffalo and New York City during the summer of 1988. The Burnett et al. (1994, 1995) studies examined admissions in southern Ontario from 1983 to 1988. In all studies, the pollutants associated with hospital admissions, sulfates and ozone, were highly correlated, making it difficult to assign an effect to any specific pollutant.

The Pope study results are selected for quantifying the hospital admissions effects for PM10, since there is little risk that these results are confounded by a concurrent ozone effect; ozone was close to background level during the study period. Pope's estimates for the two counties were based on monthly admissions. Therefore, to obtain dose-response functions, the reported linear regression coefficients were averaged and annualized. The details of the calculations are provided in the Appendix. The central estimate suggests about a three percent increase in hospital admissions for each 10 µg/m³ increase in PM10 and are applied to the total population. The low, central, and high coefficients for hospital admissions are summarized in Table 3.

2.2. Emergency Room Visits

Samet et al. (1981) analyzed the relationship between daily emergency room visits and air pollution levels (TSP) in Steubenville, Ohio in 1974 through 1977. At least two other studies provide support for an effect on emergency room visits: Mazumdar and Sussman (1983) and Schwartz et al. (1993). The former reports an association between COH and urgent and emergency room care in Allegheny County (Pittsburgh metropolitan area) during 1972 through 1977. Schwartz et al. (1993) examined the association between PM10 and emergency room visits for asthma in the Seattle metropolitan area for one year beginning September 1989. However, the Samet study is used since the COH to PM10 conversion is uncertain and all causes of emergency room visits, not only those related to asthma as in the Schwartz et al. (1993) study, are included.

In the Samet et al. study, daily counts of emergency room visits for all individuals and all causes were associated with TSP, after controlling for the influence of weather. Details of the derivation of the dose-response functions are provided in the Appendix.
2.6. Restricted Activity Days

The annual Health Interview Survey from the National Center for Health Statistics includes data on restricted activity days, including days spent in bed, days missed from work, and other days when activities are significantly restricted due to illness. Ostro (1987) reported a consistent relationship between these restrictions in adults and concentrations of fine particles, estimated from airport visibility, for 49 metropolitan areas in the United States, for each of the six years, 1976-1981. A time-series model with a fixed effect component to control for differences among cities was used. The mean of the effect over the six years is used to calculate the central estimates, and upper and lower estimates were derived from the range in the coefficients over the six years. Additional work conducted by Ostro and Rothshchild (1989) added ozone to the model specification and found that the estimated effect of particles on restrictions in activity was essentially unchanged. Based on data reported in Ostro (1990), the assumed ratio of PM2.5 to PM10 is 0.625. These latter results also indicated a similar association between restrictions in activity and fine particles measured directly from hi voi monitors. This endpoint is applied to the population above age 16. The details of the calculations are supplied in the Appendix.

Table 3. Dose-Response Coefficients Used to Estimate Annual Effects of Annual Average Change in PM10.

<table>
<thead>
<tr>
<th>Health Endpoint</th>
<th>Coefficient estimate</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Low</td>
</tr>
<tr>
<td>Mortality percent increase per 1 μg/m³</td>
<td>0.123</td>
</tr>
<tr>
<td>Respiratory Mortality percent increase per 1 μg/m³</td>
<td>0.074</td>
</tr>
<tr>
<td>Cardiovascular Mortality percent increase per 1 μg/m³</td>
<td>0.060</td>
</tr>
<tr>
<td>Respiratory Hospital Admissions cases per 1 μg/m³</td>
<td>0.657 x 10⁻¹</td>
</tr>
<tr>
<td>Emergency Room Visits cases per 1 μg/m³</td>
<td>12.83 x 10⁻²</td>
</tr>
<tr>
<td>Restricted Activity Days cases per 1 μg/m³ per adult above age 16</td>
<td>0.040</td>
</tr>
<tr>
<td>Acute Bronchitis cases per 1 μg/m³ per child below age 16</td>
<td>0.8 x 10⁻³</td>
</tr>
<tr>
<td>Asthma attacks cases per 1 μg/m³ per asthmatic</td>
<td>0.033</td>
</tr>
<tr>
<td>Respiratory Symptoms cases per 1 μg/m³</td>
<td>0.091</td>
</tr>
<tr>
<td>Chronic Bronchitis cases per 1 μg/m³ per adult above age 16</td>
<td>3.06 x 10⁻³</td>
</tr>
</tbody>
</table>

¹ All functions are for the entire population except as noted.
² Disease-specific mortality can be estimated in place of all-cause mortality.
2.d. Lower Respiratory Illness in Children

Estimates of lower respiratory illness in children are based on an analysis by Dockery et al. (1989) of children in six cities in the United States. The study related the prior year's exposure of particulate matter to the incidence of acute bronchitis, as measured during health examinations of samples of children in each city. The logistic regression results indicated a statistically significant association between particulate matter less than 15 microns in diameter (PM15) and the probability of a child having acute bronchitis in the past year. The average probability of bronchitis reported in the study sample was 6.47 percent. The results are applied to the proportion of the population below age 16.

2.e. Asthma Exacerbation

In a study of asthmatics in Los Angeles, Whittemore and Korn (1980) reported a relationship between exacerbation of asthma and daily concentrations of TSP and ozone, using logistic regression analysis. Also, Ostro et al. (1991) reported an association between several different air pollutants, including sulfates, and increases in asthma attacks among adults residing in Denver. These two studies are used and estimates are applied to the five percent of the population assumed to have asthma (Centers for Disease Control, 1992). Additional evidence for an effect of particulate matter on asthmatic children is provided by Pope et al. (1991) and Ostro et al. (1995).

The Ostro et al. (1991) study took place during the winter months when the effect of particulate matter was not confounded by ozone levels, which were close to background concentrations. However, it is likely that winter-time respiratory infections enhanced the effects of air pollution on asthma exacerbation. Therefore, this study is used to generate upper bounds of the effects. The Whittemore and Korn study is used to derive the central and low estimates. Their regression model includes both TSP and ozone and controls for weather, so the effects on asthma are specific to particles. The predicted exacerbations of asthma probably reflect a wide range of severity, based on the reporting of symptoms by the study panels, and include mild to severe cases of shortness of breath, wheeze, chest tightness, and cough.

2.f. Respiratory Symptoms

Respiratory symptoms are an additional measure of acute effects of particulate matter. Results of a pooled cross-section and time-series study by Krupnick et al. (1990) is used to determine the effects of particulate matter on acute symptoms. This study of a panel of adults in Southern California found an association between the daily occurrence of upper and lower respiratory symptoms and both particulate matter and ozone. A follow-up study by Ostro et al. (1993) confirmed the results for lower respiratory symptoms. In these studies, daily health diaries were maintained for 6 months to reflect the presence of any of 19 respiratory symptoms including chest discomfort, cough, wheeze, chest cold, and flu. A regression model using a Markov process was developed to determine the effects of air pollution on respiratory symptoms. The model incorporated the probability of illness on the prior day and controlled for autocorrelation. In addition to air pollution, the regression model included socioeconomic measures (income and education), the presence of a chronic condition, and smoking habits. The results with multiple pollutants in the separate models for children and adults showed statistically significant coefficients of roughly similar magnitude for coefficient of haze. The effects of particles versus ozone were assumed to be proportional to the estimated regression coefficients when both pollutants were included in the model along with temperature. COH was converted to TSP using monitoring data on both pollutants collected during the study. The derivation of the dose-response estimates are detailed in the Appendix and are summarized in Table 3.

The estimates are applied to the entire population. However, to avoid double counting of respiratory symptoms, net symptoms were calculated as the total minus the above estimates of asthma exacerbation, restricted activity days, emergency room visits, episodes of acute bronchitis (times 14 days each) and hospital admissions (times 5 days each).

2.g. Chronic Bronchitis

Several epidemiologic studies have related long-term exposure of air pollution to a higher prevalence of chronic respiratory disease or significant decrements in lung function. Much of the evidence, however, has been based on cross-sectional studies which compare disease prevalence in two different
A METHODOLOGY FOR ESTIMATING AIR POLLUTION HEALTH EFFECTS

Communities with different average pollution levels. For example, Dutels et al. (1991) 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 to these pollutants. Hodgkin et al. (1984) compared the chronic respiratory disease status of respondents who had lived at least 11 years in either a high or low air-pollution area in California. These cross-sectional studies are able to suggest a possible association, but are difficult to use for quantitative estimates of specific dose-response functions. This difficulty arises from uncertainty about the relevant exposure period. Chronic symptoms presumably occur as a result of long-term exposures, but cross-sectional analyses do not provide information about whether the relevant exposure period is, for example, the five-year average, the twenty-year average, or the accumulation of exposure to periodic pollution episodes. Without this information, it is difficult to predict quantitatively how risks change when exposures change.

However, recently conducted studies (Abbey et al., 1991, 1993) provide some basis for quantification of effects. These analyses are cohort studies of almost 4,000 members of the Seventh Day Adventist Church, nonsmokers who had lived for at least 11 years in California. In this study, participants above age 25 who had been interviewed in 1977 were again interviewed in 1987. Several different chronic disease outcomes were obtained in the survey. These disease outcomes were matched with long-term measures of exposure in order to provide more definitive dose-response estimates. Estimates of air pollution exposures were based on subjects' monthly reported residential locations over the 11-year period and nearby outdoor TSP monitors at each location. However, uncertainties about the effects of long-term exposure, such as the length of exposure necessary to generate an effect and the lag time between exposure and response, continue to exist.

The Abbey et al. (1991, 1993) studies obtain several different health outcomes including new cases of emphysema, chronic bronchitis, or asthma reported in 1987 by those not reporting any definite symptoms of these diseases in 1977. Disease definition was based on self-reported symptoms using the standardized respiratory symptoms questionnaire developed by the National Heart and Lung Institute for the United States. Respondents were classified as having definite symptoms of emphysema, chronic bronchitis or asthma if they met specific criteria for the disease diagnosis. Having definite chronic bronchitis was defined as having symptoms of cough and/or sputum production on most days for at least 3 months/year, for 2 years or more. Emphysema and asthma required physician's diagnosis as well as associated symptoms. Having definite symptoms of any one of these three was defined as definite airway obstructive disease (AOD). Respondents with some respiratory symptoms, but who did not meet the full criteria for that disease, were classified as possible. Logistic models were estimated for mean concentrations of TSP assigned to each person and for frequency of hours above selected levels (60, 75, 100, 150, and 200 µg/m³). The regressions included explanatory variables for past and passive smoking exposure, possible symptoms in 1977, childhood respiratory illness, gender, age and education. A statistically significant association was reported between long-term mean TSP exposure levels and chronic bronchitis as well as AOD.

The chronic bronchitis results were selected for quantification in this methodology because it is less ambiguous than AOD. The estimates based on these findings are conservative for several reasons. First, these estimates do not reflect any mortality due to chronic respiratory disease that may have occurred during the 10-year period. Subjects are in the sample only if they were alive in 1987 as well as in 1977. Second, these estimates may be conservative since they reflect only the development of new cases. The authors report evidence that increased severity of pre-existing (in 1977) chronic disease is associated with TSP exposure. Thus, it appears that TSP exposure both aggravates existing cases and causes new cases. However, two major uncertainties in the quantitative estimates based on Abbey et al. (1993) should be noted. First, the authors have reported that a few subjects who give symptoms that are classified as chronic bronchitis do not continue to report these symptoms in follow-up studies. This suggests that these were not true chronic bronchitis cases. Likewise, there may be some under-reporting of illness. The second uncertainty involves the length of time a change in PM10 exposure must exist before a change in chronic bronchitis incidence occurs. The Appendix details the derivation of the risks estimates using the Abbey et al. (1993) study.
Section V. Estimates of Health Effects for Santiago

The estimates of the annual reduction in health effects achieved from reaching an annual average PM10 level of 30 μg/m$^3$ throughout Santiago, Chile are provided in Table 4. Recall that current levels of PM10 were predicted from dispersion models developed for Santiago using local data on population and emissions, and regional data on meteorology and topography (Uliksten et al., 1994). The existing PM10 monitor network, located primarily in or near the central business district, indicated annual averages of 115 μg/m$^3$ for 1991 through 1993 (Ostro et al., 1996). However, the dispersion model for Santiago, which divided the metropolitan area into 289 5 x 5 km grid cells, indicates a range for the annual average PM10 of 66.7 μg/m$^3$ to 139.6 μg/m$^3$. When each of the cells are weighted by their respective populations, the resultant population-weighted average annual is 112.6 μg/m$^3$. The total annual average exposure above 30 μg/m$^3$ is 389,244,000 person·μg/m$^3$ of PM10. Table 4 provides the results generated from reducing each of the grid cells from their current levels to 30 μg/m$^3$. The Table provides the low, central and high estimates for the population based on the dose-response estimates indicated in Table 3.

For example, if annual average PM10 concentrations were reduced to 30 μg/m$^3$ throughout the metropolitan area, there would be an expected annual reduction in premature mortality of 5,240 with a range of 2,400 to 8,150. The central estimates for Santiago also indicate an annual reduction of 4,670 hospital admissions for respiratory disease, 91,600 emergency room visits, 1.1 million asthma attacks, 13.4 million restricted activity days including work loss, 239,000 cases of childhood bronchitis, 55 million days with respiratory symptoms and 15,000 cases of chronic bronchitis.

The sensitivity of the results to assumptions about the current and threshold PM10 concentrations are presented in Table 5 using the central estimates for mortality. Model 1 uses the default assumptions described above. The current annual average population exposure is assumed to be the weighted average of the concentrations reported for each cell based on the dispersion model (112.6 μg/m$^3$) with a presumed threshold level of 30 μg/m$^3$. Models 2 and 3 examine the implications of thresholds of 50 and zero, respectively. A threshold of 50 lowers the mortality estimate by about 24 percent while a zero threshold raises the estimate by 36 percent. Model 4 assumes a current annual average concentration based on the existing central city monitors (115.5 μg/m$^3$) with a 30 μg/m$^3$ threshold. This generates an estimate that is 3 percent above the estimate of Model 1. For Model 5, it is assumed that 50 percent of the population receives an exposure represented by the central city monitors while the other 50 percent receives an exposure that is 75 percent of these monitor readings. This generates an annual population average of 101 μg/m$^3$ with a 14 percent reduction in the estimate of mortality relative to Model 1. These results indicate that using the central city monitors generates results similar to those obtained from the dispersion model, with only a 3 percent difference. The sensitivity analysis also indicates that very different assumptions about the threshold level only generate a 2.5 percent difference in the central estimate. Regardless of the model assumed, the results of this example strongly suggest that significant health benefits will be obtained through reductions in ambient particulate matter in Santiago.
Table 4. Predicted Health Benefits of Meeting PM10 Ambient Standard of 30 μg/m³ in Santiago, Chile.

<table>
<thead>
<tr>
<th>Health Endpoint</th>
<th>Estimate of Effects of Achieving 30 μg/m³ PM10</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Low</td>
</tr>
<tr>
<td>Mortality</td>
<td>2,400</td>
</tr>
<tr>
<td>Respiratory Hospital Admissions</td>
<td>2,600</td>
</tr>
<tr>
<td>Emergency Room Visits (thousands)</td>
<td>50</td>
</tr>
<tr>
<td>Restricted Activity Days in Adults (thousands)</td>
<td>9,600</td>
</tr>
<tr>
<td>Acute Bronchitis in Children (thousands)</td>
<td>110</td>
</tr>
<tr>
<td>Asthma Exacerbation (thousands)</td>
<td>640</td>
</tr>
<tr>
<td>Respiratory Symptoms (thousands)</td>
<td>25,200</td>
</tr>
<tr>
<td>Chronic Bronchitis (thousands)</td>
<td>8</td>
</tr>
</tbody>
</table>

Note. Estimates assume total population of 4.7 million, crude mortality rate of 5/1000, 36.3 percent of the population is below age 16, and 5 percent of the population is asthmatic.

Table 5. Sensitivity of Mortality Estimates to Alternative Assumptions About Ambient Concentrations

<table>
<thead>
<tr>
<th>Model</th>
<th>Annual Average Current PM10 Concentration (μg/m³)</th>
<th>Annual Average Assumed PM10 Threshold (μg/m³)</th>
<th>Central Estimate of Mortality</th>
<th>Percent Change from Model 1</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>112.6</td>
<td>30</td>
<td>5,240</td>
<td>-</td>
</tr>
<tr>
<td>2</td>
<td>112.6</td>
<td>50</td>
<td>4,000</td>
<td>-24</td>
</tr>
<tr>
<td>3</td>
<td>112.6</td>
<td>0</td>
<td>7,150</td>
<td>+36</td>
</tr>
<tr>
<td>4</td>
<td>115.5</td>
<td>30</td>
<td>5,400</td>
<td>+3</td>
</tr>
<tr>
<td>5</td>
<td>101</td>
<td>30</td>
<td>4,500</td>
<td>-14</td>
</tr>
</tbody>
</table>

NOTE: For Models 1-3, the assumed current exposure is the concentration reported for each cell based on the dispersion model weighted by the population in each respective cell. Models 1, 2 and 3 examine the implications of thresholds of 30, 50 and zero, respectively. Model 4 assumes a current annual average exposure based on the existing central city monitors with a 30 μg/m³ threshold. For Model 5, it is assumed that 50 percent of the population receives an exposure represented by the central city monitors while the other 50 percent receives an exposure that is 75 percent of these monitor readings.
REFERENCES


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APPENDIX

1. Respiratory Hospital Admissions

Linear regression results reported by Pope (1991) suggest the following relationships between monthly respiratory hospital admissions (RHAs) and PM10 in each valley. The reported coefficients (plus and minus one standard error) are divided by the reported population in each valley to give RHA per person per unit of PM10.

- Utah Valley: Monthly RHA per person = 0.95 (± 0.53) x 10^4 x PM10m
- Salt Lake Valley: Monthly RHA per person = 1.03 (±0.36) x 10^4 x PM10m

where:

PM10m = change in monthly average PM10 in μg/m³.

Dividing by 30.4 days per month gives the average daily RHA per person for each 1 μg/m³ change in daily PM10. This is a straightforward transformation given the linear form of the original monthly regressions. The Pope results thus imply the following daily relationships.

- Utah Valley: Daily RHA per person = 3.13 (±1.8) x 10^4 x PM10d
- Salt Lake Valley: Daily RHA per person = 3.39 (± 1.2) x 10^4 x PM10d

where:

PM10d = change in 24-hour PM10 in μg/m³.

Averaging the Pope results for the two study areas and annualizing by multiplying by 365 days gives the following relationships which are used to calculate hospital admissions:

- High annual RHA = 1.56 x 10^4 (PM10) (POPj)
- Central annual RHA = 1.20 x 10^4 (PM10) (POPj)
- Low annual RHA = 0.66 x 10^4 (PM10) (POPj)

where:

POPj = population in area j
PM10 = change in annual PM10 in area j.

2. Emergency Room Visits

The Samet et al. (1981) estimated coefficient for daily TSP in a linear regression of daily emergency room visits (ERV) on TSP and temperature was 0.011. The authors do not report a standard error, but report a p-value of less than 0.05. This implies a standard error of no more than about 0.0055. The coefficient must be divided by the Steubenville population of 31,000 at the time of the study to obtain an estimate of per capita ERV. Dividing by 0.55 to convert to PM10 generates a daily relationship, and multiplying by 365 annualizes this dose-response relationship.
function to obtain the following relationship. The high and low estimates are based on plus or minus one standard error of the regression coefficient.

High annual ERV = $3.53 \times 10^4 \ (PM_j) \ (POP_j)$
Central annual ERV = $2.35 \times 10^4 \ (PM_j) \ (POP_j)$
Low annual ERV = $1.18 \times 10^4 \ (PM_j) \ (POP_j)$

where:

POP,<sub>j</sub> = population in area,<sub>j</sub>
PM,<sub>j</sub> = change in annual average PM10 in area,<sub>j</sub>

3. Restricted Activity Days

The mean of the regression coefficients from the six separate years of analysis by Ostro (1987) for restricted activity days (RAD) for all adults were selected for the central estimate for this analysis (mean coefficient = 0.0048). The high and low estimate were derived from the mean of the two highest coefficients (0.0076) and two lowest (0.0034) in the six year analysis. These coefficients give the percentage changes in RAD for a 1 μg/m<sup>3</sup> change in PM2.5. The functions need to be converted from PM2.5 to PM10 and annualized before being applied to the adult population 18 years and over. To convert to an annual coefficient, the annual average estimate of 19 RAD per person, based on Health Interview Survey data, was used. Based on information reported by Ostro (1987), the assumed ratio of PM2.5 to PM10 was 0.625. Thus, the central estimate is derived from the following:

Percent change in RAD per year = 0.0048 x Δ PM2.5

Therefore, converting from a percent change in daily RAD per μg/m<sup>3</sup> of PM2.5 to the total number of RAD per year per μg/m<sup>3</sup> of PM10 implies the following:

Total RAD per year = 0.0048 x 19 x 0.625 = 0.057 x Δ PM10

Following the same procedure for the high and low estimates, the following are obtained:

High annual RAD = 0.09 (PM,<sub>j</sub>) (POP18,<sub>j</sub>)
Central annual RAD = 0.057 (PM,<sub>j</sub>) (POP18,<sub>j</sub>)
Low annual RAD = 0.04 (PM,<sub>j</sub>) (POP18,<sub>j</sub>)

where:

POP18,<sub>j</sub> = population in location,<sub>j</sub> 18 years of age and older
PM,<sub>j</sub> = change in annual average PM10 in area,<sub>j</sub>

4. Lower Respiratory Illness in Children

(Dockery et al. 1989) used logistic regression and reported an association between the probability of acute bronchitis and PM15 (particulate matter 15 microns or less in diameter). The dose-response can be calculated by taking the partial derivative of the logistic function with respect to PM15 to obtain:

\[ \Delta B = b \times Pr \times (1 - Pr) \times \Delta PM15 \]

where:
A METHODOLOGY FOR ESTIMATING AIR POLLUTION HEALTH EFFECTS

\[ B = \text{probability of the health outcome} \]
\[ b = \text{estimated regression coefficient} \]
\[ \text{Pr} = \text{baseline prevalence of bronchitis} \]
\[ \text{PM15} = \text{annual concentration of PM15} \]

These calculations apply to the population under age 18. The function is linearized using the average probability of bronchitis in the study sample, which was 6.47 percent. The estimated regression coefficient for bronchitis was 0.0237, with plus or minus one standard error of 0.0117. For converting to PM10, a ratio of PM10 to PM15 of 0.9 was assumed. The annual central change in the number of children with bronchitis (B) in a year as a function of PM10 is calculated as follows.

\[ B = 0.0237 \times 0.0647 \times 0.9353 \times (1/9) = 1.60 \times 10^{-3} \]

Therefore, the dose-response functions become:

- High annual bronchitis = \( 2.38 \times 10^{-3} (\text{POPL18}) \)
- Central annual bronchitis = \( 1.60 \times 10^{-3} (\text{POPL18}) \)
- Low annual bronchitis = \( 0.80 \times 10^{-3} (\text{POPL18}) \)

where:

- \( \text{POPL18}_j \) = population less than age 18 years in area \( j \)
- \( \text{PM}_i \) = change in annual average PM10 in area \( j \).

5. Asthma Attacks

Since the model used by Whittemore and Korn (1980) relating asthma attacks to particles is non-linear (i.e., logistic), calculation of a dose-response requires the determination of a baseline probability rate for asthma symptoms. The average rate of asthma symptom days for the Whittemore and Korn study sample is available, but it is likely to be an overestimate since the authors report that many subjects with a low frequency of asthma exacerbation were excluded. More representative data on average asthma symptom frequency is, however, not available at this time. It is therefore necessary to make some reasonable assumptions about the average rate.

In the Los Angeles study sample, about 26 percent of the sample experienced elevated asthma symptoms on any given day. If all of the excluded potential subjects are presumed to have had no elevated asthma symptoms during the study period and this is factored into the calculation, the average daily symptom rate is reduced to 13 percent. This is similar to the 15 percent shortness of breath frequency reported by Ostro et al. (1991). Also, Holguin et al. (1985) report an average daily asthma symptom rate of 15 percent for their study sample in Houston, or 13 percent if those excluded from the study are factored in. As a check on the plausibility of these rates as representative of the active asthmatic population, asthma severity information reported by the National Center for Health Statistics (1980) can be utilized. They report that all of active asthmatics in the United States, 55 percent have mild symptoms, 32 percent have moderate symptoms and 13 percent have severe symptoms.

If we assume that mild means one symptom per month, moderate means one symptom per week, and severe means a symptom every other day, the average daily symptom rate would be 13 percent. We select this lower rate to minimize the chance of overstating the expected effect of PM10 on the average asthmatic. From Whittemore and Korn, the probability of an increased asthma symptom on a given day as a function of TSP levels is determined by taking the partial derivative of the logistic function to obtain:

\[ \Delta \lambda = b \times \text{Pr} \times (1 - \text{Pr}) \Delta \text{TSP} \]

where:
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\[ A = \text{change in daily probability of an asthma symptom} \]
\[ b = \text{the estimated logistic coefficient for TSP.} \]
\[ Pr = \text{baseline probability of an asthma attack.} \]
\[ TSP = \text{daily TSP}. \]

Substituting 13 percent for the baseline probability and the estimated TSP coefficient of 0.00079 (adjusted to PM10 by dividing by 0.55) into the above equation, and annualizing by multiplying by 365, gives the following results:

Annual asthma attacks = \[(7.9 (\pm 3.4) \times 10^{-4}/0.55) \times 0.13 \times (1 - 0.13)] \times 365 = 0.059(\pm 0.026) \times \Delta \text{PM10}\]

The Ostro et al. (1991) results suggest the following relationship between elevated asthma symptoms and the log of daily sulfate (S04) concentrations:

\[ \text{Daily asthma attacks} = 0.0077 (\pm 0.0038) \times \log(\text{sulfate}) \]

Using the reported sulfate mean for the study of 2.11 µg/m³ to linearize the function, converting to PM10 (assuming a ratio of sulfate to PM10 of 0.25), and annualizing by multiplying by 365, yields the following:

Annual asthma attacks = \[(0.0077/2.11) \times (0.25) \times (365) \times \Delta \text{PM10} = 0.33 \times \Delta \text{PM10}\]

These results show a higher sensitivity than the Whittemore and Korn results, likely because the Ostro et al. (1991) study was undertaken during the winter months in Denver. The more frequent respiratory infections that will occur are likely to render the asthmatics more sensitive to other stimuli, including air pollution.

As a central estimate, the basic regression estimate from Whittemore and Korn (1980) is used, with a lower estimate taken from their results minus one standard error. The high estimate is derived as the average of the central estimates of Ostro et al. (1991) and Whittemore and Korn. This reflects the fact that the Ostro et al. results may be applicable for only the winter months. Therefore, the dose-response estimates for asthma are:

\[ \text{High annual asthma attacks} = 0.195 (\text{PM}_{j}) (\text{POPA}) \]
\[ \text{Central annual asthma attacks} = 0.059 (\text{PM}_{j}) (\text{POPA}) \]
\[ \text{Low annual asthma attacks} = 0.033 (\text{PM}_{j}) (\text{POPA}) \]

where:

\[ \text{POPA} = \text{asthmatic population in location } j \text{ (5 percent of POP)} \]
\[ \text{PM}_{j} = \text{change in annual PM10 in area } j. \]

6. Respiratory Symptoms

The regression coefficient relating COH to respiratory symptoms in Krupnick et al. (1990), Equation 3, is 0.0088, with a standard error of 0.0046. This coefficient is used as the central estimate in the dose-response. The function is applied to both adults and children because although a specification similar to Equation 3 was not estimated for children, other specifications did show an association between COH and symptoms in children. Data provided by the authors show a ratio of COH (units/100 ft²) to TSP for the study period of 0.116. Using the PM10/TSP ratio of 0.55, this gives a COH to PM10 ratio of 0.211. The marginal effect of COH was calculated by incorporating the stationary probabilities as described in the paper. Although not included in the paper, the authors supplied mean estimates of the transitional probabilities for the all-adults sample. For all adults, p, the probability of reporting symptoms — given that symptoms were present on the previous day —

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was 0.7775 and \( p_j \), the probability of reporting symptoms given that no symptoms were present on the previous day, was 0.0468. The daily estimates were annualized by multiplying by 365. The high and low estimates are based on plus or minus one standard error of the regression coefficient and are applied to the entire exposed population.

High annual respiratory symptoms = 0.273 (PM\(_j\)) (POP\(_j\))

Central annual respiratory symptoms = 0.180 (PM\(_j\)) (POP\(_j\))

Low annual respiratory symptoms = 0.091 (PM\(_j\)) (POP\(_j\))

where:

\[ \text{POP}_j = \text{population in location } j \]
\[ \text{PM}_j = \text{change in annual average PM10 in area } j. \]

7. Chronic Bronchitis

Abbey et al. (1993) report a relative risk of 1.36 for a new case of chronic bronchitis for a 60 \( \mu \)g/m\(^3\) increment in the 10-year average exposure of TSP. This risk is based on multiple logistic regression analyses using mean TSP levels for the 10-year period. To calculate the average risk per individual per unit of TSP, the estimated logistic coefficient is needed. The following standard relationship between the logistic coefficient and relative risk is therefore used:

\[ \text{Exp}(b \times \Delta \text{TSP}) = \text{RR} \]

where:

\[ b = \text{the estimated logistic coefficient for mean TSP} \]
\[ \text{TSP} = 10 \text{ year average TSP (} \mu \text{g/m}^3 \text{)} \]
\[ \text{RR} = \text{the relative risk}. \]

Substituting the reported relative risk of 1.36 for a 60 \( \mu \)g/m\(^3\) increment of TSP into the above equation, the estimated logistic coefficient of 0.00512 per \( \mu \)g/m\(^3\) TSP is obtained. The logistic coefficient can be used to calculate the probability of developing chronic bronchitis in 10 years per unit of TSP as follows:

\[ \Delta \text{Pr} / \Delta \text{TSP} = b \times \text{Pr} \times (1 - \text{Pr}) \]

where:

\[ \text{Pr} = \text{probability of an incident case of chronic bronchitis in 10 years}. \]

Substituting the mean incidence of new cases of chronic bronchitis in the study sample (234 new cases out of 3310 subjects) and the estimated logistic coefficient into the above equation gives the predicted probability of new cases in 10 years as a function of the 10-year average TSP level.

New cases/10 years \( \approx 3.36 (\approx 1.71) \times 10^{-4} \times \Delta \text{TSP} \)

The plus and minus figure in parentheses is one standard error around the regression estimate. Standard errors were not reported by the authors, but the relative risk was reported with a p-value < 0.05. The 95 percent confidence interval was therefore estimated for a presumed \( p = 0.05 \), and the resulting confidence intervals were
new cases a year as a function of PM10, the above equation is converted by dividing by the ratio of PM10 to TSP (0.55) and then dividing by 10. This average per year probably overstates the new cases expected in the first few years after a change in PM10 levels occurs, but probably understates the number of new cases after several years of higher exposure. The exact lag between the change in exposure and the elevated risk is not known, but the Abbey et al. results indicate that it is within 10 years. The risk estimates are applied to the adult population age 25 and over since this is the minimum age in the Abbey et al. study group. The resulting dose-response used in this analysis to obtain the number of new cases each year is as follows:

\[
\text{High annual cases of bronchitis} = 9.3 \times 10^5 \times (\text{POPG25}_j \times (\text{PM}_j)
\]

\[
\text{Central annual cases of bronchitis} = 6.1 \times 10^5 \times (\text{POPG25}_j \times (\text{PM}_j)
\]

\[
\text{Low annual cases of bronchitis} = 3.0 \times 10^5 \times (\text{POPG25}_j \times (\text{PM}_j)
\]

where:

\[
\begin{align*}
\text{CB} & = \text{adult chronic bronchitis} \\
\text{POPG25}_j & = \text{population greater than age 25 in area } j \\
\text{PM}_j & = \text{change in annual average PM10 in area } j.
\end{align*}
\]
HEALTH AND ENVIRONMENT ANALYSIS
FOR DECISION-MAKING

The Health and Environment Analysis for Decision-making (HEADLAMP) project is aimed at improving information support for environmental health policies. HEADLAMP makes valid and useful information on the local and national health impacts of environmental hazards available to decision-makers, environmental health professionals and the community. It combines methodologies in environmental epidemiology, human exposure assessment and other health and environment sciences to produce and analyse data, to convert these data into information, and to present this information so that it can be understood, interpreted and acted upon by those responsible for environmental health protection.

Important elements of HEADLAMP are methods for linkage of health and environment data, the use of environmental health indicators used to quantify and monitor the local situation, and the interpretation and translation of resulting information into the decision-making process.

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