Appendix D: Human Health and Welfare Effects of Criteria Pollutants

Introduction

In responding to the mandate of section 812, EPA conducted a comprehensive benefits analysis to identify and estimate the quantifiable health and welfare benefits enjoyed by Americans due to improved air quality resulting from the CAA. Health benefits resulted from avoidance of air pollution-related health effects, such as mortality, respiratory illness, and heart disease. Welfare benefits accrued where improved air quality averted damage to ecological health and measurable resources, such as agricultural production, building materials, and visibility.

This appendix presents an overview of EPA's approach for modeling human health and welfare effects. It provides an outline of the principles used to guide the benefits analysis, details methods used to quantify criteria air pollutant exposure nationwide across the study period (1970 to 1990), and discusses several critical conceptual and implementation issues for using health and welfare effect information. Modeling results, estimates of avoided incidences of adverse health and welfare effects, are then presented. Ecological and agricultural benefits are examined in more detail in Appendices E and F, respectively. Appendix I details the approach used to translate health and welfare effects into monetary benefits.

Principles for the Section 812 Benefits Analysis

Estimating the effects of even modest shifts in environmental releases involves complex chemical, environmental, biological, psychological and economic processes. The task of estimating the broad changes associated with adoption and implementation of the Clean Air Act challenges the limits of scientific knowledge and modeling capability to synthesize available information and techniques into a practical framework. A pragmatic plan for a comprehensive assessment must fairly reflect the complexities and uncertainties, but still produce a policy-relevant analysis in a timely fashion. In order to achieve this ambitious goal, the following principles have been used to guide the section 812 benefits assessment.

Comprehensiveness: The assessment should include as many benefit categories as are reasonably believed to be affected by implementation of the Clean Air Act. Comprehensiveness requires assessing effects with which greater levels of scientific confidence are associated, as well as less well-understood effects. The degree of relative certainty among effects must be carefully described in order to fairly present a broad portrayal of the physical and social benefits accruing to the nation from implementing the Act. In addition, section 812 of the 1990 CAA Amendments explicitly directs a comprehensive benefits coverage that prohibits a default assumption of zero value for identified benefits unless a zero value is supported by specific data.

Quantification Where Feasible: The central goal of the present study is to evaluate and compare the benefits and costs of historical CAA-related programs. Effective comparison of the variety of human health, welfare, and ecological benefits with the associated compliance costs requires that these consequences be measured in terms of a common metric. Expressing the value of these various effects in economic terms is the most efficient way to accomplish this objective, and is consistent with standard practices associated with economic benefit-cost analysis. Expressing these effects in economic terms requires quantifying and presenting estimated effects in both physical and monetized economic terms. Pursuant to this paradigm, the emphasis in the present study is largely on categories having direct and perceptible effects on human health. That is, the emphasis of the analysis is on categories such as symptoms and diseases rather than on physical changes (such as cell level changes) that do not directly result in a decreased health status noticeable to the individual.

Efficient Use of Previous Research Results: Significant research effort has been spent to understand and quantify the complex relationships between air pollution and human health. The present study has relied as much as possible on available research results, making adjustments as necessary to apply the existing results to the current analysis.

Incorporate Uncertainty: To properly convey the results of any benefits assessment, it is important to include an evaluation and characterization of how much confidence the analysts have in the estimates. Ideally this would include a formal quantitative assessment of the potential for error, and the sources, directions, and potential significance of any resultant biases. A method for considering and reporting uncertainty must be built into the fundamental design of the assessment. Such a framework was developed and applied in the present study, and was supplemented where necessary by expert judgment regarding the sources and potential significance of errors in each analytical step.

General Modeling Approach

Consistent with these principles, the EPA developed an approach for quantifying the effects of reduced pollutant exposure, with particular focus on those effect categories for which monetary benefits could be estimated. As described previously, the study design adopted for the section 812 assessment links a sequence of analytical models. The macroeconomic modeling (Appendix A) estimated economy-wide effects of CAA expenditures. These effects provided a basis for the modeling of criteria pollutant emissions under the two scenarios considered (the factual control scenario and the hypothetical no-control scenario), as documented in Appendix B. The emissions estimates were used as input to the air quality models (Appendix C). Ambient pollutant concentrations estimated by the air quality models were used as inputs to the health and welfare benefits model, the focus of this appendix.

The approach developed to model health and welfare benefits is known as a "reduced form" or "embedded model" approach. The concept of a reduced form model is to use simplified versions of previously constructed complex models to characterize the impact of a series of linked physical and socioeconomic processes. The health and welfare benefits model is characterized as a reduced form model because it relies on *summaries* of the data output from the air quality models, which rely on emissions summaries and summaries of macroeconomic conditions, successively. Although results of the independent models are used in series, the models themselves have not been integrated into the health and welfare benefits model.

In general, the reduced form health and welfare benefits model relies on two fundamental inputs: (1) nationwide changes in pollutant exposures across the study period, and (2) the association between changes in exposure and expected changes in specific health and welfare effects. These inputs are discussed below.

Quantifying Changes in Pollutant Exposures

Estimating changes in pollutant exposures requires characterization of nationwide air quality improvements across the study period, as well as the populations exposed to the different levels of improvement.

Air Quality

As discussed in Appendix C, the section 812 analysis estimated ambient concentrations for both the control and no-control scenarios for the following pollutants and air quality parameters:

- Particulate matter, less than 10 microns in diameter (PM_{10})
- Ozone (O_3)
- Nitrogen dioxide (NO_2)
- Sulfur dioxide (SO_2)
- Carbon monoxide (CO)
- Visibility measures (light extinction and DeciView)[1]
- Lead (Pb)

Generally, this analysis adopted actual historical air pollution monitoring data to represent control scenario air quality. No-control scenario profiles were

[1] While the visibility measures listed are not criteria air pollutants, they provide important measures of a significant welfare effect resulting from air pollution, visibility degradation. Light extinction (which is related to DeciView, a haziness index) results from light scattered by fine particles in the atmosphere, especially sulfates and ammonium nitrates. As atmospheric concentrations of such particles increase, light is attenuated and visibility diminishes.

derived by running the control and no-control scenario emissions inventories through a suite of air quality models and then using the differences in these modeled outcomes to adjust the historical profiles. Since lead was treated differently than the other pollutants, the analysis of the CAA impacts on atmospheric lead concentrations is documented in Appendix G.

With respect to the distribution of air quality data across the two decades considered, it should be noted that both the number and location of monitors tracking air quality changed over time. Table D-1 depicts the number of monitors for each pollutant across the period of this analysis. The number of monitors generally increased throughout the 1970s and leveled off or declined at varying points during the 1980s, depending on the pollutant.

Table D-1. Criteria Air Pollutant Monitors in the U.S., 1970 - 1990.

	Pollutant				
Year	PM_{10}	O_3	NO_2	SO_2	CO
1970	245	1	43	86	82
1975	1,120	321	303	827	494
1980	1,131	546	375	1,088	511
1985	970	527	305	916	458
1990	720	627	345	753	493

For the section 812 modeling, the non-lead pollutants have been characterized as either county-level or monitor-level pollutants. The distinction was important for quantifying the population exposed to different levels of air quality improvements, as discussed below. PM_{10} is considered a county-level pollutant, since historical concentrations in monitored counties have been synthesized into a single concentration for each county.[2] In contrast, O_3, NO_2, NO, SO_2, and CO were reported at specific monitor locations, given by latitude/longitude coordinates. Finally, visibility was treated as a county-level pollutant in the western U.S. and a monitor-level pollutant in the eastern U.S.[3] Air quality data for PM_{10} and ozone were reported for each year of the study period; data for the remaining pollutants were reported only for 1975, 1980, 1985, and 1990.

In order to reduce the volume of air quality data necessary to describe pollutant concentrations for two scenarios nationwide over twenty years, annual concentration profiles were reduced to frequency distributions. That is, annual pollutant concentrations for a variety of averaging times (e.g., 1-hour, 6-hour, daily) were summarized as a distribution of values across the year. This approach reduced data management requirements significantly, while adequately capturing air quality improvements between the control and no-control scenarios.

Population Distribution

Health and some welfare benefits resulting from air quality improvements are distributed to populations in proportion to the reduction in exposure each enjoys. Predicting population exposures, then, is a necessary step in estimating health effects. Doing so for the section 812 analysis required not only an understanding of where air quality improved as a result of the CAA, but also how many individuals were affected by varying levels of air quality improvements. Thus, a critical component of the benefits analysis required that the distribution of the U.S. population nationwide be described in a manner compatible with the air quality data. Described below is the method used to allocate U.S. Census data to a symmetrical grid overlying the country.

Census Data

Three years of U.S. Census data were used to represent the geographical distribution of U.S. residents: 1970, 1980, and 1990. Population data were supplied at the census block group level, with approximately

[2] Two different measures of ambient concentrations of particulate matter were used in the United States during the period 1970 to 1990. Prior to 1987, the indicator for the National Ambient Air Quality Standard for PM was total suspended particulates (TSP). In 1987, the indicator was changed to PM_{10} (particles less than 10 μM in diameter). Widespread PM_{10} monitoring did not begin until 1985; prior to that only TSP data is available. Because the recent scientific literature reports primarily the relationship between PM_{10} and adverse health and welfare effects, PM_{10} data is preferred, if available. Where only TSP is available, PM_{10} concentrations were estimated using PM_{10}:TSP ratios that vary by area of the country and the urban/rural characterization of the area.

[3] In the western U.S., visibility was modeled using a linear-rollback model and extinction budget approach for 30 major urban centers (SAI, 1994). The modeling results, reported in DeciView, were applied to the counties in the vicinity of the urban centers and considered to share a common air basin. In the eastern U.S., Regional Acid Deposition Model (RADM) runs provided visibility estimates in terms of light extinction coefficients. These were modeled across a 60 km. X 60 km. grid, approximately covering the eastern half of the country. Since the extinction coefficients were reported at the grid cell centroids, for which the coordinates were known, visibility in the east was treated as a monitor-level pollutant.

290,000 block groups nationwide. Allocating air quality improvements to the population during intermediate years necessitated interpolation of the three years of population data. Linear interpolation was performed at the block group level in order to preserve the variability in growth rates throughout the country.

Gridding U.S. Population

To ease computational burden, block group population estimates were aggregated to a rectangular grid structure. The grid, comprised of ten kilometer by ten kilometer gridcells, spanned the entire area of the contentional United States. This grid size generated 46,885 populated gridcells throughout the U.S.

The entire population of each block group was assumed to reside at the geographical centroid of the block group area, the coordinates of which were available from the U.S. Bureau of the Census. Block group populations were aggregated to gridcells according to the block group centroids encompassed by each cell. In addition to the population of each gridcell, the state and county names for each gridcell were retained, permitting aggregation of data at the state and county level, as well as nationwide.

Allocating Exposure Estimates to the Population

Two alternative modeling strategies were used to allocate air quality improvements to the U.S. population. They differed in terms of both the certainty of the estimates and the geographic coverage:

Table D-2. Population Coverage in the "Within 50 km" Model Runs (percent of continental U.S. population).

	1975	1980	1985	1990
CO	67.4%	67.9%	68.4%	70.4%
EXT	73.2%	72.3%	72.3%	72.2%
NO_2	53.3%	58.8%	60.8%	61.5%
O_3	55.5%	70.5%	71.5%	74.4%
PM_{10}	78.5%	79.5%	75.8%	67.8%
SO_2	64.7%	73.3%	73.0%	70.6%
Pb	100%	100%	100%	100%

Method One

Air quality improvements (difference between control and no-control scenarios) were applied to individuals living in the vicinity of air quality monitors. For pollutants with monitor-level data, it was assumed that the individuals in a gridcell were exposed to air quality changes estimated at the nearest monitor, as long as the monitor was within 50 kilometers. Likewise, for PM_{10} (for which data was available at the county level) the population of each monitored county was assumed to be exposed to the air quality changes reported for that county.[4] The remainder of the population was excluded from the analysis.

Unfortunately, by limiting the quantitative analysis to populations within 50 km of a monitor (or within a monitored county, for PM), a significant portion of the U.S. population was left out of the analysis (see Table D-2). For most pollutants in most years (excepting lead), less than three-quarters of the population lived within 50 km of a monitor (or within a PM-monitored county). Clearly, an analysis that excluded 25 percent of the population from the benefits calculations (thus implicitly assuming that the CAA had no impact on that population) would understate the physical effects of the CAA. Conversely, ascribing air pollution reduction benefits to persons living great distances from air quality monitors is a speculative exercise, and could overstate benefits.

Method Two

As an alternative modeling strategy, air quality improvements were applied to almost all individuals nationwide. Where monitor data were not available within 50 kilometers, data from the closest monitor, regardless of distance, were used. Similarly, PM_{10} concentrations were extrapolated using regional air quality models to all counties (even those for which monitoring data was unavailable) and applied to the populations of those counties.

Although subject to less certain air quality data, the second alternative extrapolates pollutant exposure estimates to almost the entire population using the closest monitoring data available (see Table D-3).[5] This second alternative was chosen as the preferred approach in the benefits analysis. The sensitivity of

[4] Since the lead (Pb) analysis, which was handled separately from that of the other criteria pollutants, did not require air quality modeling data, the issue of proximity to monitors is irrelevant. The Pb analysis extended to 100 percent of the population.

[5] While this alternative captures the vast majority of the U.S. population, it does not model exposure for everyone. To improve computational efficiency, those gridcells with populations less than 1,000 were not modeled; these cells account for less than five percent of the U.S. population.

the benefits estimate to the extrapolation of air quality data beyond monitored areas is explored in Appendix I.

Table D-3. Population Coverage for "Extrapolated to All U.S." Model Runs (percent of continental U.S. population).

	1975	1980	1985	1990
CO	97.2%	97.2%	98.7%	100.0%
EXT	75.6%	74.8%	74.7%	74.7%
NO_2	97.2%	97.2%	98.7%	100.0%
O_3	96.6%	97.2%	98.7%	100.0%
PM_{10}	95.9%	95.8%	97.2%	98.5%
SO_2	95.4%	95.6%	97.0%	98.4%
Pb	100%	100%	100%	100%

Estimating Human Health Effects of Exposure

It is impossible to estimate all of the physical effects that would have occurred without the Clean Air Act. While scientific information is available that makes it possible to estimate certain effects, many other, potentially very important, health and welfare effects cannot be estimated at this time. Other physical effects can be quantified, but it is impossible to assess the economic value of those endpoints based on the current economics literature. Table D-4 shows the health and welfare effects for which quantitative analysis has been prepared, as well as some of the health effects that have not been quantified in the analysis.

In order to translate the reductions in pollutant exposure estimated to result from the CAA into health benefits, it is necessary to quantify the relationship between such exposures and adverse health effects. As indicated below, this analysis relies on concentration-response relationships published in the scientific literature which provide estimates of the number of fewer individuals that incur an adverse health effect per unit change in air quality. Such relationships are combined with the air quality improvement and population distribution data to estimate changes in the incidence of each health endpoint. By evaluating each concentration-response function for every gridcell throughout the country, and aggregating the resulting incidence estimates, it was possible to generate national estimates of avoided incidence.

It should be noted that a slightly different approach was used to compute health effects associated with exposure to gasoline lead. Instead of relating health outcomes to ambient pollutant concentrations, the concentration-response functions for lead-induced effects link changes in health effects directly to changes in the population's mean blood lead level. This value is directly related to the concentration of lead in gasoline in a particular year. Appendix G documents both the methods used to characterize mean blood lead levels and the approach for estimating human health effects from lead exposure.

The discussion below outlines the types of health studies considered for this analysis, and issues critical to selecting specific studies appropriate for use in the section 812 context. Next, details regarding use of the results of the studies are explored. Finally, the concentration-response functions used to model health benefits from reductions in non-lead criteria pollutants are outlined.

Types of Health Studies

Scientific research about air pollution's adverse health impacts uses a broad array of methods and procedures. The research methods used to investigate the health effects of air pollution have become considerably more sophisticated over time, and will continue to evolve in the future. This progress is the result of better available research techniques and data, and the ability to focus further research more sharply on key remaining issues based on the contributions of earlier work.

The available health effects studies that could potentially be used as the basis of the section 812 assessment are categorized into epidemiology studies and human clinical studies. Epidemiological research in air pollution investigates the association between exposure to air pollution and observed health effects in the study population. Human clinical studies involve examination of human responses to controlled conditions in a laboratory setting. Research has been conducted on health effects from exposure to pollution using each approach, and studies using these techniques have been considered in various formal regulatory proceedings. Each type of study (as it is used

Table D-4. Human Health Effects of Criteria Pollutants.

Pollutant	Quantified Health Effects	Unquantified Health Effects	Other Possible Effects
Ozone	Mortality* Respiratory symptoms Minor restricted activity days Respiratory restricted activity days Hospital admissions Asthma attacks Changes in pulmonary function Chronic Sinusitis & Hay Fever	Increased airway responsiveness to stimuli Centroacinar fibrosis Inflammation in the lung	Immunologic changes Chronic respiratory diseases Extrapulmonary effects (e.g., changes in structure, function of other organs)
Particulate Matter/ TSP/ Sulfates	Mortality* Bronchitis - Chronic and Acute Hospital admissions Lower respiratory illness Upper respiratory illness Chest illness Respiratory symptoms Minor restricted activity days All restricted activity days Days of work loss Moderate or worse asthma status (asthmatics)	Changes in pulmonary function	Chronic respiratory diseases other than chronic bronchitis Inflammation in the lung
Carbon Monoxide	Hospital Admissions - congestive heart failure Decreased time to onset of angina	Behavioral effects Other hospital admissions	Other cardiovascular effects Developmental effects
Nitrogen Oxides	Respiratory illness	Increased airway responsiveness	Decreased pulmonary function Inflammation in the lung Immunological changes
Sulfur Dioxide	In exercising asthmatics: Changes in pulmonary function Respiratory symptoms Combined responses of respiratory symptoms and pulmonary function changes		Respiratory symptoms in non-asthmatics Hospital admissions
Lead	Mortality Hypertension Non-fatal coronary heart disease Non-fatal strokes IQ loss effect on lifetime earnings IQ loss effects on special education needs	Health effects for individuals in age ranges other than those studied Neurobehavioral function Other cardiovascular diseases Reproductive effects Fetal effects from maternal exposure Delinquent and anti-social behavior in children	

* This analysis estimates excess mortality using PM_{10} as an indicator of the pollutant mix to which individuals were exposed.

for air pollution research) is described below, and the relative strengths and weaknesses for the purposes of the section 812 assessment are examined.

Epidemiological Studies

Epidemiological studies evaluate the relationship between exposures to ambient air pollution and health effects in the human population, typically in a "natural" setting. Statistical techniques (typically variants of multivariate regression analysis) are used to estimate quantitative concentration-response (or exposure-response) relationships between pollution levels and health effects.

Epidemiology studies can examine many of the types of health effects that are difficult to study using a clinical approach. Epidemiological results are well-suited for quantitative benefit analyses because they provide a means to estimate the incidence of health effects related to varying levels of ambient air pollution without extensive further modeling effort. These estimated relationships implicitly take into account at least some of the complex real-world human activity patterns, spatial and temporal distributions of air pollution, synergistic effects of multiple pollutants and other risk factors, and compensating or mitigating behavior by the subject population. Suspected relationships between air pollution and the effects of both

long-term and short-term exposure can be investigated using an epidemiological approach. In addition, observable health endpoints are measured, unlike clinical studies which often monitor endpoints that do not result in observable health effects (e.g. forced expiratory volume). Thus, from the point of view of conducting a benefits analysis, the results of epidemiological studies, combined with measures of ambient pollution levels and the size of the relevant population, provide all the essential components for associating measures of ambient air pollution and health status for a population in the airshed being monitored.

Two types of epidemiological studies are considered for dose-response modeling: individual level cohort studies and population level ecological studies. Cohort-based studies track individuals that are initially disease-free over a certain period of time, with periodic evaluation of the individuals' health status. Studies about relatively rare events such as cancer incidence or mortality can require tracking the individuals over a long period of time, while more common events (e.g., respiratory symptoms) occur with sufficient frequency to evaluate the relationship over a much shorter time period. An important feature of cohort studies is that information is known about each individual, including other potential variables correlated to disease state. These variables, called confounders, are important to identify because if they are not accounted for in the study they may produce a spurious association between air pollution and health effect.

A second type of study used in this analysis is a population-level ecological study. The relationship between population-wide health information (such as counts for daily mortality, hospital admissions, or emergency room visits) and ambient levels of air pollution are evaluated. One particular type of ecological study, time-series, has been used frequently in air-pollution research. An advantage of the time-series design is that it allows "the population to serve as its own control" with regard to certain factors such as race and gender. Other factors that change over time (tobacco, alcohol and illicit drug use, access to health care, employment, and nutrition) can also affect health. However, since such potential confounding factors are unlikely to vary over time in the same manner as air pollution levels, or to vary over periods of months to several years in a given community, these factors are unlikely to affect the magnitude of the association between air pollution and variations in short-term human health responses.

Drawbacks to epidemiological methods include difficulties associated with adequately characterizing exposure, measurement errors in the explanatory variables, the influence of unmeasured variables, and correlations between the pollution variables of concern and both the included and omitted variables. These can potentially lead to spurious conclusions. However, epidemiological studies involve a large number of people and do not suffer extrapolation problems common to clinical studies of limited numbers of people from selected population subgroups.

Human Clinical Studies

Clinical studies of air pollution involve exposing human subjects to various levels of air pollution in a carefully controlled and monitored laboratory situation. The physical condition of the subjects is measured before, during and after the pollution exposure. Physical condition measurements can include general biomedical information (e.g., pulse rate and blood pressure), physiological effects specifically affected by the pollutant (e.g., lung function), the onset of symptoms (e.g., wheezing or chest pain), or the ability of the individual to perform specific physical or cognitive tasks (e.g., maximum sustainable speed on a treadmill). These studies often involve exposing the individuals to pollutants while exercising, increasing the amount of pollutants that are actually introduced into the lungs.

Clinical studies can isolate cause-effect relationships between pollutants and certain human health effects. Repeated experiments altering the pollutant level, exercise regime duration and types of participants can potentially identify effect thresholds, the impact of recovery (rest) periods, and the differences in response among population groups. While cost considerations tend to limit the number of participants and experimental variants examined in a single study, clinical studies can follow rigorous laboratory scientific protocols, such as the use of placebos (clean air) to establish a baseline level of effects and precise measurement of certain health effects of concern.

There are drawbacks to using clinical studies as the basis for a comprehensive benefits analysis. Clinical studies are appropriate for examining acute symptoms caused by short-term exposure to a pollutant. While this permits examination of some important health effects from air pollution, such as bronchoconstriction in asthmatic individuals caused by sulfur dioxide, it excludes studying more severe

effects or effects caused by long term exposure. Another drawback is that health effects measured in some well-designed clinical studies are selected on the basis of the ability to measure precisely the effect, for example forced expiratory volume, rather than a larger symptom. The impact of some clinically measurable but reversible health effects such as lung function on future medical condition or lifestyle changes are not well understood.

Ethical limits on experiments involving humans also impose important limits to the potential scope of clinical research. Chronic effects cannot be investigated because people cannot be kept in controlled conditions for an extended period of time, and because these effects are generally irreversible. Participation is generally restricted to healthy subjects, or at least to exclude people with substantial health conditions that compromise their safe inclusion in the study. This can cause clinical studies to avoid providing direct evidence about populations of most concern, such as people who already have serious respiratory diseases. Ethical considerations also limit the exposures to relatively modest exposure levels, and to examining only mild health effects that do no permanent damage. Obviously for ethical reasons human clinical evidence cannot be obtained on the possible relationship between pollution and mortality, heart attack or stroke, or cancer.

One potential obstacle to using dose-response information from clinical research methods in a benefits assessment is the need for an exposure model. The dose-response functions developed from clinical research are specific to the population participating in the study and the exposure conditions used in the laboratory setting. It is therefore difficult to extrapolate results from clinical settings to daily exposures faced by the whole population. For example, many clinical studies evaluate effects on exercising individuals. Only a small portion of the population engages in strenuous activity (manual labor or exercise) at any time. Reflecting these fundamental differences between the laboratory setting and the "real world" imposes a formidable burden on researchers to provide information about human activity patterns, exercise levels, and pollution levels. This requirement adds an additional step in the analytical process, introducing another source of uncertainty and possible error.

To apply the clinical results to model the general population, two decisions must be made. First, how far can the conditions in the clinical setting be expanded? For example, if the subjects in the clinical study were healthy male college students, should the results be applied to the entire population, including children? Second, how many people in the general population are exposed to conditions similar to those used in the clinical setting? Frequently, clinical studies are conducted at relatively high exercise levels (increasing the dose, or the quantity of pollutants actually delivered to the lungs). In the general population few people experience these conditions very often, and people do not reach these exercise levels with equal frequencies during the day and night.

In addition, the analyst must determine the number of people that are exposed to the levels of ambient conditions seen in the laboratory. Air quality varies throughout a city and is typically reported by data from monitors located at various places throughout the city. However, people are not exposed to the conditions at any one monitor all day. As people move around in the city, they are exposed to ambient air quality conditions represented by different monitors at different times during the day. To further compound the problem, air quality also varies between indoors and outdoors, within a car or garage, and by such factors as proximity to a roadway or major pollution source (or sink). The exposure model must account for the ambient conditions in the "microenvironments" that the population actually experiences.

The issues of study subjects, exercise and microenvironments can influence the choice of clinical studies selected for the section 812 assessment. Clinical studies that use exposure regimes and exercise levels more similar to what larger groups of the population see are easier to apply in a benefits model than are more narrow studies. Similarly, studies that use a diverse group of subjects are easier to apply to the general population than are more narrow studies.

Given the major advantages of epidemiological studies—exposures do not need to be modeled and health effects are observed in a large, more heterogeneous population—epidemiological studies are used as the basis for determining the majority of health effects and dose-response curves. The diverse activity patterns, microenvironments, and pollution levels are already considered in the aggregate through the concentration-response functions derived from epidemiological studies. Clinical studies are used if there are health effects observed in clinical studies not observed in epidemiological studies.

Issues in Selecting Studies To Estimate Health Effects

A number of issues arise when selecting and linking the individual components of a comprehensive benefits analysis. The appropriate procedure for handling each issue must be decided within the context of the current analytical needs, considering the broader analytical framework. While more sophisticated or robust studies may be available in some circumstances, the potential impact on the overall analysis may make using a simpler, more tractable approach the pragmatic choice. In considering the overall impact of selecting a study for use in the section 812 assessment, important factors to consider include the likely magnitude the decision will have on the overall analysis, the balance between the overall level of analytical rigor and comprehensiveness in separate pieces of the analysis, and the effect on the scientific defensibility of the overall project.

This section discusses ten critical issues in selecting health information for use in the section 812 assessment: use of peer-reviewed research, confounding factors, uncertainty, the magnitude of exposure, duration of exposure, threshold concentrations, the target population, statistical significance of relationships, relative risks, and the need for baseline incidence data. The previous discussion about the types of research methods available for the health information alluded to some of these issues, as they are potentially important factors in selecting between studies using different methods. Other issues address how scientific research is used in the overall analytical framework.

Peer-Review of Research

Whenever possible, peer reviewed research rather than unpublished information has been relied upon. Research that has been reviewed by the EPA's own peer review processes, such as review by the Clean Air Science Advisory Committee (CASAC) of the Science Advisory Board (SAB), has been used whenever possible. Research reviewed by other public scientific peer review processes such as the National Academy of Science, the National Acidic Precipitation Assessment Program, and the Health Effects Institute is also included in this category.

Research published in peer reviewed journals but not reviewed by CASAC has also been considered for use in the section 812 assessment, and has been used if it is determined to be the most appropriate available study. Research accepted for publication by peer reviewed journals ("in press") has been considered to have been published. Indications that EPA intends to submit research to the CASAC (such as inclusion in a draft Criteria Document or Staff Paper) provide further evidence that the journal-published research should be used.

Air pollution health research is a very active field of scientific inquiry, and new results are being produced constantly. Many research findings are first released in University Working Papers, dissertations, government reports, non-reviewed journals and conference proceedings. Some research is published in abstract form in journals, which does not require peer review. In order to use the most recent research findings and be as comprehensive as possible, unpublished research was examined for possible use in the section 812 assessment. Any unpublished research used is carefully identified in the report, and treated as having a higher degree of uncertainty than published results. The peer review of the section 812 assessment by the Advisory Council on Clean Air Compliance Analysis provides one review process for all components of the assessment, as well as for the way in which the components have been used.

Confounding Factors

Confounding can occur when the real cause of disease is associated with a number of factors. If only one contributing factor is evaluated in an epidemiological study, a false association may occur. For example, in epidemiology studies of air pollution, it is important to take into account weather conditions, because weather is associated with both air pollution and health outcomes. If only air pollution is evaluated, a false association between air pollution and health could result; one may incorrectly assume that a reduction in air pollution is exclusively responsible for a reduction in a health outcome. Potential confounders include weather-related variables, age and gender mix of the subject population, and pollution emissions other than those being studied. Studies that control for a broad range of likely confounders can offer a more robust conclusion about an individual pollutant, even if the statistical confidence interval is larger due to the inclusion of more variables in the analysis.

In many cases, several pollutants in a "pollutant mix" are correlated with each other—that is, they tend to occur simultaneously. Therefore, although there may be an association between a health effect and each of several pollutants in the mix, it may not be clear which pollutant is causally related to the health effect (or whether more than one pollutant is causally related). This analysis includes epidemiological modeling of the health effects that have been associated with exposure to a number of pollutants. In most cases where the health effect is being modeled for the several correlated pollutants of interest, regression coefficients based on PM as a surrogate for the mixture were chosen in preference to multiple pollutant models and single pollutant models. The most important example of this occurs in estimating mortality effects. There is substantial evidence that exposure to criteria pollutants, either individually or collectively, is significantly associated with excess mortality. Generally, this association is related to particulate matter. Therefore, even though particulate matter cannot be shown to be the sole pollutant causing pollution-related excess mortality, it can be used as an indicator of the pollutant mixture which appears to result in excess mortality. This analysis estimates excess mortality (for all criteria pollutants other than lead) using PM as an indicator of the pollutant mix to which individuals were exposed. This issue is discussed further below, where details on estimating mortality effects are explored.

The one exception to the use of single pollutant regression models is estimating hospital admissions. Both PM and ozone are generally found to have a statistically significant and separate association with hospital admissions. Using separate regressions (from single pollutant models) for each pollutant may overstate the number of effects caused by each pollutant alone. On the other hand, using PM as a single indicator of the pollutant mix could underestimate the total hospital admissions caused by different mechanisms. Separate PM and ozone coefficients for hospital admissions are selected from regression models that consider the effects of both pollutants simultaneously.

Uncertainty

The stated goal of the section 812 assessment is to provide a comprehensive estimate of benefits of the Clean Air Act. To achieve this goal, information with very different levels of confidence must be used. Benefit categories are not to be omitted simply because they are highly uncertain or controversial, but those benefit categories that are reasonably well understood must be distinguished from those which are more tentative.

The ideal approach to characterizing uncertainty is to conduct a formal quantitative uncertainty analysis. A common approach develops an estimated probability distribution for each component of the analysis. A Monte Carlo procedure draws randomly from each of these distributions to generate an estimate of the result. Evaluating the result for many such random combinations, creates a distribution of results that reflects the joint uncertainties in the analysis.

The most serious obstacle to preparing a formal quantitative uncertainty analysis is identifying all the necessary distributions for each component of the analysis. The Monte Carlo procedure requires that all components of the model be rerun many times. However, the section 812 project links the outputs from independent modeling activities. It would be impractical to simultaneously rerun the macroeconomic, emissions, air quality, and exposure models because of the diverse origins of the models. Therefore, instead of a complete formal uncertainty analysis, the section 812 assessment includes a less rigorous analysis of the inherent uncertainties in the modeling effort. The uncertainty analysis combines quantitative and qualitative elements designed to sufficiently describe the implications of the uncertainties. A primary goal of the sensitivity/uncertainty analysis is to identify the health effects that make a sizable contribution to the overall assessment of the monetary benefits. There may be situations where there are significant differences in the available information used to predict the incidence of a particular health effect (i.e., the uncertainty bounds are large). It is important to alert the reader to situations where using the lower incidence estimates may portray the health effect as only modestly contributing to the overall total benefits, but using reasonable alternative higher estimated incidence figures (or higher monetized values) would substantially impact not only the monetized value of the individual health effect, but actually make a noticeable difference in the total benefits assessment.

Consideration of the overall uncertainties inherent in the section 812 assessment has several important implications for health study selection. It was important to carefully examine the balance between the level of uncertainties in the analysis and the need for

comprehensive coverage of all benefit categories. There were frequently situations in which a direct tradeoff existed between more comprehensive coverage and the restriction of the analysis to more certain information. Also, the relationship between the uncertainty in other parts of the analysis and the uncertainty for each particular health effect was carefully considered.

Magnitude of Exposure

One component of the section 812 analysis estimates the air pollution levels that would have occurred in the absence of the Clean Air Act. These estimates are larger than currently observed levels of U.S. air pollution, and perhaps even levels currently observed elsewhere in the world. This aspect of the analysis poses difficulties for the application of concentration-response functions that have been based on exposures at much lower pollution levels. The shape of the concentration-response function much above observed exposures levels is unknown. It is possible that biological mechanisms affecting response that are unimportant at low levels of exposure may dominate the form of response at higher levels, introducing nonlinearity to the mathematical relationship. In general, studies that include exposure levels spanning the range of interest in the section 812 assessment are preferable to studies at levels outside of the range, or that only include a narrow part of the range. A possible drawback to this approach is that studies which fit this criterion have often been conducted outside the U.S. The application of foreign studies to U.S. populations introduces additional uncertainties regarding the representativeness of the exposed population and the relative composition of the air pollution mix for which the single pollutant is an indicator. These difficult issues were considered in selecting studies for the benefits analysis.

Duration of Exposure

Selection of health studies for the section 812 assessment must consider the need to match the health information to the air quality modeling conducted for the assessment. For example, information on the health effects from short term (five minute) exposure to sulfur dioxide cannot be readily combined with information on average daily sulfur dioxide levels. In selecting studies for the benefits analysis, preference was shown for studies whose duration of exposure matched one of the averaging times of the air quality data.

Thresholds

Exposure-response relationships are conceptualized as either exhibiting a threshold of exposure below which adverse effects are not expected to occur, or as having no response threshold, where any exposure level theoretically poses a non-zero risk of response to at least one segment of the population. The methods employed by health researchers to characterize exposure-response relationships may or may not explicitly analyze the data for the existence of a threshold. Studies may analyze relationships between health and air pollution without considering a threshold. If a threshold for population risk exists but is not identified by researchers, then Clean Air Act benefits could be overestimated if CAA levels are below the threshold, because the risk reduction from the no-control scenario could be overstated. On the other hand, if a threshold is artificially imposed where one does not exist, the relative benefits of the Clean Air Act may be underestimated. In general, those studies that explicitly consider the question of a threshold (whether a threshold is identified or not) provide stronger evidence; consideration of this question is a positive feature when selecting studies for this analysis.

Target Population

Many of the studies relevant to quantifying the benefits of air pollution reductions have focused on specific sensitive subpopulations suspected to be most susceptible to the effects of the pollutant. Some of these effects may be relevant only for the studied subpopulation; effects on other individuals are either unknown, or not expected to occur. For such studies, the challenge of the analysis is to identify the size and characteristics of the subpopulation and match its occurrence to exposure. Other studies have examined specific cohorts who may be less susceptible than the general population to health effects from air pollution (e.g., healthy workers), or who differ in age, gender, race, ethnicity or other relevant characteristics from the target population of the benefits analysis. Extrapolating results from studies on nonrepresentative subpopulations to the general population introduces uncertainties to the analysis, but the magnitude of the uncertainty and its direction are often unknown. Because of these uncertainties, benefit analyses often limit the application of the dose-response functions only to those subpopulations with the characteristics of the study population. While this approach has merit in minimizing uncertainty in the analysis, it can also

severely underestimate benefits if, in fact, similar effects are likely to occur in other populations. For these reasons, studies that examine broad, representative populations are preferable to studies with narrower scope because they allow application of the functions to larger numbers of persons without introducing additional uncertainty.

Many studies included in the section 812 analysis focus on a particular age cohort of the population for the identification of health effects. The choice of age group is often a matter of convenience (e.g., extensive Medicare data may be available for the elderly population) and not because the effects are, in reality, restricted to the specific age group (even though their incidence may vary considerably over the life span). However, since no information is available about effects beyond the studied population, this analysis applies the given concentration-response relationships only to those age groups corresponding to the cohorts studied. Likewise, some studies were performed on individuals with specific occupations, activity patterns, or medical conditions because these traits relate to the likelihood of effect. In these cases, application of dose-response functions has been restricted to populations of individuals with these same characteristics.

Statistical Significance of Exposure-Response Relationships

The analysis includes as many studies related to a given health effect as possible, except for studies inapplicable to the current analysis. For some endpoints, the group of adequate studies yielded mixed results, with some showing statistically significant responses to pollutant concentrations and others with insignificant associations. Unless study methods have been judged inadequate, dose-response functions with both statistically significant and insignificant coefficients have been included to characterize the possible range of risk estimates. Excluding studies exclusively on the basis of significance could create an upward bias in the estimates by not reflecting research that indicates there is a small, or even zero, relationship between pollution and specific health effects. It should be noted, however, that some studies that found insignificant effects for a pollutant could not be used because they did not report the insignificant coefficient values.

In some cases, a single study reported results for multiple analyses, yielding both significant and nonsignificant results, depending on the nature of the input parameters (e.g., for different lag periods or concurrent exposures). In these cases, only significant results were included.

Relative Risks

Many studies reported only a relative risk value (defined as the ratio of the incidence of disease in two groups exposed to two different exposure levels). The analysis required conversion of these values to their corresponding regression coefficients when the coefficients were not reported. When converting the relative risk to a coefficient value, the analysis used the functional form of the regression equation reported by the authors of the study.

The coefficients from a number of studies measured the change in the number of health effects for the study population rather than a change per individual. These coefficients were divided by the size of the study population to obtain an estimate of change per individual. The coefficient could then be multiplied by the size of the population modeled in the current analysis to determine total incidence of health effects.

Baseline Incidence Data

Certain dose-response functions (those expressed as a change relative to baseline conditions) require baseline incidence data associated with ambient levels of pollutants. Incidence data necessary for the calculation of risk and benefits were obtained from national sources whenever possible, because these data are most applicable to a national assessment of benefits. The National Center for Health Statistics provided much of the information on national incidence rates. However, for some studies, the only available incidence information come from the studies themselves; in these cases, incidence in the study population is assumed to represent typical incidence nationally.

Studies were excluded if health endpoints could not be defined in the U.S. population. For example, in Pope and Dockery (1992) the authors developed a unique definition of symptomatic children in Utah which has no correlation in the incidence data bases which were available; consequently, the results could not be applied to the general population.

Estimating Mortality Effects

Using PM as an Indicator

There is substantial evidence that exposure to criteria pollutants, either individually or collectively, is significantly associated with excess mortality. This association is most closely and consistently related to the ambient air concentrations of PM.

Several studies have found small but statistically significant relationships between ozone and mortality, while other studies have not found a significant relationship. There is inconclusive evidence whether ozone has an effect independent of the effect of other pollutants (e.g., PM or CO), has a synergistic effect in combination with other effects, or is a confounder in the relationship between mortality and other pollutants. For example, in a recent study HEI (1996) found a significant and relatively stable ozone coefficient for most of the model specifications presented in the study. However, the measured ozone effect was largest and most significant in the winter and autumn, when ozone levels are low.

This analysis estimates excess mortality (for all criteria pollutants other than lead) using PM as an indicator of the pollutant mix to which individuals were exposed. Even if particulate matter exposure cannot be shown to be an independent causal factor of excess mortality, it is, at a minimum, a good indicator measure of the exposure to the pollutant mixture that has been shown to be related to excess mortality. Because PM is used as an indicator, the concentration-response functions from single pollutant models (i.e., statistical models including PM as the only pollutant) are preferred. To the extent that ozone is correlated with PM, the effect of ozone, either as an independent association or acting in combination with other pollutants, will be captured by this approach.

Estimating the Relationship Between PM and Premature Mortality

Long-term exposure versus short-term exposure studies and the degree of prematurity of mortality. Both long-term exposure (cohort) studies and short-term exposure (longitudinal or time-series) studies have estimated the relationship between exposure to PM and premature mortality. While there are advantages and disadvantages to each type of study (as discussed above), the long-term studies may capture more of the PM-related premature mortality, as well as premature mortality that is more premature, than the short-term studies.

The degree of prematurity of pollution-related death may be an important uncertainty in the effort to estimate the benefits of reducing pollution concentrations, as discussed in Appendix I. The willingness to pay to save a few days of life may be significantly less than the willingness to pay to save a few, or many, years of life. Evidence concerning the degree of prematurity of pollution-related death would, in this case, be crucial. Such evidence is, however, still scarce. There is some limited evidence that the relative risk of mortality from exposure to PM is higher for older individuals than for younger individuals. This, combined with the fact that the baseline incidence of mortality consists disproportionately of people 65 and over, suggests that PM-related mortality is disproportionately among older individuals. The extent to which prematurity of death among older individuals is on the order of days or weeks versus years, however, is more uncertain. The short-term exposure studies can provide little information on this. It is possible that premature deaths on high pollution days would have occurred only days later, if the individuals were sick and therefore particularly susceptible. The fact that the long-term exposure mortality studies found substantially larger relative risks, however, suggests that not all of the premature mortality is on the order of days or even weeks. Shortening of life of such a small duration would not be detectable in a long-term epidemiology study, ensuring that the effects detected in such studies must represent longer periods of life shortening. This suggests that at least some of the premature mortality associated with exposure to PM may reduce lifespans by substantially longer amounts of time.

Even if an individual's PM-related premature mortality is of very short duration, on the order of days, however, it may be misleading to characterize such a PM-related loss as only those few days if the individual's underlying susceptibility was itself exacerbated by chronic exposure to elevated levels of pollution. Suppose, for example, that long-term exposure to elevated PM levels compromises the cardiopulmonary system, making the individual more susceptible to mortality on peak PM days than he otherwise would have been. If this is the case, then the underlying susceptibility would itself be either caused by chronic exposure to elevated PM levels or exacer-

bated by it. Characterizing the individual's loss as a few days could, in this case, be a substantial underestimate.

In addition, the long-term studies estimate significantly more PM-related mortality than the annual sum of the daily estimates from the short-term studies, suggesting that the short-term studies may be missing a component of PM-related mortality that is being observed in the long-term studies. For example, if chronic exposure to elevated PM levels causes premature mortality that is not necessarily correlated with daily PM peak levels, this type of mortality would be detected in the long-term studies but not necessarily in the short-term studies. Two of the long-term exposure studies suggest, moreover, that the association between ambient air pollution and mortality cannot be explained by the confounding influences of smoking and other personal risk factors.

Uncertainties surround analyses based on epidemiological studies of PM and mortality. In addition to the uncertainty about the degree of prematurity of mortality, there are other uncertainties surrounding estimates based on epidemiological studies of PM and mortality. Although epidemiological studies are generally preferred to human clinical studies, there is nevertheless uncertainty associated with estimates of the risk of premature mortality (and morbidity) based on studies in the epidemiological literature. Considering all the epidemiological studies of PM and mortality, both short-term and long-term, there is significant interstudy variability as well as intrastudy uncertainty. Some of the difference among estimates reported by different studies may reflect only sampling error; some of the difference, however, may reflect actual differences in the concentration-response relationship from one location to another. The transferability of a concentration-response function estimated in one location to other locations is a notable source of uncertainty.

Although there may be more uncertainty about the degree of prematurity of mortality captured by short-term exposure studies than by long-term exposure studies, certain sources of uncertainty associated with long-term exposure studies require mention. Although studies that are well-executed attempt to control for those factors that may confound the results of the study, there is always the possibility of insufficient or inappropriate adjustment for those factors that affect long-term mortality rates and may be confounded with the factor of interest (e.g., PM concentrations). Prospective cohort studies have an advantage over ecologic, or population-based, studies in that they gather individual-specific information on such important risk factors as smoking. It is always possible, however, that a relevant, individual-specific risk factor may not have been controlled for or that some factor that is not individual-specific (e.g., climate) was not adequately controlled for. It is therefore possible that differences in mortality rates that have been ascribed to differences in average PM levels may be due, in part, to some other factor or factors (e.g., differences among communities in diet, exercise, ethnicity, climate, industrial effluents, etc.) that have not been adequately controlled for.

Another source of uncertainty surrounding the prospective cohort studies concerns possible historical trends in PM concentrations and the relevant period of exposure, which is as yet unknown. TSP concentrations were substantially higher in many locations for several years prior to the cohort studies and had declined substantially by the time these studies were conducted. If this is also true for PM_{10} and or $PM_{2.5}$, it is possible that the larger PM_{10} and or $PM_{2.5}$ coefficients reported by the long-term exposure studies (as opposed to the short-term exposure studies) reflect an upward bias. If the relevant exposure period extends over a decade or more, then a coefficient based on PM concentrations at the beginning of the study or in those years immediately prior to the study could be biased upward if pollution levels had been decreasing markedly for a decade or longer prior to the study.

On the other hand, if a downward trend in PM concentrations continued throughout the period of the study, and if a much shorter exposure period is relevant (e.g., contained within the study period itself), then characterizing PM levels throughout the study by those levels just prior to the study would tend to bias the PM coefficient downward.

The relevant exposure period is one of a cluster of characteristics of the mortality-PM relationship that are as yet unknown and potentially important. It is also unknown whether there is a time lag in the PM effect. Finally, it is unknown whether there may be cumulative effects of chronic exposure — that is, whether the relative risk of mortality actually increases as the period of exposure increases.

Estimating the relationship between PM and premature mortality. The incidence of PM-related mortality used for estimating the benefits of the CAA is

based on the concentration-response relationship reported by one of the two recent long-term exposure (prospective cohort) studies (Pope et al., 1995, and Dockery et al., 1993). Because it is based on a much larger population and many more locations than Dockery et al. (1993), the concentration-response function from Pope et al. (1995) was used in this analysis. The results of Pope et al. are consistent with those of Dockery et al., which reported an even larger response, but in only six cities. Moreover, Pope et al. is also supported by several ecological cross-sectional studies of annual mortality based on 1960 and 1970 census data (using either TSP or sulfate as indicators of PM), including the work of Lave and Seskin (1977) and Lipfert (1984).

Numerous short-term exposure (time series) studies have also reported a positive and statistically significant relationship between PM and mortality. Of the fourteen studies that estimated the relationship between daily PM_{10} concentrations and daily mortality listed in Table 12-2 of the PM Criteria Document, twelve reported positive and statistically significant findings (Pope et al., 1992; Pope and Kalkstein, 1996; Dockery et al., 1992; Schwartz, 1993a; Ozkaynak et al., 1994; Kinney et al., 1995: Ito et al., 1995; Ostro et al., 1996; Saldiva et al., 1995; Styer et al., 1995; Ito and Thurston, 1996; Schwartz et al., 1996). While these studies lend substantial support to the hypothesis that there is a relationship between PM_{10} and mortality, they may be capturing only the portion of that relationship involving short-term effects. For this reason, they are considered in this analysis only as supporting evidence to the results of the study by Pope et al.

The Pope et al. study has several further advantages. The population followed in this study was largely white and middle class, decreasing the likelihood that interlocational differences in premature mortality were due in part to differences in socioeconomic status or related factors. In addition, the generally lower mortality rates and possibly lower exposures to pollution among this group, in comparison to poorer minority populations, would tend to bias the PM coefficient from this study downward, counteracting a possible upward bias associated with historical air quality trends discussed above.

Another source of downward bias in the PM coefficient in Pope et al. is that intercity movement of cohort members was not considered in this study. Migration across study cities would result in exposures of cohort members being more similar than would be indicated by assigning city-specific annual average pollution levels to each member of the cohort. The more intercity migration there is, the more exposure will tend toward an intercity mean. If this is ignored, differences in exposure levels, proxied by differences in city-specific annual median PM levels, will be exaggerated, resulting in a downward bias of the PM coefficient (because a given difference in mortality rates is being associated with a larger difference in PM levels than is actually the case).

In summary, because long-term exposure studies appear to have captured more of the PM-related premature mortality, as well as premature mortality that is more premature, they are preferable to the short-term exposure studies. Among the long-term exposure studies, the Pope et al. study has several advantages, as discussed above, which are likely to reduce the possibility of a key source of confounding and increase the reliability of the concentration-response function from that study. For these reasons, the concentration-response function estimated in this study is considered the most reasonable choice for this analysis.

Matching PM Indices in the Air Quality Profiles and Concentration-Response Function. The Pope et al. study examined the health effects associated with two indices of PM exposure: sulfate particles and fine particles ($PM_{2.5}$). The reported mortality risk ratios are slightly larger for $PM_{2.5}$ than for sulfates (1.17 versus 1.15 for a comparison between the most polluted and least polluted cities). The $PM_{2.5}$ relationship is used in this analysis because it is more consistent with the PM_{10} air quality data selected for the analysis. Estimated changes in $PM_{2.5}$ air quality must be matched with the $PM_{2.5}$ mortality relationship. However, only PM_{10} profiles were used for the entire 20 year period. Therefore, the same regional information about the PM_{10} components (sulfate, nitrate, organic particulate and primary particulate) used to develop the PM_{10} profiles were used to develop regional $PM_{2.5}/PM_{10}$ ratios. Although both urban and rural ratios are available, for computational simplicity, only the regional urban ratios were used to estimate the $PM_{2.5}$ profiles from the PM_{10} profiles used in the analysis. This reflects the exposure of the majority of the modeled population (i.e., the urban population), while introducing some error in the exposure changes for the rural population. In the east and west, where the rural ratio is larger than the urban ratio, the change in $PM_{2.5}$ exposure will be underestimated for the rural population.

In the central region the $PM_{2.5}$ change will be overestimated. These ratios were used in each year during 1970-1990, introducing another source of uncertainty in the analysis. Table D-5 summarizes the $PM_{2.5}/PM_{10}$ ratios used in this analysis.

Table D-5. $PM_{2.5}/PM_{10}$ Ratios Used to Estimate $PM_{2.5}$ Data Used With Pope et al. (1995) Mortality Relationship.

	East	Central	West	National
Urban	0.59	0.58	0.48	0.55
Rural	0.68	0.53	0.49	0.57

Prematurity of Mortality: Life-Years Lost as a Unit of Measure

Perhaps the most important health effect that is examined in this analysis is mortality. Although this analysis does not take into account the degree of prematurity of death (that is, the ages of those individuals who die prematurely from exposure to PM are not considered), considerable attention has been paid to this issue and, in particular, to life-years lost as an alternative to lives lost as a measure of the mortality-related effects of pollution.

Because life-years lost is of potential interest and because there is a substantial potential for confusion in understanding apparently disparate estimates of life-years lost from pollution exposure, this section attempts to present a clear discussion of the various possible measures of life-years lost, what they depend on, and how they are related to each other.

Because the actual number of years any particular individual is going to live cannot be known, "life-years lost" by an individual actually refers to an *expected* loss of years of life by that individual. The expected loss of years of life by an individual depends crucially on whether the expectation is contingent on the individual only having been exposed to PM or on the individual actually having died from that exposure.

An *ex ante* estimate of life-years lost per individual is contingent not on the individual having died prematurely but only on the individual having been exposed. Suppose, for example, that a 25 year old has a life expectancy of 50 more years in the absence of exposure and only 49 more years in the presence of exposure. Given (chronic) exposure from the age of 25 on, the 25 year old exposed to (some elevated level of) PM might expect a shortening of life expectancy of one year, for example. That is one expected life-year lost due to chronic exposure. This is the life-years lost that can be expected by every *exposed* individual.

An *ex post* estimate of life-years lost per individual is contingent on the individual actually having died from exposure to PM. When an individual dies of exposure to PM, he is said to have lost the number of years he would have been expected to live, calculated, for example, from age- and gender-specific life expectancy tables. Suppose that the life expectancy of 25 year olds is 75 — that is, a 25 year old can expect to live 50 more years. A 25 year old who dies from exposure to PM has therefore lost 50 expected years of life. This is the life-years lost that can be expected by every 25 year old *affected* individual (i.e., every 25 year old who actually dies from exposure to PM).

Estimates of the total life-years lost by a population exposed to PM depend on several factors, including the age distribution and the size of the exposed population, the magnitude of the change (or changes) in PM being considered, the relative risk assumed to be associated with each change in PM, and the length of time exposure (i.e., the change in PM) is presumed to occur. A population chronically exposed to a given increase in PM will lose more life-years than a population exposed to the same increase in PM for only a year or two.[6] A population that is generally older will lose fewer life-years, all else equal, than one that is generally younger, because older individuals have fewer (expected) years of life left to lose. And a population exposed to a greater increase in PM will lose more life-years than if it were exposed to a smaller increase in PM. Finally, the life-years lost by the population will increase as the relative risk associated with the increase in PM increases.

Life-years lost are usually reported as averages over a population of individuals. The population being averaged over, however, can make a crucial dif-

[6] Even in the absence of cumulative effects of exposure, exposure of a population for many years will result in a greater total number of pollution-related deaths than exposure for only a year or two, because the same relative risk is applied repeatedly, year after year, to the population, rather than for only a year or two.

ference in the reported average life-years lost, as noted above. The average life-years lost *per exposed individual* (the *ex ante* estimate) is just the total life-years lost by the population of exposed individuals divided by the number of exposed individuals. This average will depend on all the factors that the total life-years lost depends on except the size of the exposed population. The average life-years lost by an exposed individual is a statistical expectation. It is the average of the numbers of life-years actually lost by each member of the exposed population. Alternatively, it can be thought of as a weighted average of possible numbers of years lost, where the weights are the proportions of the population that lose each number of expected years of life. Although those individuals who do die prematurely from exposure to PM may lose several expected years of life, most exposed individuals do not actually die from exposure to PM and therefore lose zero life-years. The average life-years lost per exposed individual in a population, alternatively referred to as the average decrease in life expectancy of the exposed population, is therefore heavily weighted towards zero. The average number of life-years lost *per individual who dies of exposure to PM* (the *ex post* measure of life-years lost) is an average of the numbers of expected years of life lost by individuals who actually died prematurely because of PM. Because everyone who dies prematurely from exposure to PM loses some positive number of expected years of life, this average, by definition, does not include zero.

An example of an *ex ante* measure of life-years lost is given by a study in the Netherlands (WHO, 1996), which considered a cohort of Dutch males, aged 25-30, and compared the life expectancy of this cohort to what it would be in a hypothetical alternative scenario in which these individuals are continuously exposed to concentrations of $PM_{2.5}$ that are 10 $\mu g/m^3$ lower than in the actual scenario. The life expectancy of this cohort of 25-30 year old Dutch males was calculated to be 50.21 years in the actual scenario, based on a 1992 life table from the Netherlands. Assuming that the relative risk of mortality associated with an increase of 10 $\mu g/m^3$ $PM_{2.5}$ is 1.1 (the average of the relative risks of 1.14 from Dockery et al., 1993, and 1.07 from Pope et al., 1995), the study authors calculated death rates in the hypothetical "cleaner" scenario by dividing the age-specific death rates in the actual scenario by 1.1. Using these slightly lower death rates, and assuming that the effect of PM does not begin until 15 years of exposure, the authors constructed a life table for the cohort in the hypothetical "cleaner" scenario. Based on this new life table in a cleaner world, the life expectancy of the cohort of 25-30 year old Dutch males was calculated to be 51.32 years in the hypothetical cleaner scenario. (In calculating life expectancies in both the "dirty" scenario and the "clean" scenario, it is assumed that any individual who does not survive to the next 5-year age group lives zero more years. For example, a 30 year old individual either survives to age 35 or dies at age 30.) The change in life expectancy for this cohort of 25-30 year old Dutch males, due to a change in PM exposure of 10 $\mu g/m^3$ for the rest of their lives (until the age of 90), was therefore 51.32 years - 50.21 years = 1.11 years. That is, the average life-years lost by an exposed individual in this population, under these assumptions, is 1.11 years.

The estimate of 1.11 years of expected life lost depends on several things, as mentioned above. If the study authors had used the relative risk from Pope et al., 1995, alone, (1.07 instead of 1.1), for example, the change in life expectancy (the *ex ante* measure of life-years lost) for this cohort of 25-30 year old Dutch males would have been 0.80 years. Similarly, changing the assumption about the duration of exposure also changes the estimate of *ex ante* life-years lost. Using a relative risk of 1.1, but assuming that exposure lasts only during the first 5 years (i.e., that the death rate in the first five years, from age 25 through age 30, is lower but that after that it is the same as in the "dirty" scenario), the average life-years lost by an exposed individual in this population is reduced from 1.11 years to 0.02 years.

By their construction and definitions, the average life-years lost per exposed individual and the average life-years lost per affected individual (i.e., per individual who dies prematurely from PM) take the same total number of life-years lost by the exposed population and divide them by different denominators. The average life-years lost per exposed individual divides the total life-years lost by the total population exposed; the average life-years lost per affected individual divides the same total life-years lost by only a small subset of the total population exposed, namely, those who died from PM. The average per exposed individual is therefore much smaller than the average per affected individual. Because both types of average may be reported, and both are valid measurements, it is important to understand that, although the numbers will be very dissimilar, they are consistent with each other and are simply different measures of the estimated mortality impact of PM.

To calculate the total (estimated) life-years lost by a population, it is necessary to follow each age cohort in the population through their lives in both scenarios, the "dirty" scenario and the "clean" scenario, and compute the difference in total years lived between the two scenarios, as WHO (1996) did for the cohort of Dutch males 25-30 years old. This method will be referred to as Method 1. In practice, however, it is not always possible to do this. (Other changes to the population, such as those from recruitment and immigration, for example, would make such an exercise difficult.) An alternative method, which approximates this, is to predict the numbers of individuals in each age category who will die prematurely from exposure to PM (i.e., who will die prematurely in the "dirty" scenario), and multiply each of these numbers by the corresponding expected number of years remaining to individuals in that age category, determined from life expectancy tables. This method will be referred to as Method 2. Suppose, for example, that individuals age 25 are expected to live to age 75, or alternatively, have an expected 50 years of life remaining. Suppose that ten 25 year olds are estimated to die prematurely because of exposure to PM. Their expected loss of life-years is therefore 50 years each, or a total of 500 life-years. If the same calculation is carried out for the individuals dying prematurely in each age category, the sum is an estimate of the total life-years lost by the population.

Using Method 1 (and retaining the assumptions made by WHO, 1996), the average life-years lost per PM-related death among the cohort of Dutch males is calculated to be 14.28 years. Using Method 2 it is estimated to be 14.43 years.

Although this *ex post* measure of life-years lost is much larger than the *ex ante* measure (1.11 life-years lost per exposed individual), it only applies to those individuals who actually die from exposure to PM. The number of individuals in the age 25-30 Dutch cohort example who eventually die from exposure to PM (7,646) is much smaller than the number of individuals in the age 25-30 Dutch cohort who are exposed to PM (98,177). The total life-years lost can be calculated either as the number of exposed individuals times the expected life-years lost per exposed individual (98,177*1.11 = 109,192.1) or as the number of affected individuals times the expected life-years lost per affected individual (7,646*14.28 = 109,192.1).

To further illustrate the different measures of life-years lost and the effects of various input assumptions on these measures, death rates from the 1992 U.S. Statistical Abstract were used to follow a cohort of 100,000 U.S. males from birth to age 90 in a "dirty" scenario and a "clean" scenario, under various assumptions. Death rates were available for age less than 1, ages 1-4, and for ten-year age groups thereafter. The ten-year age groups were divided into five-year age groups, applying the death rate for the ten-year group to each of the corresponding five-year age groups. *Ex ante* and *ex post* measures of life-years lost among those individuals who survive to the 25-29 year old category were first calculated under the assumptions in the WHO (1996) study. These assumptions were that the relative risk of mortality in the "dirty" scenario versus the "clean" scenario is 1.1; that exposure does not begin until age 25; that the effect of exposure takes fifteen years; that individuals at the beginning of each age grouping either survive to the next age grouping or live zero more years; and that all individuals age 85 live exactly five more years. Under these assumptions, the expected life-years lost per exposed individual in the 25-29 year old cohort is 1.32 years. There are 96,947 exposed individuals in this age cohort. The expected life-years lost per affected individual (i.e., per PM-related death) is 16.44 years (Method 1). There are 7,804 affected individuals. The total life-years lost by individuals in this cohort is 128,329.3 (1.32*96,947 = 16.44* 7,804 = 128,329.3).

If the relative risk is changed to 1.07, the expected life-years lost per exposed individual in the cohort of 25-29 year old U.S. males is reduced from 1.32 to 0.95 years. The expected life-years lost per affected individual (i.e., per PM-related death) is 16.44 years (Method 1). Using a relative risk of 1.1 but assuming no lag (i.e., assuming that exposure starts either at birth or at age 25 and has an immediate effect), the expected life-years lost per exposed individual in the 25-29 year old cohort changes from 1.32 to 1.12. The expected life-years lost per affected individual (i.e., per PM-related death) becomes 19.7 years (Method 1).

Estimating Morbidity Effects

In addition to mortality effects, this analysis quantifies effects for a number of non-fatal health endpoints. Several issues arise in implementing the studies selected for this analysis.

Overlapping Health Effects

Several endpoints reported in the health effects literature overlap with each other. For example, the literature reports relationships for hospital admissions for single respiratory ailments (e.g. pneumonia or chronic obstructive pulmonary disease) as well as for all respiratory ailments combined. Similarly, several studies quantify the occurrence of respiratory symptoms where the definitions of symptoms are not unique (e.g., shortness of breath, upper respiratory symptoms, and any of 19 symptoms). Measures of restricted activity provide a final example of overlapping health endpoints. Estimates are available for pollution-induced restricted activity days, mild restricted activity days, activity restriction resulting in work loss. This analysis models incidence for all endpoints. Double-counting of benefits is avoided in aggregating economic benefits across overlapping endpoints (see Appendix I).

Studies Requiring Adjustments

Applying concentration-response relationships reported in the epidemiological literature to the national scale benefits analysis required by section 812 required a variety of adjustments.

Normalization of coefficients by population. To be applied nationwide, concentration-response coefficients must reflect the change in risk per person per unit change in air quality. However, some studies report the concentration-response coefficient, , as the change in risk for the entire studied population. For example, Thurston et al. (1994) reported the total number of respiratory-related hospital admissions/day in the Toronto, Canada area. To normalize the coefficient so that it might be applied universally across the country, it was divided by the population in the geographical area of study (yielding an estimate of the change in admissions/person/day due to a change in pollutant levels).

Within-study meta-analysis. In some cases, studies reported several estimates of the concentration-response coefficient, each corresponding to a particular year or particular study area. For example, Ostro and Rothschild (1989) report six separate regression coefficients that correspond to regression models run for six separate years. This analysis combined the individual estimates using a fixed coefficient meta-analysis on the six years of data.

Conversion of coefficients dependent on symptom status during the previous day. Krupnick et al. (1990) employed a Markov process to determine the probability of symptoms that were dependent on symptom status of the previous day. The current analysis adjusts the regression coefficients produced by the model in order to eliminate this dependence on previous day's symptom status.

Concentration-Response Functions: Health Effects

After selecting studies appropriate for the section 812 analysis, taking into account the considerations discussed above, the published information was used to derive a concentration-response function for estimating nationwide benefits for each health effect considered. In general, these functions combine air quality changes, the affected population and information regarding the expected per person change in incidence per unit change in pollutant level. The following tables present the functions used in this analysis, incorporating information needed to apply these functions and references for information.

Particulate Matter

The concentration-response functions used to quantify expected changes in health effects associated with reduced exposure to particulate matter are summarized in Table D-6. The data profiles selected for use in this analysis are PM_{10}. In those cases in which PM_{10} was not the measure used in a study, this analysis either converted PM_{10} air quality data to the appropriate air quality data (e.g., $PM_{2.5}$ or TSP) or, equivalently, converted the pollutant coefficient from the study to the corresponding PM_{10} coefficient, based on location-specific information whenever possible.

Table D-6. Summary of Concentration-Response Functions for Particulate Matter.

Except where noted otherwise, the functional form is

$$\Delta cases = cases * (e^{\beta * \Delta PM\text{-}10} - 1)$$

where "cases" refers to incidence at the first pollution level.

Health Endpoint (ICD-9 code)	Baseline Incidence (per 100,000)	Expos Meas. from Original Study	Study Pop.	Applied Pop.	Functional form[a]	Uncert & Var.	Sources
mortality (long-term exposure)	non-accidental deaths by county[b]	annual median $PM_{2.5}$	50 cities, all deaths	over age 30	$\beta_{PM2.5} = 0.006408$ PM_{10} data converted to $PM_{2.5}$ data[c]	s.e. = 0.00148	Pope et al., 1995 American Cancer Society cohort
hospital admissions--all resp. illnesses (ICD 460-519)	504[d]/year (incidence in pop. > 65 years of total U.S. pop.)	same day PM_{10}	65 and older in New Haven, CT, Tacoma, WA	65 and older	New Haven: 0.00172 Tacoma: 0.00227 average: 0.0020	c.i. = New Haven: 1.00-1.12 s.e. = 0.00093 Tacoma: 0.97-1.29 s.e. = 0.00146	Schwartz, 1995 New Haven and Tacoma
hospital admissions -- all resp. illnesses (ICD 460-519)	n/a	mean monthly PM_{10}	variety of ages in Salt Lake Valley, Utah	all	Δ cases = $\beta * \Delta PM_{10} *$ Pop. where β = 0.8047 monthly admissions / Salt Lake Valley population (780,000). $= 3.4 \times 10^{-8}$ (converted from monthly to daily admissions)	s.e. = 0.28	Pope, 1991 Salt Lake Valley
daily respiratory admissions (total) includes 466, 480, 481, 482, 485, 490, 491, 492, 493	n/a	same-day PM_{10}	Toronto metro area	all	Δ cases = $\beta * \Delta PM_{10} *$ Pop where β = 0.0339 daily admissions / Toronto population (2.4 million) $= 1.4 \times 10^{-8}$ (model also includes O_3)	s.e. = 0.034/2.4 million $= 1.4 \times 10^{-8}$	Thurston et al. 1994 Toronto
hospital admissions pneumonia (480-487)	229[d]/year (incidence in pop. > 65 years of total U.S. pop.)	same day PM_{10}	over 65, Birmingham AL	over 65	β = 0.00174	c.i. = 1.07 - 1.32 s.e. = 0.000536	Schwartz, 1994a Birmingham

Health Endpoint (ICD-9 code)	Baseline Incidence (per 100,000)	Expos Meas. from Original Study	Study Pop.	Applied Pop.	Functional form[a]	Uncert & Var.	Sources
hospital admissions COPD (490-496)	103[d]/year (incidence in pop. > 65 years of total U.S. pop.)	same day PM_{10}	over 65, Birmingham AL	over 65	$\beta = 0.00239$	c.i. = 1.08 - 1.50 s.e. = 0.00084	Schwartz, 1994a Birmingham
hospital admissions pneumonia (480-487)	229[d]/year (incidence in pop. > 65 years of total U.S. pop.)	same day PM_{10}	over 65, Detroit	over 65	$\beta = 0.00115$	s.e. = 0.00039	Schwartz, 1994b Detroit
hospital admissions COPD (490-496)	103[d]/year (incidence in pop. > 65 years of total U.S. pop.)	same day PM_{10}	over 65, Detroit	over 65	$\beta = 0.00202$	s.e. = 0.00059	Schwartz, 1994b Detroit
hospital admissions pneumonia (480-487)	229[d]/year (incidence in pop. > 65 years of total U.S. pop.)	same day PM_{10}	65 and over in Mpls	over 65	$\beta = 0.00157$	c.i. = 1.02 - 1.33 s.e. = 0.00068	Schwartz, 1994c Mpls, St. Paul
hospital admissions COPD (490-496)	103[d]/year (incidence in pop. > 65 years of total U.S. pop.)	current and previous day PM_{10}	65 and over in Mpls	over 65	$\beta = 0.00451$	c.i. = 1.20 - 2.06 s.e. = 0.00138	Schwartz, 1994c Mpls, St. Paul
hospital admissions for congestive heart failure (ICD 428)	231[d]/year (incidence in pop. > 65 years of total U.S. pop.)	avg same and previous day PM_{10}	65 and older in Detroit	65 and older	$\beta = 0.00098$	c.i. = 1.012-1.052 s.e. = 0.00031	Schwartz and Morris, 1995 Detroit
hospital admissions for ischemic heart disease (ICD 410-414)	450[d]/year (incidence in pop. > 65 years of total U.S. pop.)	24 hr avg PM_{10} same day	65 and older in Detroit	65 and older	$\beta = 0.00056$	c.i. = 1.005-1.032 s.e. = 0.00021	Schwartz and Morris, 1995 Detroit

Health Endpoint (ICD-9 code)	Baseline Incidence (per 100,000)	Expos Meas. from Original Study	Study Pop.	Applied Pop.	Functional form[a]	Uncert & Var.	Sources
hospital admissions -- all resp. illnesses (ICD 460-519)	504[d]/year (incidence in pop. > 65 years of total U.S. pop.)	24 hr avg PM_{10}	over 65, Spokane	over 65	$\beta = 0.00163$	s.e. = 0.00047	Schwartz, 1996, Spokane
hospital admissions COPD (490-496)	103[d]/year (incidence in pop. > 65 years of total U.S. pop.)	24 hr avg PM_{10}	over 65, Spokane	over 65	$\beta = 0.00316$	s.e. = 0.00084	Schwartz, 1996, Spokane
hospital admissions pneumonia (480-487)	229[d]/year (incidence in pop. > 65 years of total U.S. pop.)	24 hr avg PM_{10}	over 65, Spokane	over 65	$\beta = 0.00103$	s.e. = 0.00068	Schwartz, 1996, Spokane
LRS defined as cough, chest pain, phlegm, and wheeze	not applicable	same day PM_{10}	8-12 yr olds	0-12 yr olds	$\Delta cases = \frac{P_0}{(1-P_0) * e^{\beta * \Delta PM10}} - P_0$ where P_0 = the probability of a child in the study pop suffering from LRS in the base case = 1.45 % and $\beta = 0.014176$	s.e. = 0.0041	Schwartz et al., 1994d
shortness of breath, days	not applicable	24 hour avg PM_{10}	African-American asthmatics between ages 7 and 12	same as study pop.	$\Delta cases = \frac{P_0}{(1-P_0) * e^{\beta * \Delta PM10}} - P_0$ where P_0 = the probability of a child in the study pop. suffering from shortness of breath in the base case = 5.6 % and $\beta = 0.008412$	s.e. = 0.00363	Ostro et al., 1995

Health Endpoint (ICD-9 code)	Baseline Incidence (per 100,000)	Expos Meas. from Original Study	Study Pop.	Applied Pop.	Functional form[a]	Uncert & Var.	Sources
URI defined as runny or stuffy nose, wet cough, burning, aching, or red eyes	1,192[e] (ages 10-12) 5,307[e] (ages <= 12)	same day PM_{10}	10-12 yr old non-symptomatic	12 and under	$\beta = 0.0036$	s.e. = 0.0015	Pope et al., 1991 Utah
acute bronchitis (ICD 466)	n/a	PM_{10} annual avg (converted)	10 to 12 year olds	18 and under	$\beta = 0.0330$ $\Delta cases = \frac{P_0(e^{\beta * \Delta PM-10}) }{1 - P_0 + P_0(e^{\beta * \Delta PM-10})} * Pop$ P_0 = baseline probability of having bronchitis =0.065[e]	s.e. = 0.0216	Dockery et al., 1989 6 cities
chronic bronchitis	710/year (of study pop.)	annual mean TSP	Seventh Day Adventists in California	all	$\beta = 0.00512$ convert PM_{10} to TSP: $\Delta TSP = \frac{\Delta PM10}{0.56}$ where 0.56 is the specific conversion based on region and initial TSP conc.	not available	Abbey et al., 1993

Health Endpoint (ICD-9 code)	Baseline Incidence (per 100,000)	Expos Meas. from Original Study	Study Pop.	Applied Pop.	Functional form[a]	Uncert & Var.	Sources
chronic bronchitis	600/year	annual mean TSP	adults 30-74 years old in 53 U.S. urban areas	all	Δ cases/year = (p_1 - p_0) * Pop where $p_1 = \frac{1}{1 + e^{-(\ln \frac{p_0}{1 - p_0} + \beta * \Delta PM_{10})}}$ where p_0 = 0.006 = the probability of developing physician-diagnosed chronic bronchitis per individual per year and β = 0.0012, the PM_{10} coefficient, converted from the TSP coefficient, using the relationship: $\Delta TSP = \frac{\Delta PM10}{0.56}$ where 0.56 is the specific conversion based on region and initial TSP conc.	95% CI = (1.02 - 1.12) for odds ratio corresponding to a 10 µg/m3 increase in annual TSP	Schwartz, 1993b
presence of any of 19 acute respiratory symptoms	not applicable	24 hour average COH in units/ 100 ft)[g] COH = coeff. of haze	adult members of families of elementary school-aged children in Glendora-Covina-Azusa, CA	adults 18-65	Δ $Sympt_{days}$/day = (p_1 - p_0) * Pop where $p_1 = \frac{1}{1 + e^{-(\ln \frac{p_0}{1 - p_0} + \beta * \Delta O_3)}}$ and p_0 = the probability of $Sympt_{days}$ per individual for a 24-hour period in the base case = 0.19 β = 0.00046[g] (Model includes O_3, COH, SO_2)	s.e. = 0.00024[h]	Krupnick et al., 1990

Health Endpoint (ICD-9 code)	Baseline Incidence (per 100,000)	Expos Meas. from Original Study	Study Pop.	Applied Pop.	Functional form[a]	Uncert & Var.	Sources
moderate or worse asthma status	n/a	average $PM_{2.5}$ during 9:00 am to 4:00 pm ($\mu g/m^3$)	Denver asthmatics between ages 18 and 70	asthmatic (4%[i] of total pop.)	Δ asthma status= $\beta[\ln(X_1/X_0)]$*Pop where X_0 = PM_{10} concentrations with CAA, X_1 = PM_{10} concentrations without CAA, and $\beta = 0.00038$[j] (model includes $PM_{2.5}$ and modeled $PM_{2.5}$ measures for periods where $PM_{2.5}$ measures were missing)	s.e. = 0.00019	Ostro et al., 1991 Denver
Restricted Activity Days (RADs)	400,531 days/year[k] (of the total U.S. pop)	2-wk average $PM_{2.5}$ ($\mu g/m^3$)	All adults 18-65 in metropolitan areas in the U.S.	adults aged 18-65	Δ health effects determined over a 2 wk period $\beta = 0.0030$[j, l]	s.e. = 0.00018[l]	Ostro, 1987
respiratory and nonrespiratory conditions resulting in a minor restricted activity day (MRAD)	780,000 days/year (cited as 7.68 days per person per year in study)	$PM_{2.5}$ averaged over a 2-wk period	employed adults across the U.S. between the ages of 18-65	adults aged 18-65	number of health effects determined over a 2-week period $\beta = 0.00463$[j, l] (Model includes fine particulates and O_3)	s.e. = 0.00044[l]	Ostro and Rothschild, 1989
respiratory restricted activity days (RRADs)	306,000 days/year (cited as 3.06 days per person per year in study)	$PM_{2.5}$ averaged over a 2 wk period	employed adults across the U.S. between the ages of 18-65	adults aged 18-65	number of health effects determined over a 2-wk period $\beta = 0.00936$[j, l] (Model includes fine particulates and O_3)	s.e. = 0.00103[l]	Ostro and Rothschild, 1989
Work Loss Days (WLDs)	150,750[m] (of total U.S. pop)	2-wk average $PM_{2.5}$ ($\mu g/m^3$)	All adults 18-65 in metropolitan areas in the U.S.	adults aged 18-65	Δ health effects determined over a 2 wk period $\beta = 0.0029$[j, l]	s.e. = 0.00022[l]	Ostro, 1987

NOTES:

[a] Pollutant coefficients reflect changes in health effects per change in $\mu g/m^3$ PM_{10}.

[b] Mortality baseline incidence data for each county taken from Vital Statistics of the United States, Vol. II - Mortality, Part B, (U.S. Dept. of Health and Human Services). Incidence rates were generated for total mortality excluding accidental deaths and adverse effects, suicide, homicide, and other external causes (ICD E800-E999). Rates calculated based on 1990 population.

[c] PM_{10} data converted to $PM_{2.5}$ data by using national urban average $PM_{2.5}/PM_{10}$ ratio = 0.56.

[d] Centers for Disease Control, 1992. Vital and Health Statistics, Detailed Diagnoses and Procedures, National Hospital Discharge Survey, 1990. Number of 1990 discharges divided by 1990 U.S. population (248,709,873) from City and County Databook, 12th edition, 1994, U.S. Dept. of Commerce, Bureau of the Census, Washington, D.C.

[e] Pope et al., 1991 NOTE: rates were not available from standard incidence sources and so were calculated from incidence in the study of 10-12 year olds. This may not be entirely appropriate for older or younger individuals. Children of this age are less likely to have colds than much younger children and may be more representative of the adult population.

[f] Dockery et al., 1989.

[g] Coefficient and standard error are converted from a β and s.e. for coefficient of haze (COH) to a β and s.e. for PM_{10}. This was done by using a ratio of COH to TSP of 0.116 from the study authors (as cited in ESEERCO, 1994) and a ratio of PM_{10} to TSP of 0.55 (U.S. EPA, 1986).

[h] Coefficient and standard error incorporate the stationary probabilities as described in Krupnick et al. (1990). To do this, the calculation used the transitional probabilities supplied by the authors and presented in ESEERCO, 1994.

[i] U.S. EPA, 1994a.

[j] β converted from a change in health effects per change in $\mu g/m^3$ $PM_{2.5}$ to a change per $\mu g/m^3$ PM_{10} using the following relationship: 1 $\mu g/m^3$ $PM_{2.5}$ = 0.56 $\mu g/m^3$ PM_{10} (ESEERCO, 1994)

[k] Number of RADs for all acute conditions from: National Center for Health Statistics. Current Estimates from the National Health Interview Survey: United States, 1990. (Hyattsville, MD). This number is divided by the U.S. population for 50 states for 1990 (248,709,873) and multiplied by 100,000 (to obtain the incidence per 100,000).

[l] Based on fixed-weight meta-analysis of single-year coefficients and standard errors reported in study.

[m] Number of WLDs of 374,933,000 from: National Center for Health Statistics. Current Estimates from the National Health Interview Survey from 1990. (Hyattsville, MD). Series 10, No. 181. This number is divided by the U.S. population for 50 states for 1990 (248,709,873) and multiplied by 100,000 (to obtain the incidence per 100,000).

Ozone

The health effects literature includes studies of the relationships between ozone and a variety of non-fatal health effects. Many of these relationships are provided by the same studies that reported the particulate matter relationships shown above. For some health endpoints, most notably hospital admissions, multiple studies report alternative estimates of the concentration-response relationship. The variability between these reported estimates is incorporated into the Monte Carlo approach used to combine estimates of avoided health effects with economic valuations (discussed in Appendix I). Table D-7 documents the concentration-response functions used in this analysis.

Table D-7. Summary of Concentration-Response Functions for Ozone.

Except where noted otherwise, the functional form is

$$\Delta cases = cases * (e^{\beta * \Delta O3} - 1)$$

where "cases" refers to incidence at the first pollution level.

Health Endpoint (ICD-9 code)	Baseline Incidence (per 100,000)	Expos Meas from original study	Study Pop.	Applied Pop.	Functional form[a]	Uncert & Var.	Sources
hospital admissions -- all resp. illnesses (ICD 460-519)	504/year[b] (incidence in pop .> 65 years of total U.S. pop.)	24 hr avg ($\mu g/m^3$)	65 and older in New Haven, CT, Tacoma, WA	over 65 only	β = New Haven: 0.0027 Tacoma: 0.007 where 1 µg/m3= 0.510 ppb (two pollutant model with PM_{10} and O_3)	New Haven: s.e. = 0.0014 Tacoma: s.e. = 0.0025 where $1\mu g/m^3$ = 0.51 ppb	Schwartz, 1995 New Haven and Tacoma
daily respiratory admissions-includes 466, 480, 481, 482, 485, 490, 491, 492, 493	n/a	1 hour daily max ozone (ppb)	all	all	for Toronto: β = 0.0388/2.4 million $=1.62x10^{-8}$ Δ cases/day = $\beta * \Delta O_3 *$ pop (ozone and PM_{10} model used)	se = 0.0241/2.4 million $=1.0x10^{-8}$	Thurston et al., 1994 Toronto
hospital admissions pneumonia (480-487)	229/year[b] (incidence in pop .> 65 years of total U.S. pop.)	24-hr avg ppb	over 65, Birmingham AL	over 65	β = 0.00262 for O_3 alone (single pollutant model only avail.)	s.e. = 0.00196	Schwartz, 1994a Birmingham

Health Endpoint (ICD-9 code)	Baseline Incidence (per 100,000)	Expos Meas from original study	Study Pop.	Applied Pop.	Functional form[a]	Uncert & Var.	Sources
hospital admissions COPD (490-496)	103/year[b] (incidence in pop .> 65 years of total U.S. pop.)	24-hr avg ppb	over 65, Birmingham AL	over 65	$\beta = 0.00314$ for O_3 only (only single pollutant model avail.)	s.e. = 0.00316	Schwartz, 1994a Birmingham
hospital admissions pneumonia (480-487)	229/year[b] (incidence in pop .> 65 years of total U.S. pop.)	24-hr avg ppb	over 65, Detroit	over 65	$\beta = 0.00521$ (two pollutant model with O_3 and PM_{10}) note: authors suggest a threshold of 25 ppb	s.e. = 0.0013	Schwartz, 1994b Detroit
hospital admissions COPD (490-496)	103/year[b] (incidence in pop .> 65 years of total U.S. pop.)	24-hr avg ppb	over 65, Detroit	over 65	$\beta = 0.00549$ (two pollutant model with O_3 and PM_{10}) note: authors suggest a threshold of 25 ppb	s.e. = 0.00205	Schwartz, 1994b Detroit
hospital admissions pneumonia (ICD 480-487)	229/year[b] (incidence in pop .> 65 years of total U.S. pop.)	24 hr avg ppb	65 and over in Mpls	over 65	$\beta = 0.002795$ (two pollutant model with O_3 and PM_{10})	s.e. = 0.00172	Schwartz 1994c Mpls, St. Paul
hospital admissions -- all resp. illnesses (ICD 460-519)	504/year[b] (incidence in pop .> 65 years of total U.S. pop.)	1 hour daily max ozone (ppb)	over 65, Spokane	over 65	$\beta = 0.008562$	s.e. = 0.004326	Schwartz, 1996, Spokane
hospital admissions COPD (490-496)	103/year[b] (incidence in pop .> 65 years of total U.S. pop.)	1 hour daily max ozone (ppb)	over 65, Spokane	over 65	$\beta = 0.004619$	s.e. = 0.007739	Schwartz, 1996, Spokane

Health Endpoint (ICD-9 code)	Baseline Incidence (per 100,000)	Expos Meas from original study	Study Pop.	Applied Pop.	Functional form[a]	Uncert & Var.	Sources
hospital admissions pneumonia (ICD 480-487)	229/year[b] (incidence in pop .> 65 years of total U.S. pop.)	1 hour daily max ozone (ppb)	over 65, Spokane	over 65	$\beta = 0.00965$	s.e. = 0.006011	Schwartz, 1996, Spokane
presence of any of 19 acute respiratory symptoms	n/a	daily one-hour max. O_3 (pphm)	adult members of families of elementary school-aged children in Glendora-Covina-Azusa, CA	adults 18-65	$\Delta\ Sympt_{days}/day = (p_1 - p_0) * Pop$ where $p_1 = \frac{1}{1 + e^{-(\ln \frac{p_0}{1 - p_0} + \beta * \Delta O_3)}}$ and p_0 = the probability of having $Sympt_{days}$ per individual for a 24-hour period in the base case = 0.19 $\beta = 1.4 \times 10^{-4}$[c] (Model includes O_3, COH, SO_2)	s.e. 6.7×10^{-5}[c]	Krupnick et al., 1990

Health Endpoint (ICD-9 code)	Baseline Incidence (per 100,000)	Expos Meas from original study	Study Pop.	Applied Pop.	Functional form[a]	Uncert & Var.	Sources
self-reported asthma attacks	n/a	1 hour daily max. oxidants (ppm)	asthmatics in Los Angeles	all asthmatics (4%[d] of the total population)	Δ asthma attacks/day = $(p_1 - p_0) * Pop$ where $p_1 = \frac{1}{1 + e^{-(\ln\frac{p_0}{1-p_0} + \beta * \Delta O_3)}}$ and p_0 = the probability of attacks per asthmatic for a 24-hour period in the base case, = 0.027[e] $\beta = 1.9 \times 10^{-3}$ [f] 1.11 = factor to convert measured O_3 levels to oxidants (only model includes oxidants and TSP)	s.e. = 7.2×10^{-4} [f,g]	Whittemore and Korn, 1980 and U.S. EPA, 1993b
respiratory and nonrespira-tory conditions resulting in a minor restricted activity day (MRAD)	780,000/ year[h] (of study pop.)	1 hour daily max. O_3 (ppm) averaged over 2 weeks	employed adults across the U.S. between the ages of 18-65 (urban residents)	all adults aged 18-65	equation predicts daily change in MRAD $\beta = 2.2 \times 10^{-3}$ [i] (Model includes O_3 and fine particulates)	s.e. = 6.6×10^{-4} [j]	Ostro and Rothschild, 1989
respiratory restricted activity days (RRADs)	310,000/ year[k] (of study pop.)	1 hour daily max O_3 (ppm) averaged over 2 weeks	employed adults across the U.S. between the ages of 18-65 (urban residents)	all adults aged 18-65	equation predicts daily change in RRAD $\beta = -0.0054$[i] (Model includes O_3 and fine particulates)	s.e. = 0.0017[j]	Ostro and Rothschild, 1989

Health Endpoint (ICD-9 code)	Baseline Incidence (per 100,000)	Expos Meas from original study	Study Pop.	Applied Pop.	Functional form[a]	Uncert & Var.	Sources
sinusitis and hay fever	n/a	hourly O_3 averaged over six years (1974-1979) in ppm	adults in urban areas surveyed in the National Health Interview Survey	all	$\Delta cases = \frac{[\Phi(\alpha + \beta x_1) - \Phi(\alpha + \beta x_0)]}{6} * Pop$ where: Φ = standard normal distribution function x_1 = average hourly O_3 concentration over six years in the no-CAA scenario x_0 = average hourly O_3 concentration over six years in the CAA scenario a = -1.13[l] β = 0.017 maximum likelihood probit model	s.e. = 0.0070[m]	Portney and Mullahy, 1990

Health Endpoint (ICD-9 code)	Baseline Incidence (per 100,000)	Expos Meas from original study	Study Pop.	Applied Pop.	Functional form[a]	Uncert & Var.	Sources
The following two rows should be combined, e.g., cases of $DFEV_1 \geq 15\%$ for heavy exercisers (using equation based on Avol et al., 1984) should be added to cases of $DFEV_1 \geq 15\%$ for moderate exercisers (using equation based on data from Seal et al., 1993)							
Decrements in lung function as measured by forced expiratory volume in one second (FEV_1)	n/a	Exposure to ozone for 1.33 hours during which individuals were exercising continuously for one hour (controlled setting)	Heavily exercising male and female bicyclists (mean age = 26.4 yrs)	all under age 50[n]	$\Delta cases = \alpha * \beta * \Delta O_3 * Pop.$ where, β = 0.00297 for $DFEV_1 \geq 15\%$ = 0.00268 for $DFEV_1 \geq 20\%$ α = 0.06656[o]	--	Avol et al., 1984
Decrements in lung function as measured by FEV_1	n/a	Exposure to ozone for 2.33 hours during which individuals were exercising intermittently (total exercise time = 1 hour) (controlled setting)	Moderately exercising male and female college students (ages 18-35)	all under age 50[n]	$= \sum_{i=1}^{3} \left\{ \Phi \left[\frac{\ln\left(\frac{X_o \cdot d_i}{1 - X_o \cdot d_i}\right) - a}{b} \right] e_i - \Phi \left[\frac{\ln\left(\frac{X_1 \cdot d_i}{1 - X_1 \cdot d_i}\right) - a}{b} \right] e_i \right\} \cdot Pop$ where, a = -0.664 for $DFEV_1 \geq 15\%$ = -0.326 for $DFEV_1 \geq 20\%$ b = 0.000840 for $DFEV_1 \geq 15\%$ = 0.000919 for $DFEV_1 \geq 20\%$ d_1 = 1.06[p] d_2 = 1.00 d_3 = 0.70 e_1 = 0.288[q] e_2 = 0.224 e_3 = 0.640 X_0 and X_1 are ozone concentrations in the CAA and No-CAA scenarios	--	Seal, et al., 1993

NOTES:

[a] Pollutant coefficients expressed as a change in health effects per change in ppb O_3.

[b] Centers for Disease Control, 1992. Vital and Health Statistics, Detailed Diagnoses and Procedures, National Hospital Discharge Survey, 1990. Number of 1990 discharges divided by 1990 U.S. population (248,709,873) from City and County Databook, 12th edition, 1994, U.S. Dept. of Commerce, Bureau of the Census, Washington, D.C.

[c] Determined the incremental effect/unit O_3 by incorporating stationary probabilities from transitional probabilities. ESEERCO (1994) obtained transitional probabilities for adults from original study authors.

[d] U.S. EPA, 1994a.

[e] Calculated as baseline asthma attack rate (number of attacks per person per year) divided by 365 days per year. Number of attacks per person per year = 9.9 from National Center for Health Statistics, National Health Interview Survey, 1979 (as cited by Krupnick and Kopp, 1988).

[f] β coefficient and s.e. converted to Δ in cases/ppb O_3 based on the following relationship: 1 ppb O_3 = 1.11 ppb oxidants.

[g] Study did not report a s.e. Thus, the analysis assumed the largest s.e. possible (at p = 0.01, using a two-tailed test of significance)

[h] Ostro and Rothschild (1989) report average annual MRADs as 7.8 per person, using data from 6 years.

[i] β is a weighted mean using separate coefficients for six years. Each year's coefficient was weighted by the inverse of the variance for that coefficient.

[j] Standard error is the square root of the sum of the weights (sqrt[sum(1/var_i)], where I indicates the individual year).

[k] Ostro and Rothschild (1989) report average annual RRADs as 3.1 per person, using data from 6 years.

[l] Obtained by determining the products of beta coefficients for other independent variables and their mean values and summing these and the constant value.

[m] Calculated by dividing β by asymptotic t-ratio.

[n] From Table 12 in 1992 Statistical Abstracts, the percent of individuals in the U.S. population under age 50 = 75%.

[o] Factor to adjust for differences in concentration among microenvironments and amount of time spent in different microenvironments at heavy exercise rates.

[p] The values, d_i, adjust ozone concentrations for various microenvironments (outdoor — near road, outdoor — other, and indoor) using values reported in U.S. EPA, 1993.

[q] The values, e_i, adjust the response rates by the percent of time spent in each microenvironment at the relevant exercise rates (i.e., percent of time at a fast rate is used for Avol et al., 1984, and percent of time at a moderate rate is used for Seal et al., 1993). U.S. EPA (1993) presents information to determine e_i values.

Nitrogen Oxides

Nitrogen dioxide (NO_2) is the primary focus of health studies on the nitrogen oxides and serves as the basis for this analysis. The primary pathophysiology of NO_2 in humans involves the respiratory system and the concentration-response function identified for NO_2 describes the relationships between measures of NO_2 and respiratory illness.

A number of epidemiological studies of NO_2 are available; however, most have either confounded exposures (with other pollutants) or insufficient exposure quantification (e.g., exposure assessment indicates only absence or presence of a gas stove). Most studies consider NO_2 generated by gas stoves or other combustion sources in homes and are therefore not directly usable in concentration-response functions. However, studies by Melia et al, 1980 and Hasselblad et al, 1992 provide a reasonable basis for development of a concentration response function. Table D-8 presents the function obtained from their work. The function relates NO_2 to respiratory illness in children.

Table D-8. Summary of Concentration-Response Functions for NO_2.

Health Endpoint	Exposure Measure from Original Studies	Study Population	Applied Population	Functional Form[a]	Uncertainty/ Variab.	Sources
respiratory illness (as indicated by respiratory symptoms)	NO_2 measurements in bedrooms with Palmes tubes (one year time weighted average concentration in $\mu g/m^3$)	children ages 6 to 7	all (combining functions for men and women)	$\Delta Resp\ cases = \Delta Prob(Resp) * Pop$ where: Prob(resp) = probability of respiratory illness during a one year period: $Prob(resp) = \frac{1}{1 + e^{-\log odds}}$ and $\log odds\ Resp = -0.536 + 0.0275\ NO_2 - 0.0295\ gender$ gender = 1 for boys and 0 for girls (the term drops out for girls)	s.e. = 0.0132	Hasselblad, et al., 1992.

NOTES:

[a] This equation was obtained from two sources. The NO_2 coefficient was reported in Hasselblad et al., 1992. The background and gender intercepts were obtained via personal communication with V. Hasselblad 2/28/95 by Abt Associates. The equation was based on an evaluation by Hasselblad et al. of study results obtained by Melia et al. (1980). See text for further discussion.

Carbon Monoxide

Three concentration-response relationships are available for estimating the health effects of carbon monoxide. The first relates ambient CO levels to hospital admissions for congestive heart failure (Morris et al., 1995). The second equation (Allred et al., 1989a,b, 1991) relates the CO level in the bloodstream to the relative change in time of onset of angina pain upon exertion. The third relates the CO level in the bloodstream to the relative change in time of onset of silent ischemia. Due to the lack of quantitative information relating silent ischemia to a meaningful physical health effect, this analysis uses only the first two dose-response functions shown in Table D-9.

Table D-9. Summary of Concentration-Response Functions for Carbon Monoxide.

Health Endpoint	Baseline Incidence	Exposure Measure	Study Population	Applied Population	Functional Form	Uncert./ Variability	Sources
Hospital admiss. for congestive heart failure	n/a	average of hourly max CO (ppm)	Medicare population in 7 large U.S. cities (96% of which are ≥65)	65 and over	$\Delta cases = \beta * \Delta CO * Pop$ where $\beta = 1.1x10^{-7}$	s.e. = 1.9×10^{-8}	Morris et al., 1995 7 large U.S. cities
percent change in time to angina	baseline time to onset of angina during treadmill test from Allred et al. studies = 515 seconds at %COHb = 0.63[a]	CO (in ppm) averaged over 1 or 8 hours	men, age 35-75 years, stable angina, nonsmokers (of at least 3 months) at time of study	Angina patients in U.S. = 3,080,000 in 1989[b] Frequency of angina attacks for the study population = 4.6 per week (range = 0 - 63)[c, d]	percent change in time to angina $=\beta*\Delta$%COHb where: $\beta = -1.89\%$ and COHb = blood level of carboxyhemoglobin and Δ %COHb = 0.45 * ΔCO^e, where: CO = concentration of CO (ppm), for non-smoking adults undertaking light exercise (alveolar ventilation rate of 20L/min) for one hour at low altitude, with an initial COHb = 0.5%. OR Δ %COHb = 0.12 * ΔCO^e, where: conditions are the same as above except that study individuals are at rest (alveolar ventilation rate of 10L/min) for 8 hours.	s.e. = 0.81%	Allred, et al., 1989a,b, 1991

NOTES:

[a] Calculated as the mean of means from 3 pre-exposure treadmill tests and 1 post-exposure test (control exposure to air) (Allred et al., 1991).

[b] American Heart Association (1991)

[c] Allred et al. (1991)

[d] Multiple daily events are not modeled. Although it is possible that angina attacks may occur more than once per day, the average frequency of attacks was 4.6 per week (< 1 per day).

[e] Equation calculated from figure in U.S. EPA (1991a), p. 2-7.

Sulfur Dioxide

This analysis estimated one concentration-response function for SO_2 using clinical data from two sources on the responses of exercising asthmatics to SO_2, as measured by the occurrence of respiratory symptoms in mild and moderate asthmatics (see Table D-10).

Table D-10. Summary of Concentration-Response Functions for Sulfur Dioxide.

Health Endpoint	Expos Meas. from original study	Study Pop.	Applied Pop.	Functional Form	Uncert & Var.	Sources
Any Symptom (chest tightness, shortness of breath, etc.)	5-minute SO_2 concentration, ppm (using peak to mean ratio from hourly SO_2 concentration of 2:1 to 3:1)	generally young exercising asthmatics (ventilation rate 0.4 m^3/min)	exercising asthmatics - defined as 4% of general population, of whom 1.7% (range 0.2% to 3.3%) are exercising during waking hours	$\log odds\ Symp = -5.65 + 0.0059\ SO_2 + 1.10\ status$ where *status* = asthma status (0 for mild, 1 for moderate) $Prob(symp) = \frac{1}{1 + e^{-\log odds}}$ $Cases = Prob_{mild}(effects) \cdot Pop_{mild} + Prob_{mod}(effects) \cdot Pop_{mod}$ Cases = number of individuals with occurrences of at least moderate effects for all three measures. where Pop_{mild} = exposed population of exercising mild asthmatics (assumed to be 2/3 of asthmatic population); Pop_{mod} = exposed population of exercising moderate asthmatics (assumed to be 1/3 of asthmatic population)	s.e. for: const. term= 2.60 for SO_2 coeff = 0.0025 for *status* coeff = 1.44	data from Linn et al.(1987, 1988, 1990), Roger et al. (1985)

Estimating Welfare Effects of Exposure

In addition to avoided incidences of adverse human health effects, the air quality improvements estimated to result from the CAA yield additional benefits, namely welfare benefits. Table D-10 indicates a variety of benefits expected to have accrued through the avoidance of air pollution damage to resources. As indicated, data supporting quantified estimates of welfare benefits are more limited than those quantifying the relationship between air pollution exposure and human health. While evidence exists that a variety of welfare benefits result from air quality improvements, currently available data supports quantifying only a limited number of potential effects at this time. The Table lists the effects quantified in the section 812 analysis; each is discussed below.

Table D-11. Selected Welfare Effects of Criteria Pollutants.

Pollutant	Quantified Welfare Effects	Unquantified Welfare Effects
Ozone	Agriculture - Changes in crop yields (for 7 crops) Decreased worker productivity	Changes in other crop yields Materials damage Ecological - effects on forests Ecological - effects on wildlife
Particulate Matter/ TSP/ Sulfates	Materials Damage - Household soiling Visibility	Other materials damage Ecological - effects on wildlife
Nitrogen Oxides	Visibility	Crop losses due to acid deposition Materials damage due to acid deposition Effects on fisheries due to acid deposition Effects on forest
Sulfur Dioxide	Visibility	Crop losses due to acid deposition Materials damage due to acid deposition Effects on fisheries due to acid deposition Effects on forest

Agricultural Effects

This analysis was able to quantify the benefits to economic welfare attributable to the increased crop yields expected from CAA-related air quality improvements. Appendix F describes the method used to estimate such benefits using reported relationships between ozone exposure and yields of a variety of commodity crops.

It should be noted that the method used to allocate monitor-level ozone concentrations to estimate crop exposure differed from that used to estimate ozone health effects. Instead of assigning concentrations from the nearest monitor, the agricultural benefits analysis estimated ozone concentrations for each county nationwide. This was necessary because of two factors specific to the agricultural analysis. First, crop production is reported at the county level, so changes in crop yields associated with changes in ozone levels must be estimated for each county. Second, much of the nation's agricultural production of "commodity crops" (corn, wheat, soybeans, etc.) occurs at significant distances from the location of the population-oriented ozone monitors. Thus, an algorithm was used to assign ozone concentrations for the agricultural analysis for the control and no-control scenarios to county centroids based on a planar interpolation of concentrations at the nearest three monitors. Appendix F documents the details of the triangulation of ozone air quality data.

Materials Damage

Welfare benefits also accrue from avoided air pollution damage, both aesthetic and structural, to architectural materials and to culturally important articles. At this time, data limitations preclude the ability to quantify benefits for all materials whose deterioration may have been promoted and accelerated by air pollution exposure. However, this analysis does address one small effect in this category, the soiling of households by particulate matter. Table D-11 documents the function used to associate nationwide PM-10 levels with household willingness to pay to avoid the cleaning costs incurred for each additional $\mu g/m^3$ of PM-10.

Visibility

In addition to the health and welfare benefits estimated directly from reduced ambient concentrations of individual criteria air pollutants, this analysis also estimates the general visibility improvements attributed to improved air quality. Visibility effects are measured in terms of changes in DeciView, a measure useful for comparing the effects of air quality on visibility across a range of geographic locations for a range of time periods. It is directly related to two other common visibility measures, visual range (measured in km) and light extinction (measured in km^{-1}); however, it characterizes visibility in terms of perceptible changes in haziness independent of baseline conditions.

Visibility conditions under the control and no-control scenarios were modeled separately for the eastern and western U.S. In the east, the Regional Acid Deposition Model (RADM) generated extinction coefficient estimates for each of 1,330 grid cells in the RADM domain (essentially the eastern half of the country). The extinction coefficients were translated to DeciView using the relationship reported in Pitchford and Malm (1994). In the Western U.S., a conventional extinction budget approach provided DeciView estimates for 30 metropolitan areas (SAI, 1994). A linear rollback model provided the corresponding no-control estimates. Visibility estimates for both portions of the country were generated for the target years 1975, 1980, 1985, and 1990.

Table D-12 summarizes the methodology used to predict visibility benefits attributable to the CAA. Physical benefits for a given year are reported in terms of the average DeciView change per person in the modeled population.

Worker Productivity

Available data permits quantification of a final human welfare endpoint, worker productivity. Crocker and Horst (1981) and U.S. EPA (1994c) present evidence regarding the inverse relationship between ozone exposure and productivity in exposed citrus workers. This analysis applies the worker productivity relationship (reported as income elasticity with respect to ozone) to outdoor workers in the U.S. (approximately one percent of the population). Table D-12 details the form of the concentration response function.

Ecological Effects

It is likely that the air pollution reductions achieved under the CAA resulted in improvements in the health of aquatic and terrestrial ecosystems. To the extent that these ecosystems provide a variety of services (e.g., fishing, timber production, and recreational opportunities), human welfare benefits also accrued. However, due to a lack of quantified concentration-response relationships (or a lack of information concerning affected population), ecological effects were not quantified in this analysis. Appendix E provides discussion of many of the important ecological benefits which may have accrued due to historical implementation of the CAA.

Table D-12. Summary of Functions Quantifying Welfare Benefits.

Endpoint	Expos Meas.	Applied Pop.	Functional Form	Uncert & Var.	Sources
Household Soiling Damage (change in dollar valuation)	annual mean PM_{10}	all households (study based on households in 20 metropolitan areas)	Soiling Damage = β * Pop/PPH * ΔPM_{10} where β = \$2.52 PPH = people per household (2.68) [a]	Beta distribution with mean = \$2.52 s.e. = \$1.00 interval = [\$1.26 - \$10.08] slope parameters: α = 1.2, β = 7.3	Manuel et al. (1982); McClelland, et al. (1991); Watson and Jaksch (1982); ESEERCO (1994)
Visibility (average change in DeciView per person) [b, c]	Eastern U.S.: Extinction coefficient (Ext) in units of m^{-1} Western U.S.: DeciView, dv (unitless)	all	$$\Delta Vis = \frac{\sum_i (dv_{No\text{-}CAA,\,i} - dv_{CAA,\,i}) \times Pop_i}{\sum_i Pop_i}$$ where, ΔVis = avg. change in DeciView per person in modeled population i = modeled area $dv_{No\text{-}CAA}$ = DeciView under no control scenario dv_{CAA} = DeciView under control scenario Pop_i = modeled population in modeled area, i In the East, Ext (in units of km^{-1}) is converted to dv as follows: $$deciview = 10 \ln\left(\frac{Ext}{0.01\ km^{-1}}\right)$$	not available	Pitchford and Malm (1994)

Endpoint	Expos Meas.	Applied Pop.	Functional Form	Uncert & Var.	Sources
worker productivity (resulting in changes in daily wages)	hourly O_3 concentration averaged over a workday or 24-hours (ppm)	individuals in occupations that require heavy outdoor physical labor (study based on citrus workers in S. California)	$\Delta I = I*\eta*(X_1 - X_0)/X_0 * Pop * W$ ΔI = change in total daily income, η = income elasticity with respect to O_3 conc., η = -0.14 for 24-hour period, I = total daily income per worker engaged in strenuous outdoor labor = $73[d] W = proportion of outdoor workers in the U.S. population = 0.012[e] X_0 = average hourly O_3 concentrations with CAA, X_1 = average hourly O_3 concentrations without CAA (NOTE: Average number of days worked per year for workers engaged in strenuous outdoor labor = 213)[f] (model includes O_3 only)	not available	Estimated using data from Crocker and Horst (1981) and U.S. EPA, 1994c

NOTES:

[a] 1990 Census

[b] Visibility is measured in two ways: (1) in terms of extinction coefficient in the eastern U.S. (based on modeling of RADM domain); and (2) as DeciView (dv) in the west (modeling of 30 western cities) (SAI, 1994).

[c] DeciView is a haziness index used to characterize visibility through uniform hazes.

[d] Average daily wage, assuming an 8-hour day, by workers in the job categories listed below, taken from U.S. Bureau of the Census, Earnings by Occupation and Education, 1990.

[e] Full- and part-time workers (total of 3,100,000) taken from U.S. Bureau of the Census, Earnings by Occupation and Education, 1990. Includes the following job categories: farm workers; groundskeepers and gardeners, except farm; forestry workers, except logging; timber cutting and logging occupations; brickmasons and stonemasons; brickmason and stonemason apprentices; roofers; structural metal workers; construction trades, n.e.c.; construction laborers; garbage collectors; and stevedores. Value is divided by total U.S. population.

[f] Average number of days worked per year, assuming an 8-hour day, by workers in the job categories listed above, taken from U.S. Bureau of the Census, Earnings by Occupation and Education, 1990.

Modeling Results

This section summarizes results of the health and welfare effects modeling. As indicated previously, the Project Team adopted a Monte Carlo approach in an effort to capture uncertainty in the benefits analysis. With respect to estimating avoided incidence of adverse health and welfare effects, two sources of variability are considered. The first is the statistical uncertainty associated with each concentration-response relationship reported in the literature. In addition to an estimate of a concentration-response function coefficient, studies typically report a standard error of the reported estimate. The second source of uncertainty lies in the choice of studies, where multiple studies offer estimates for the same endpoint. Different published results reported in the scientific literature typically do not report identical findings; in some instances the differences are substantial. This between-study variability is captured by considering the range of estimates for a given endpoint.

Table D-13 summarizes health and welfare effects for each study included in the analysis. The values presented are mean estimates of the number of cases of each endpoint *avoided* due to implementation of the CAA. A distribution is associated with each mean estimate, capturing the uncertainty inherent in the estimate of the concentration-response coefficient. The distribution of estimated effects corresponding to a given study was generated by randomly sampling from the distribution of coefficients (given by the estimated coefficient and its standard error reported in the study) and evaluating the concentration-response function, yielding an estimate of avoided incidence for the given effect. This procedure was repeated many times. While only the central estimates of the resulting distributions are presented here, the distributions were retained for use in monetizing and aggregating economic benefits (see Appendix I).[7]

As shown, for some health endpoints more than one concentration-response function was used, each representing a different study. The alternative concentration-response functions provide differing measures of the effect. These can be used to derive a range of possible results. In the case of lead (Pb), alternative functions were not used; rather, two analytical procedures were implemented (labeled the "backward-looking" and "forward looking" analyses), giving a range of results for most Pb endpoints (see Appendix G for discussion of Pb health effects).

The table presents the results of modeling "all U.S. population" (although, with the exception of Pb, not all of the 48 state population is modeled, with up to five percent being excluded in a given year). The results depict the *pattern* of health effects incidence across years. The accuracy of the *scale* of incidence is less certain (due to the extrapolation of air quality data). These results are almost certainly more accurate than the corresponding "50 km" results, but rely on the assumption that (for a portion of the population) distant air quality monitors provide a reasonable estimate of local air quality conditions. Thus, the results presented here are somewhat speculative. It is likely that the estimated health effects are overstated for that population group (20 to 30 percent of total population in the case of PM) for which distant monitors are used. (Note, however, that the scaling of unmonitored county PM concentrations based on regional-scale grid model projections significantly mitigates this potential overestimation in the case of PM; see Appendix C for details). Conversely, there is an implied zero health impact for that portion of the population (three to four percent in the case of PM) excluded from the analysis altogether, an understatement of health impacts for that group.

The results indicate the growth of benefits over the study period, consistent with increasing improvements in air quality between the control and no-control scenarios from 1970 to 1990.

The mortality effects documented above can be disaggregated by age. Table D-14 indicates the estimated proportions of premature mortalities for various age groups (Pb-induced mortality estimates for children, men, and women are grouped). Also presented is the average life expectancy for each group, indicating the degree of prematurity of PM and Pb-related mortality.

Table D-15 presents estimated incidence reductions for several health effects which could be quantified but not monetized for this analysis.

[7] With the exception of visibility, welfare endpoints estimated economic benefits directly and are therefore included in the monetary benefits results presented in Appendix I.

Table D-13. Criteria Pollutants Health Effects -- Extrapolated to 48 State U.S. Population (Cases per year - mean estimates).

Endpoint	Study	Pollutant(s)	1975	1980	1985	1990
MORTALITY						
Mortality (long-term exposure)	Pope et al., 1995	PM_{10}	58,764	145,884	169,642	183,539
Mortality (Pb exposure) -Male	Average of Backward & Forward	Pb	822	5,281	10,340	12,819
Mortality (Pb exposure) -Female	Average of Backward & Forward	Pb	231	1,474	2,866	3,537
Mortality (Pb exposure) -Infant	Average of Backward & Forward	Pb	456	2,342	3,933	4,944
CHRONIC BRONCHITIS						
Chronic Bronchitis	Schwartz, 1993b	PM_{10}	198,973	554,632	720,166	741,775
	Abbey et al., 1993	PM_{10}	173,571	454,309	564,753	602,990
OTHER Pb-INDUCED AILMENTS						
Lost IQ Points	Average of Backward & Forward	Pb	1,028,492	5,031,157	8,559,426	10,378,268
IQ < 70	Average of Backward & Forward	Pb	3,780	20,074	36,520	45,393
Hypertension-Men	Average of Backward & Forward	Pb	830,299	5,276,999	10,087,115	12,646,876
Cor. Heart Disease	Average of Backward & Forward	Pb	1,313	8,444	16,671	21,069
Atherothrombotic brain infarction - Men	Average of Backward & Forward	Pb	181	1,128	2,165	2,690
Atherothrombotic brain infarction - Women	Average of Backward & Forward	Pb	84	529	1,020	1,255
Initial cerebrovascular accident - Men	Average of Backward & Forward	Pb	260	1,635	3,154	3,926
Initial cerebrovascular accident - Women	Average of Backward & Forward	Pb	120	758	1,466	1,804
HOSPITAL ADMISSIONS						
All Respiratory	Schwartz, 1995, Tacoma	PM_{10} & O3	32,004	77,827	95,435	106,777
	Schwartz, 1996, Spokane	PM_{10} & O3	29,393	69,449	93,137	119,290
	Pope, 1991, Salt Lake Valley	PM_{10}	30,982	73,093	86,407	95,486
	Schwartz, 1995, New Haven	PM_{10} & O3	23,137	55,096	66,385	73,842
	Thurston et al., 1994, Toronto	PM_{10} & O3	13,746	32,383	39,691	46,013
COPD + Pneumonia	Schwartz, 1994c	PM_{10} & O3	21,898	53,928	64,217	70,528
	Schwartz, 1996, Spokane	PM_{10} & O3	19,769	47,294	63,116	80,113
	Schwartz, 1994a	PM_{10} & O3	16,942	40,882	49,290	55,227
	Schwartz, 1994b	PM_{10} & O3	13,006	30,679	37,434	43,410
Ischemic Heart Disease	Schwartz and Morris, 1995	PM_{10}	6,348	14,709	17,289	19,098
Congestive Heart Failure	Schwartz and Morris, 1995	PM_{10}	5,733	13,365	15,742	17,362
	Morris et al., 1995	CO	3,022	8,543	17,028	21,835
OTHER RESPIRATORY-RELATED AILMENTS						
- Adults						
Any of 19 Acute Symptoms	Krupnick et al., 1990	PM_{10} & O3	41,631,456	98,876,110	117,275,400	129,529,717
- Children						
Shortness of breath, days	Ostro et al., 1995	PM_{10}	20,752,402	50,758,872	58,575,484	68,375,216
Acute Bronchitis	Dockery et al., 1989	PM_{10}	1,936,260	6,255,801	7,644,924	8,541,833
Lower Respiratory Symptoms	Schwartz et al., 1994d	PM_{10}	2,994,048	6,100,276	6,977,680	7,804,860
Upper Respiratory Symptoms	Pope et al., 1991	PM_{10}	500,395	1,292,922	1,557,177	1,683,854
- All Ages						
Asthma Attacks	Ostro et al., 1991	PM_{10}	264,430	548,306	686,953	841,916
	Whittemore and Korn, 1980; EPA ,1983	O3	193	482	816	1,080
Increase in Respiratory Illness	Hasselblad, 1992	NO2	729,306	2,686,813	6,113,639	9,776,267
Any Symptom	Linn et al. (1987, 1988, 1990)	SO2	104,896	319,192	282,846	265,650
RESTRICTED ACTIVITY AND WORK LOSS DAYS						
RAD	Ostro, 1987	PM_{10}	19,170,337	47,445,314	56,939,271	62,187,720
MRAD	Ostro and Rothschild, 1989	PM_{10} & O3	60,871,610	155,799,151	190,333,140	209,924,785
RRAD	Ostro and Rothschild, 1989	PM_{10} & O3	47,669,732	237,799,482	176,850,171	174,329,691
Work Loss Days	Ostro, 1987	PM_{10}	6,966,775	17,213,581	20,648,906	22,562,752
HUMAN WELFARE						
Household Soiling Damage	ESEERCO, 1994	PM_{10}	direct economic valuation			
Visibility - East (DeciView chg. per person)	Pitchford and Malm, 1994	DeciView	0.4	1.4	1.9	2.0
Visibility - West (DeciView chg. per person)	Pitchford and Malm, 1994	DeciView	2.4	4.9	5.0	6.0
Decreased Worker Productivity	Crocker & Horst, 1981 and EPA, 1994c	O3	direct economic valuation			
Agriculture (Net Surplus)	Minimum Estimate	O3	direct economic valuation			
	Maximum Estimate	O3	direct economic valuation			

Table D-14. Mortality Distribution by Age: Proportion of PM- and Pb-related Premature Mortalities and Associated Life Expectancies.

Age Group	Proportion of Premature Mortalities by Age[a]		Life Expectancy (years)
	PM[b]	Pb[c] Forward (Backward)[d]	
Infants		33% (20%)	75
5-30			
30-34	2%		48
35-39	4%		38
40-44		11% (13%)	
45-54	6%	21% (25%)	29
55-64	13%	22% (27%)	21
65-74	24%	12% (15%)	14
75-84	29%		9
85+	22%		6
	100%	100%	

Notes:

[a] Distribution of premature mortalities across ages is fairly consistent across years.

[b] PM-related mortality incidence estimated only for individuals 30 years and older, consistent with the population studied by Pope et al., 1995.

[c] Pb-related mortality incidence was estimated for infants, women aged 45-74, and men in three age groups (40-54, 55-64, 65-74), each with a distinct concentration-response relationship.

[d] Forward (backward) analysis holds other lead sources at constant 1970 (1990) levels - see Appendix G. Values may not sum to 100% due to rounding.

Table D-15. Quantified Benefits Which Could Not Be Monetized -- Extrapolated to the Entire 48 State Population.

Endpoint	Study	Pollutant	1975	1980	1985	1990	Units
Pulmonary Function Decrements							
Decreased FEV by 15 % or more	Avol et al. 1984 & Seal et al. 1993	O3	53	121	196	312	*million person-days with decreased FEV (per year)*
Decreased FEV by 20 % or more	Avol et al. 1984 & Seal et al. 1993	O3	39	87	141	224	*million person-days with decreased FEV (per year)*
Chronic Sinusitis and Hay Fever	Portney and Mullahy, 1990	O3	6	8	8	9	*million cases/year*
Time to Onset of Angina Pain	Allred, et al., 1989a,b, 1991	CO	0.1%	0.4%	0.7%	0.8%	*fractional increase in time to onset of angina attack*

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